

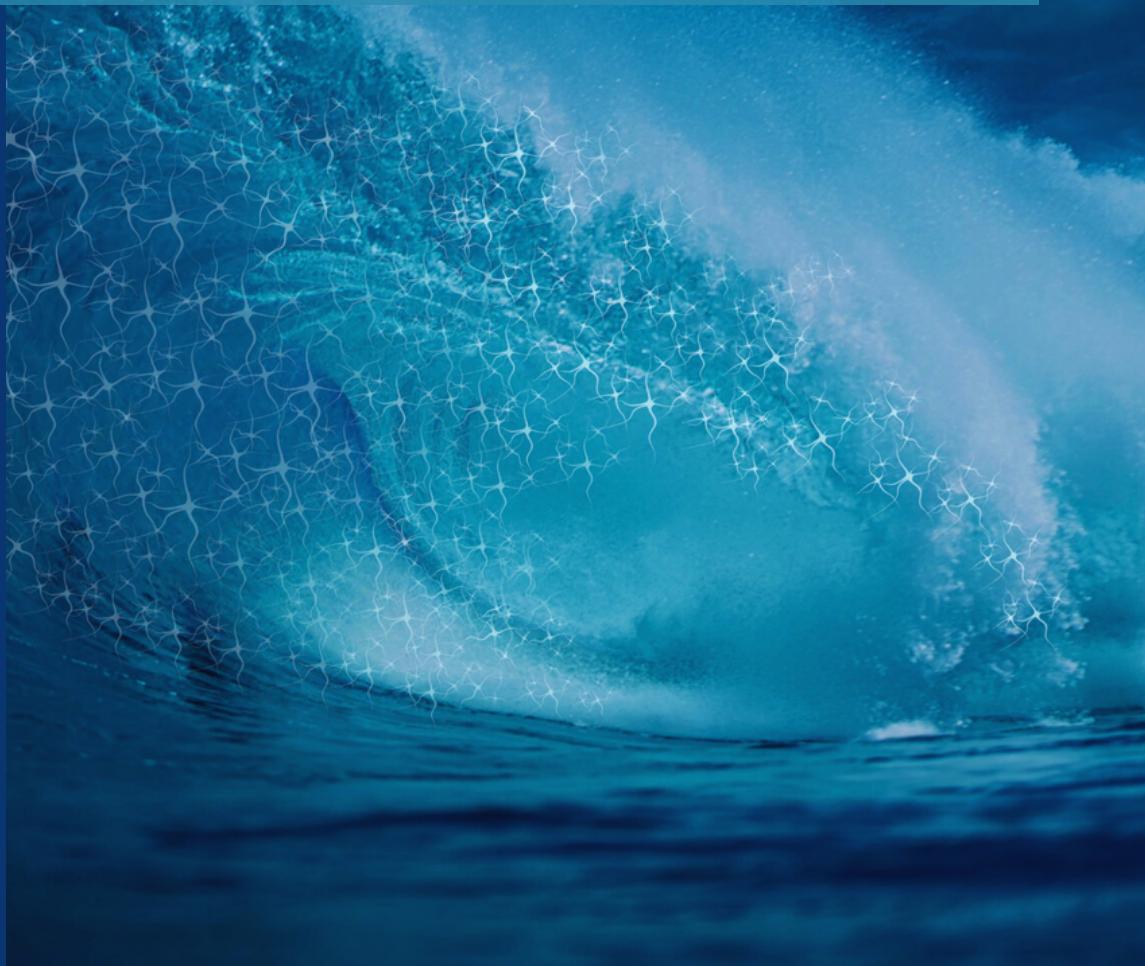


ASNAC

17TH ANNUAL CONGRESS

CONNECTOMES IN NEUROLOGY
NETWORKING FOR PRECISION CARE

Association of Sri Lankan Neurologists



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Proceedings
&
Abstracts

| 1st to 4th
MARCH 2024

Cinnamon Grand
Colombo, Sri Lanka

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Sponsors



**Association of Sri Lankan Neurologists
Council 2023/2024**

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Dr Anomali Vidanagamage, Prof. Jithangi Wanigasinghe, Dr. Kamal Gunaratne, Dr Kishara Gooneratne, Dr AT Alibhoy, Dr Arjuna Fernando, Dr Dilum Palliyaguruge, Dr Darshana Wijegunasinghe, Dr Gamini Pathirana, Prof. Thashi Chang, Dr Sanjaya Fernando, Dr Senaka Bandusena, Dr Saamir Mohideen, Dr Ajantha Keshavaraj

Seated from Left to Right:

Dr Manjula Caldera, Prof. Udaya Ranawaka, Prof. Saman Gunatilake, Dr Padma Gunaratne, Prof. Saraji Wijesekara (President-elect), Dr Pyara Ratnayake (President), Dr JB Peiris (Patron), Dr Darshana Sirisena (Immediate Past President), Dr MTM Riffsy, Dr Bimsara Senanayake, Dr Kumarangie Vithanage

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DR. PYARA RATNAYAKE

President

Association of Sri Lankan Neurologists

It is with great pleasure that I warmly welcome you to ASNAC 2024, the 17th annual congress of the Association of Sri Lankan Neurologists.

The theme of ASNAC 2024 is Connectomes in Neurology- Networking for precision care. The connectome is the complex network of neurons that results in the unmatched efficiency of the functioning of our brain. True to the concept we hope to provide opportunity for the participants to network and access the expanding armory of options to improve precision at the point of care delivery.

ASNAC 2024 has been endorsed by the World Federation of Neurology and the International Parkinson and Movement Disorder Society. The conference boasts of world renowned speakers and designs to offer a mixture of practical and futuristic neurology. The program opens with two pre congress workshops namely on neurological emergencies and EEG recording and interpretation for EEG technologists. This will be followed by two whole days of academic activities pertaining to a range of neurological conditions. The two post congress workshops are dedicated to EEG hands on learning for neurologists and discussion of difficult epilepsy cases. Several other case-based discussions are also taking place around the country taking

advantage of the goodwill of our speakers. These include case discussion sessions in myology, ophthalmology, paediatric movement disorders and autoimmunity as well as central nervous system tuberculosis. I wish to thank all the speakers for spending their precious time and energy to share their expertise with us.

I am grateful to the organizing team for their camaraderie and dedication to excellence. The dynamic team consists of Dr. Kumarangi Vithanage and Dr. Manjula Caldera (Joint secretaries), Dr. Kishara Gooneratne (Treasurer), Dr. Sanjaya Fernando, Prof. Saraji Wijesekera (President-elect) and Dr. Anomali Vidanagamage who were ably supported by Drs. Dilanka, Kavith, Tharindunie, Himal and Ruwani.

I gratefully acknowledge the pharmaceutical companies for their continued support to the ASN for the activities carried out throughout the year. ASNAC 2024, we hope will be an impetus as the Association of Sri Lankan Neurologists continues to drive for excellence of neurological care in the country. Your active participation is the crucial factor in the success of the conference.

Do enjoy the learning and the fellowship.





DR. MANJULA CALDERA
Consultant Neurologist



DR. KUMARANGIE VITHANAGE
Consultant Neurologist

As Joint Secretaries it gives us immense pleasure to send this message of welcome to all of you at the 17th Association of Sri Lankan Neurologists Annual Congress (ASNAC) 2024.

The year 2023 has been a challenging year for all of us with the economic crisis leaving us, with many financial and logistical constraints as sequelae.

Dissemination of knowledge was carried out through a series of updates by specialists in different fields related to neurology while providing an opportunity for the trainees to discuss important and interesting cases in clinical neurology at the neurology grand rounds. We managed to conduct a few regional meetings in Anuradhapura, Polonnaruwa, and Matale in collaboration with the regional clinical societies, which were deemed to be a huge success with huge participation. The interfaculty neurology quiz too was a great success which induced neurology enthusiasm among medical students.

We wish to thank our President, Dr Pyara Ratnayake for her excellent leadership with utmost confidence and commitment towards the activities of ASN.

We extend our sincere gratitude to the foreign faculty who accepted our invitation to join us physically at the ASNAC 2024. We are also grateful to all the overseas resource persons joining us virtually.

We place our appreciation to the Patron, Dr J.B. Peiris, and to all council members who actively participated in the activities to achieve the objective of "Connectomes in neurology, Networking for Precision Care" for ASNAC 2024. We also wish to thank our event manager, Ms Nimalka Morahela, Mr Nalina Wanasinghe at Impress Events PVT LTD for the audio-visual assistance, the staff of the Cinnamon Grand Colombo, our generous sponsors and all other well-wishers who helped us in numerous ways to make this conference a memorable one.

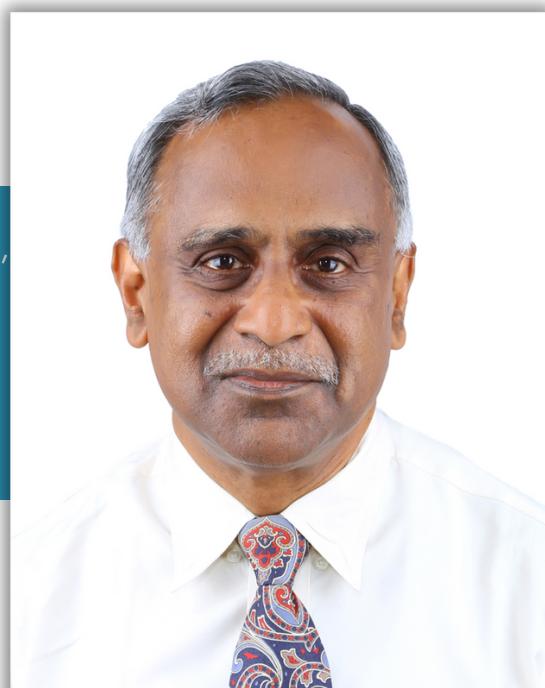
We look forward to your active participation and hope this event will provide opportunities to gain knowledge, to network, and expand fellowship.

Prof. Kurupath Radhakrishnan

MBBS, MD (Internal Medicine), MD (Neurology),
MNAMS (Neurology)

**Senior Neurologist and Epileptologist,
Avitis Institute of Medical Sciences, Nemmara,
Palakkad, and KIMS HEALTH,
Thiruvananthapuram, Kerala, India**

My connection with the Association of Sri Lankan Neurologists extends to more than 25 years. For me, the opportunity to be part of ASNAC 2024 is indeed an honour. The theme of the congress "Connectomes in Neurology Networking for Precision Care" aptly summarizes not only the way in which neurology has advanced over the last two decades, but also the way it is surging ahead. The tremendous advances that have happened in the fields of genomics and connectomics over the last decade have thoroughly transformed the concept of neurological disorders, which are now considered to be disorders of brain networks. In the neurological subspecialty of my expertise, epileptology, we have largely moved away from epileptogenic lobes, focus, and zones to structural and functional networks. The innovations in structural and functional neuroimaging have enabled us to delineate the epileptogenic and functional networks involved in the pathogenesis of not only the epileptic seizures but also the associated comorbidities afflicting speech, cognition, and behaviour. Based on the concept of genomics - connectomics interaction, most of the epilepsies, irrespective of whether they are idiopathic or acquired, are considered to have a genetic basis. We are born with a genetic predisposition to develop epilepsy, which



gets precipitated in later life by an insult, be it fever, trauma, tumour, or stroke. These changing concepts coupled with the tremendous advances in computing using artificial intelligence and machine learning have made precision medicine no longer hype but a reality. The classification of epileptic seizures and epilepsy syndromes are soon going to be genome-connectome based, the selection of antiseizure medications (ASM) will be influenced by pharmacogenomics, and we will be able to reliably predict the response, adverse effects, and resistance to ASMs. We will be able to select more ideal candidates for epilepsy surgery and predict their long-term postoperative outcome. The resective epilepsy surgery is soon going to be outdated and replaced by more precise and targeted minimally invasive approaches such as laser interstitial thermocoagulation. Modulation of brain networks through both invasive and noninvasive neuromodulation devices will replace resective and ablative surgical procedures. At the fag-end of my career, I envy the young neurologists and epileptologists; how lucky they are to be a part of the exciting times ahead.

I extend my gratitude for the privilege of being the Guest of Honour at the Association of Sri Lankan Neurologists Annual Congress, ASNAC 2024. Your unwavering commitment to neurological care amid economic crises is commendable. The Association's dedication to provide exemplary clinical care and advocacy for patients with neurological disorders during challenging times is truly inspiring. In the face of unprecedented challenges, the neurology community's resilience shines as a beacon of hope. Your advocacy efforts, promoting awareness and understanding of neurological disorders, contribute significantly to breaking down barriers and ensuring deserving individuals receive the necessary care and support.

As we convene for ASNAC 2024, let us celebrate the achievements of the past and collectively envision a future where compassion, innovation, and inclusivity continue to guide our healthcare journey. I eagerly anticipate the exchange of knowledge and collaborative efforts that will shape the landscape of neurological care in Sri Lanka. Thank you for your dedication. I am excited about the enlightening talks and progress that will undoubtedly emerge from this congress.



Dr Palitha Mahipala

Secretary
Ministry of Health

SCIENTIFIC PROGRAMME



WORLD FEDERATION
OF NEUROLOGY

ASNAC 2024

Association of Sri Lankan Neurologists

17th Annual Congress

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NETWORKING FOR PRECISION CARE



International Parkinson and
Movement Disorder Society



1st March 2024 (Friday) | Pre -Congress on Neurological Emergencies
 ClinMARC Auditorium, National Hospital of Sri Lanka

07:30 - 08:00	Registration	
08:00 - 08:30	Acute visual loss, neurologist's perspective Prof. U Thirugnanam	
08:30 - 09:00	Neuroprotection in intensive care Dr Sankalpa Vithanage	
09:00 - 09:30	Emergency care of central nervous system infections Dr Nicholas Davies	
09:30 - 10:00	Status epilepticus in the ICU Prof. Andrew Bleasel	
10:00 - 10:30	Tea	
10:30 - 11:00	Movement disorder emergencies Prof. Shen-Yang Lim	
11:00 - 11:30	Neurovascular emergencies Prof. Udaya Ranawaka	
11:30 - 12:00	Functional neurological disorders presenting as emergencies Prof. Jon Stone	
12:00 - 12:30	Neuromuscular emergencies Prof. Thashi Chang	
12:30 - 13:30	Lunch	

1st March 2024 (Friday) | Pre – Congress Workshop for EEG Technologists
 Epilepsy Auditorium, National Hospital of Sri Lanka

09:00 - 09:40	EEG recording techniques Dr Sudath Gunasekara	
09:40 - 10:00	Activation procedures Dr Sanjaya Fernando	
10:00 - 10:40	Identifying common EEG artifacts and troubleshooting Prof. Deepak Gill	
10:40 - 11:00	Tea	
11:00 - 11:40	Identifying normal variants and uncommon patterns of doubtful significance Prof. Kurupath Radhakrishnan	
11:40 - 12:20	Normal EEG, ictal changes and non-ictal pathological variants Dr Kishara Gooneratne	
12:20 - 12:40	Long-term EEG recording; VEEG and in ICU Dr Lakmini Pathberiya	
12:40 - 13:00	How to generate a factual report Prof. Deepak Gill	
13:00 - 14:00	Lunch	

2nd March 2024 (Saturday) | Congress Day 1

07:30 - 08:00	Registration	
08.00 - 09.00	Abstract Presentations	
09.00 - 09.30	Tuberculosis and the central nervous system Dr Nicholas Davies	
09.30 - 10.00	Functional neurological disorders, advances in management Prof. Jon Stone	
10.00 - 10.30	Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), an overview Prof. Russell Dale	
10.30 - 11.00	Tea	
11.00 - 11.30	Parkinson disease: What is new? Prof. Shen-Yang Lim	
Symposium on Networks in Neurology		
11.30 - 12.00	Treatment of reperfusion haemorrhage Prof. Andrew Lee	
12.00 - 12.30	Temporal lobe epilepsies: Epileptogenic zones to networks Prof. Kurupath Radhakrishnan	
12.30 - 13.00	Brain networks and neuropsychiatric disorders Prof. Ed Bullmore	
13:00 - 14:00	Lunch	
14:00 - 14:30	Management of large infarct core Prof. Andrew Lee	
Symposium on Infection and Inflammation		
14:30 - 15:00	Viral encephalitis and its mimics Dr Nicholas Davies	
15:00 - 15:30	Infection triggered encephalopathy syndromes Prof. Russell Dale	
15:30 - 16:00	Inflammatory muscle disorders Dr Channa Hewamadduma	
16:00 - 16:30	Case-based reviews with neuroimaging Prof. Bejoy Thomas	
16:30 - 17:30	TB complex case discussion Dr Arjuna Fernando Dr Nicholas Davies	 
17:30 - 18:00	Tea	

3rd March 2024 (Sunday) Congress Day 2		
07:30 - 08:00	Registration	
08:00 - 09:00	Abstract Presentations	
09:00 - 09:30	Use of rituximab in neurological disorders Prof. Anu Jacob	
09:30 - 10:00	Paradigm shift in the practice of medicine due to artificial intelligence: What a neurologist needs to know Dr Yudara Kularathne	
10:00 - 10:30	Neuroimaging in the era of AI: Our experience Dr. Sajitha Weerasinghe	
Tea		
Symposium on Epilepsy		
11:00 - 11:30	An update on idiopathic generalized epilepsy spectrum disorders Prof. Andrew Bleasel	
11:30 - 12:00	Emerging advances in the treatment of epilepsies Prof. Kurupath Radhakrishnan	
12:00 - 12:30	Genetic diagnosis for precision therapy in epilepsy Prof. Deepak Gill	
12:30 - 13:00	Concurrent use of anti-platelets and anticoagulants in acute ischaemic stroke Prof. Andrew Lee	
Lunch		
Symposium on Rare Neurological Disorders		
14:00 - 14:30	Metabolic disorders for the neurologist Prof. Jithangi Wanigasinghe	
14:30 - 15:00	An update on neurosarcoidosis Prof. Anu Jacob	
15:00 - 15:30	Case discussions: Rare neurological disorders	
15:30 - 16:00	Case studies in peripheral neuropathy Dr Satish Khadilkar	
Tea		
16:30 - 17:30	Neurology quiz	

4th March 2024 (Monday) | Post- Congress Workshop**EEG masterclass for Neurologists and Neurology Trainees****Epilepsy Auditorium, National Hospital of Sri Lanka**

08:50 – 09:00	Registration	
09:00 – 09:40	Understanding the chaotic lines Prof. Andrew Bleasel	
09:40 – 10:20	Misleading EEG artifacts and normal variants Prof. Kurupath Radhakrishnan	
Tea		
10:40– 11:20	Normal awake and sleep EEG Prof. Deepak Gill	
11:20 – 11:40	EEG in common focal and generalized epilepsy syndromes Prof. Andrew Bleasel	
11:40 – 12:20	EEG in epileptic encephalopathies and special patterns Prof. Deepak Gill	
12:20 – 13:00	EEG reading, interpreting and reporting Prof. Kurupath Radhakrishnan	
Lunch		
13:40 - 15:20	Hands on teaching session on EEG interpretation: Small group discussion	

FACULTY



WORLD FEDERATION
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International Parkinson and
Movement Disorder Society

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NETWORKING FOR PRECISION CARE





PROF ANDREW BLEASEL

B(MED)SC, MBBS, PHD, FRACP
**UNIVERSITY OF SYDNEY,
WESTMEAD CLINICAL SCHOOL
NEUROLOGY, EPILEPSY UNIT
WESTMEAD HOSPITAL
NEUROLOGY, JOHN HUNTER HOSPITAL
AUSTRALIA**

Dr Andrew Bleasel is a Neurologist at Westmead Hospital and John Hunter Hospital. He trained in Neurology at Royal Prince Alfred Hospital and then completed a PhD in cellular neurophysiology at Sydney University. Following a Clinical Fellowship in Epilepsy at the Cleveland Clinic in USA he was appointed to Westmead Hospital in 1996 where he was the Director of the Epilepsy Unit and then Head of Department, Neurology. In 2016 he was appointed as Academic Leader, Education, Sydney Medical School, Westmead. He became the Head of the Clinical School, 2021-2024. In 2023 he was appointed as a Staff Specialist in Neurology to John Hunter Hospital, Newcastle. He has a strong interest in medical education. He has been the Director of Physician Training at Westmead Hospital and a member of the Senior Examining Panel for the Royal Australasian College of Physicians. In his position at the Westmead Clinical School, he teaches all stages of medical students, manages the Medical Educators, and oversees the delivery of the Medical Curriculum.

He teaches Clinical Neurophysiology within Australasia for the Australian and New Zealand Association of Neurologists, the Epilepsy Society of Australia and the Asian Epilepsy Academy of the International League Against Epilepsy.

His research interests have concerned the investigation and management of refractory focal epilepsy with a focus upon seizure semiology, propagation pathways and stereoencephalography. In collaboration with other centres he has worked on projects dealing with Sudden unexpected death in Epilepsy patients (SUDEP), epidemiology of epilepsy and health service utilization of patients with refractory epilepsy. He supervises PhD students for the University of Sydney.



PROF. ED BULLMORE

MB PHD

PROFESSOR IN PSYCHIATRY
UNIVERSITY OF CAMBRIDGE
UNITED KINGDOM

Ed Bullmore MB PhD trained in medicine at the University of Oxford and St Bartholomew's Hospital, London and completed a PhD in brain MRI statistical analysis at the Institute of Psychiatry, Kings College London. He moved to Cambridge as Professor of Psychiatry in 1999 and was Head of the Department of Psychiatry from 2014-2021. He is currently Deputy Head of the School of Clinical Medicine and a Non-Executive Director of Cambridgeshire & Peterborough Foundation NHS Trust. From 2005-2019, he worked half-time for GlaxoSmithKline, as VP Experimental Medicine, focusing on immuno-psychiatry, as described in his best-selling book "The Inflamed Mind" (2018). His scientific work on brain networks and development of mental health disorders has been highly cited (H-index > 190). He has been elected as a Fellow of the Royal College of Physicians and the Royal College of Psychiatrists, as a Fellow and Treasurer of the Academy of Medical Sciences, and as an Honorary Fellow of Downing College.



PROF. THASHI CHANG

MBBS, MD, MRCP(UK), MRCP(UK)
(NEURO), DPHIL(OXON), FCCP,
FRCP(LOND)

PROFESSOR IN NEUROLOGY
DEPARTMENT OF CLINICAL MEDICINE
FACULTY OF MEDICINE
UNIVERSITY OF COLOMBO

Thashi Chang is a Professor in Neurology in the Department of Clinical Medicine at the Faculty of Medicine of the University of Colombo and an Honorary Consultant Neurologist in the Professorial Unit in Medicine at the National Hospital of Sri Lanka. He graduated from the University of Colombo with first-class honours. He is a Commonwealth Scholar and obtained his DPhil in Clinical Neurology from the University of Oxford, United Kingdom. He was the President of the Association of Sri Lankan Neurologists from 2019 to 2020.



PROF. RUSSELL DALE

MBCHB, MSC, MRCP, PhD
PROFESSOR OF PAEDIATRIC NEUROLOGY
UNIVERSITY OF SYDNEY AND
CHILDREN'S HOSPITAL AT WESTMEAD
AUSTRALIA

Professor Russell Dale is a Paediatric Neurologist and clinical academic at the Children's Hospital at Westmead and University of Sydney. He is Head of Clinical School and Head of Speciality of Child and Adolescent Health of University of Sydney, and Clinical Director of the Kids Neuroscience Centre, a research group of 110 clinicians and scientists.

He is an internationally recognised researcher in immune and autoimmune disorders of the brain, and neurodevelopmental and movement disorders of childhood. He has published 335 peer reviewed publications; his work has been cited 28,000 times and his H index is 86 (Google Scholar). His work has resulted in therapies for immune mediated brain disease being approved by government agencies and academic societies around the world, such as National Institute for Clinical Excellence, American Academy of Neurology, and European League of Rheumatology. He has led international consensus guidelines for the treatment of autoimmune brain disease in children and been senior author on discovery papers of essential diagnostic biomarkers which are now part of routine clinical care around the world. He has been awarded over AU\$25M in research funding and is a current NHMRC Leadership fellow (2021-5).



DR NICHOLAS DAVIES

BSC, PHD, MBBS, MRCP
CONSULTANT NEUROLOGIST
LONDON, UNITED KINGDOM

Dr Davies is a Consultant Neurologist at Chelsea and Westminster, Charing Cross, and the Royal Marsden Hospitals in London, UK. He is neurologist to the UK's National Centre for Human Retrovirology and leads the clinical service for HTLV-related neurological disease. His subspecialty and research interest is neurological infection. He trained in neurology in London. Prior to his consultant appointment he undertook a fellowship in HIV neurology at St Vincent's Hospital, Sydney, Australia. He is the chair of the Encephalitis Society's Scientific Advisory Panel.



PROF. DEEPAK GILL

MBBS, BSC, MRCP (UK), MRCPCH,
FRACP

**CONSULTANT PAEDIATRIC NEUROLOGIST
CHILDREN'S HOSPITAL AT WESTMEAD
AUSTRALIA**

Deepak Gill is a Paediatric Neurologist at The Children's Hospital at Westmead in Sydney. He is Associate Clinical Professor at the University of Sydney and Head of the Epilepsy Research Group of the Kids Neuroscience Centre. A/Prof Gill trained in medicine at Guy's Hospital in London, where he also attained a BSc in Radiological Sciences, and completed his fellowship in Paediatric Neurology in Sydney in 2000. A/Prof Gill has over 23 years' experience as a specialist and has co-authored 100 research papers in children's neurology, 29 of these papers have been related to describing novel observations or discoveries in the genetics of neurological disorders. His interests include the surgical treatment of epilepsy, including stereo-EEG, the developmental and epileptic encephalopathies, and the neuroimaging of epilepsy. A/Prof Gill has supervised and trained 27 fellows in clinical epilepsy, most who have gone on to continue their clinical expertise in children's epilepsy. A/Prof Gill is a former Vice-President of the Epilepsy Society of Australia and as part of the Asian Epilepsy Academy has conducted teaching across the region.



DR CHANNA HEWAMADDUMA

MBBS(HONS), FRCP(NEURO), FRCPE,
MSC(GENOMICS), PHD
NEUROMUSCULAR LEAD
CONSULTANT NEUROLOGIST AND
HONORARY SENIOR LECTURER
SHEFFIELD TEACHING HOSPITALS
FOUNDATION TRUST, SHEFFIELD.
SHEFFIELD INSTITUTE FOR
TRANSLATIONAL NEUROSCIENCES
(SITRAN), UNIVERSITY OF SHEFFIELD.

Dr Channa Hewamadduma is a consultant neurologist with a specialist interest in neuromuscular disorders at Sheffield Teaching Hospitals Foundation Trust (STHFT) and an Honorary Senior Lecturer at the University of Sheffield. He is a graduate of the University of Colombo with 1 st Class honours in the final clinical stream. He completed his PhD in neuro-genetics in Motor neuron disease, funded by a prestigious MRC Clinical Training Fellowship under Professor Dame Pamela Shaw in Sheffield, UK. He is also the co-chair of the South Yorkshire and Humber neuromuscular network. Dr Hewamadduma is the lead clinician of the neuromuscular service at STHFT and runs the regional myasthenia gravis referral service for Rituximab and Efgartigimod. He conducts a portfolio of neuromuscular clinical trials and basic science research in neuro-genetics and neuro-inflammatory disorders in SITRAN (Sheffield Institute for Translational Neurosciences) and at STHFT. Investigating new therapies and understanding how diseases progress using biomarkers and patient reported outcomes (PROMS) are his key focusses. He is a member of the NICE/NHSE advisory panel on genomic therapies in SMA and an invited clinical expert in NICE appraisal of neuroinflammatory conditions such as myasthenia gravis. He enjoys teaching and has set up master classes to help teach clinicians, neurology trainees and allied health professionals.



PROF. ANU JACOB

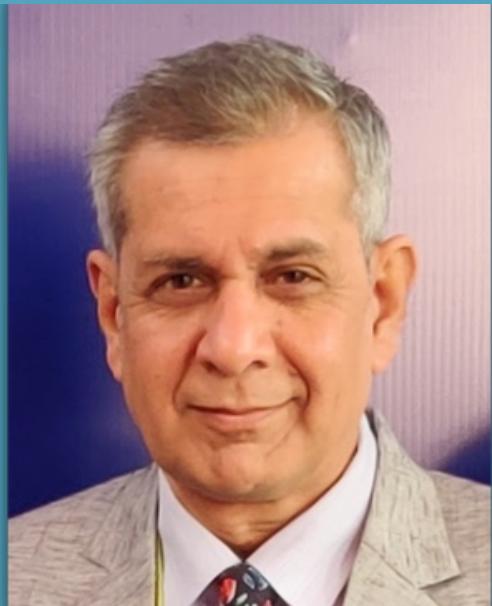
DM, FRCP
**DIRECTOR OF MS AND AUTOIMMUNE
NEUROLOGY**
CLEVELAND CLINIC
ABU DHABI

Professor Anu Jacob, DM, FRCP, is the Director of Multiple Sclerosis and Autoimmune Neurology and Staff Physician in the Neurological Institute at Cleveland Clinic Abu Dhabi.

Prior to joining Cleveland Clinic Abu Dhabi, Dr. Jacob was a consultant neurologist at the Walton Center for neurology and neurosurgery in Liverpool, United Kingdom.

Dr. Jacob completed his neurology training in prestigious Indian and UK institutes and his Multiple Sclerosis fellowship at Mayo Clinic, Rochester, Minnesota.

Dr. Jacob was the Director for Multiple Sclerosis services for North Wales, and founder director for the United Kingdom NHS specialist service for Neuromyelitis optica. He was awarded the Royal College of Physicians Excellence in Patient Care award for this work. He has published more than 125 peer reviewed research articles.



PROF S V KHADILKAR

MD, DM, DNBE, FIAN, FICP, FAMS,
FRCP (LONDON)

**DEAN, MEDICAL FACULTY, BHIMS
PROFESSOR AND HEAD, DEPARTMENT
OF NEUROLOGY, BOMBAY HOSPITAL
INSTITUTE OF MEDICAL SCIENCES,
MUMBAI
CONSULTANT NEUROLOGIST, BOMBAY
HOSPITAL INSTITUTE OF MEDICAL
SCIENCES, MUMBAI**

Prof. S. V. Khadilkar is a leading figure in the field of Neurology in India. He currently serves as Dean of the Medical Faculty and Professor and Head of the Neurology Department at Bombay Hospital Institute of Medical Sciences (BHIMS) in Mumbai. Previously, Prof. Khadilkar held the prestigious position of Professor and Head of Neurology at Grant Medical College and Sir JJ Group of Hospitals. Throughout his career, he has also served as a Consultant Neurologist at BHIMS and has held various leadership positions within the field, including Editor-in-Chief of the Annals of Indian Academy of Neurology, President of the Indian Academy of Neurology and the Nerve Muscle Society of India, and Secretary of the Asian Oceanian Myology Center. Prof. Khadilkar's dedication to the field extends beyond clinical practice, as he actively contributes to academic advancement through his numerous publications (over 250 in national and international journals), 8 books, and 57 book chapters. His commitment to education is further evidenced by his role as a teacher and examiner for DM Neurology and his recognition as the Best Teacher at GMC and JJH in 2006 and 2014. Prof. Khadilkar's extensive experience, leadership roles, and dedication to research and education solidify his position as a prominent figure in Indian Neurology.



DR YUDARA KULARATNE

FAMS, MACEP (USA), MMED (EM),
MBBS,
CEO HEHEALTH
CONSULTANT PHYSICIAN

Dr. Yudara Kularathne is a Consultant Emergency Physician, Artificial Intelligence (AI) Innovator and entrepreneur currently based in USA and Singapore. He was born in Sri Lanka and began his medical education at the Faculty of Medicine, University of Colombo. Subsequently, he moved to Singapore to complete his medical degree at the National University of Singapore (NUS). He then completed his Master of Medicine (MMed) at NUS in 2017 and also finished his training in the SingHealth Emergency Medicine Residency program, graduating in 2019 as a Consultant Emergency Physician.

From 2020 to 2022, Dr. Kularathne served as a Medical Specialist in the Emergency Department at Sengkang General Hospital under the SingHealth Group. During this period, he spearheaded the SKH Paediatric Emergency team and cared for more than 10,000 paediatric emergencies under his leadership. He was also involved with multiple innovation projects during this period.

In 2022, Dr. Kularathne opted to step out of academia to pursue his passion for innovations and founded HeHealth, a pioneering digital diagnostic startup. HeHealth leverages Artificial Intelligence (AI) to enable early diagnosis of Sexually transmitted infections (STIs), thereby facilitating timely interventions. HeHealth has developed multiple cutting-edge technologies that employ patented algorithms to screen and diagnose infectious diseases and facilitate population interventions. His work has been recognized by international organizations like WHO, CDC and ISSTDR.

Dr. Kularathne overall has a keen interest in Emergency Medicine, Engineering, and Healthcare Innovations using AI. Outside of his professional pursuits, Dr. Kularathne is an avid scuba diver and a certified PADI diving instructor, often spending his leisure time exploring the ocean depths and swimming with sharks.



PROF. ANDREW LEE

MBBS, MPH (JOHNS HOPKINS UNIVERSITY), FRACP
ASSOCIATE PROFESSOR
FLINDERS UNIVERSITY
ADELAIDE
SOUTH AUSTRALIA

Dr Andrew Lee is a Stroke Neurologist and an Associate Professor of Neurology, Flinders University, College of Medicine and Public Health and the Director of the Centre for Neuroscience Innovation. He completed training in both Internal Medicine and Neurology at The Queen Elizabeth Hospital, Adelaide South Australia before taking up a Stroke Research Fellowship at the Johns Hopkins University Hospital School of Medicine, Baltimore Maryland under the mentorship of Prof. Argye Hillis. While there he completed a Masters of Public Health at the Bloomberg School of Public Health, Johns Hopkins University researching effective ways of treating transient ischemic attack.

He returned to Adelaide and was appointed inaugural Director of Stroke at Flinders Medical Centre where he was on staff from 2007-2016. He is the recipient of a National Health and Medical Research Council (NHMRC-NICS) Fellowship looking at the implementation of safe thrombolysis in acute ischemic stroke. He has research interests in thrombolysis in stroke, intracranial hemorrhage and amyloid angiopathy and has numerous publications in peer reviewed journals.

He is the current director of the Centre for Neuroscience Innovation performing research in stroke as well as MS.

He is a reviewer for a number of high impact journals in neurology and stroke and is on the editorial board of the journal Stroke as well as the International Journal of Stroke.

His recent achievements include founding with colleagues a not-for-profit organisation, the Australian Stroke Academy, the purpose of which is to further training of medical practitioners in stroke medicine and furthering research in stroke. He served as the first chief executive officer.

He is currently on the board of the World Stroke Organisation as well as the Council of the Royal Australian College of Physicians.



PROF. SHEN-YANG LIM

MBBS MD FRACP FASC

**NEUROLOGIST AND PROFESSOR AT
THE UNIVERSITY OF MALAYA,
MALAYSIA.
CHAIR,
MOVEMENT DISORDERS SOCIETY
ASIA-OCEANIAN SECTION.**

Dr Lim is a Neurologist and Professor at the University of Malaya, Kuala Lumpur, Malaysia, where he runs a busy clinical practice specializing in Parkinson's and related disorders. Dr Lim has also published extensively in these areas, in major scientific journals.

He has been an active member of the International Parkinson & Movement Disorder Society (MDS), being closely involved in multiple task forces and committees. He is currently Chair of the MDS Asian-Oceanian Section (AOS).

Dr. Lim's main research interests are in the following areas:

- (i) Parkinson's Disease (PD) (particularly genetics and genotype-phenotype correlations; evidence-based medicine /clinical trials; non-motor features including gastrointestinal aspects; comorbidities; patient-centered care; and rating scales/disease staging)
- (ii) Parkinson-plus syndromes and other miscellaneous/"orphan" movement disorders, including progressive supranuclear palsy (PSP).

Weblink:

<https://umexpert.um.edu.my/limshenyang.html>



PROF. KURUPATH RADHAKRISHNAN

**MBBS, MD (INTERNAL MEDICINE),
MD (NEUROLOGY), MNAMS
(NEUROLOGY)**
**SENIOR NEUROLOGIST AND
EPILEPTOLOGIST,**
**AVITIS INSTITUTE OF MEDICAL
SCIENCES, NEMMARA, PALAKKAD
AND KIMS HEALTH**
**THIRUVANANTHAPURAM, KERALA
INDIA**

After completing Fellowships in EEG, Epilepsy and Neuroepidemiology from the Mayo Clinic, Rochester, Minnesota, USA, Kurupath Radhakrishnan joined as the Professor and Head of Department of Neurology at the Sree Chitra Tirunal Institute for Medical Sciences and Technology (SCTIMST), Trivandrum, Kerala, India, in January 1994. Within a year, he established the R. Madhavan Nayar Center for Comprehensive Epilepsy Care (RMNC), the first facility of this type in India. The objectives of RMNC were three-fold:

- 1) to undertake population-based studies on the burden, awareness and psychosocial issues with regard to people with epilepsy (PWE)
- 2) to evolve cost-effective medical and surgical treatment strategies for PWE
- 3) human resource development by imparting training to medical, surgical and paramedical personnel not only for India, but also in neighboring countries like Sri Lanka and Bangladesh.

By July 2013, when Dr. Radhakrishnan retired as the Director of SCTIMST, Trivandrum, he has accomplished more than what was envisaged, as evidenced by the following achievements:

- 1) Generating one of the most widely quoted epidemiological data on the prevalence of epilepsy, knowledge, attitude and practice (KAP) on epilepsy, psychosocial, occupational, and economic aspects of epilepsy from Kerala
- 2) The RMNC became not only a leading epilepsy surgery center in Asia with over 2000 epilepsy surgeries undertaken, but also helped to train over 100 epilepsy fellows and over 100 EEG technologists, and helped them to develop 30 epilepsy surgery programs in different parts of India and one in Sri Lanka
- 3) Conducting epilepsy clinics in rural areas, undertaking awareness programs for the public, and training programs for primary and secondary care physicians on basic care of PWE.

Additionally, RMNC has offered close to 100 mini-fellowships to neurologists from India, Sri Lanka, Bangladesh, Nepal and Kenya for

periods from 2 weeks to 6 months to undergo training in EEG and epileptology. For his contributions to the advancement of epilepsy care in the Asia and Oceania, Radhakrishnan was awarded "Outstanding Achievement Epilepsy Award" by AOEC, ILAE in 2012. After retirement from SCTIMST, Trivandrum, he joined as the Head of Neurology at Kasturba Medical College, Manipal, Karnataka, India, where he established another Comprehensive Epilepsy Program including epilepsy surgery. During the two years when Dr. Radhakrishnan was attached to Amrita Institute of Medical Sciences, Kochi, the EEG Laboratory and Epilepsy Surgery Program of this institute saw many new developments. More recently, at the age of 74 years, he is engaged in the development of a comprehensive epilepsy programs in KIMS HEALTH, Trivandrum and in a semi urban location at Nemmara, Palakkad, Kerala.

Dr. Radhakrishnan was instrumental in initiating the National Epilepsy Surgery Support Activity Network (NESSAN) in 2014, a platform for epileptologists to interact with each other, and discuss and debate on the selection of ideal candidates for epilepsy surgery. Over the last 6 years, NESSAN has helped many upcoming epilepsy surgery centers to obtain expert assistance in running their programs. The NESSAN also conducts EEG training courses every year, which has vastly helped in improving the way in which EEG is performed, read and interpreted in India. During the last 6 years, close to 900 neurology postgraduates and junior consultants have attended and certified through this course. The EEG book he compiled "EEG in Clinical Practice" has become immensely popular not only in India but the world over. Dr. Radhakrishnan has published over 300 articles on different aspects of epilepsy in international journals of high impact factors as evidenced by an H-index of 57 and number of citations over 11,508. In a recent compilation

of the top 2% of the scientists in the world by Stanford University, Dr. Radhakrishnan is the only one in the field of epileptology from India. In the World Online Ranking of Best Neuroscientists – 2023 Report by Research.com, Dr. Radhakrishnan was ranked Number 1 in India (and Number 3714 in the World).



PROF. UDAYA RANAWAKA

MBBS (NCMC), MD (COL), MRCP (UK),
FRCP (LONDON), FAAN, FAHA, FCCP
PROFESSOR IN NEUROLOGY,
DEPARTMENT OF MEDICINE, FACULTY OF
MEDICINE, UNIVERSITY OF KELANIYA

Professor Udaya Ranawaka is Professor in Neurology at the University of Kelaniya, and Honorary Consultant Neurologist and Head of the Stroke Unit at the Colombo North Teaching Hospital, Ragama, Sri Lanka.

He is a Past President of the Association of Sri Lankan Neurologists, National Stroke Association of Sri Lanka and the Ceylon College of Physicians. He is the Chairperson of the Sri Lanka Clinical Trials Registry and is a Member of the Advisory Group of the International Clinical Trials Registry Platform, WHO. He is a Fellow of the Ceylon College of Physicians, Royal College of Physicians of London, American Stroke Association and the American Academy of Neurology.

His main research interests include stroke, CNS infections, tropical neurology, Guillain-Barre syndrome and clinical trial registration. He has been a National Coordinator/Principal Investigator in several international clinical trials and research collaborations. He has won many national and international research awards, and has over 80 peer-reviewed publications to his credit.



PROF. JON STONE

MB CHB MEDICINE, MRCP (UK), PHD (UK), FRCR (UK)

**PROFESSOR OF NEUROLOGY
UNIVERSITY OF EDINBURGH
EDINBURGH UNITED KINGDOM**

Professor Jon Stone is Professor of Neurology at the University of Edinburgh and Consultant Neurologist with NHS Lothian.

Jon was a medical student in Edinburgh before working in Oxford, Leeds and Newcastle and then returning to Scotland. Since 1999 Jon has promoted a new transparent, pragmatic and multidisciplinary approach to functional neurological disorders (FND) which had been a neglected and stigmatised problem.

In 2009 he made the first website (and now app) for patients with FND at www.neurosymptoms.org which is now widely used across the world. He has published over 350 articles in the area including large cohort, mechanism and treatment studies and led on new diagnostic criteria for FND in DSM-5 and ICD-11. He is the first Secretary and co-founder, with Mark Hallett and Alan Carson of the new international FND society (www.fndsociey.org). His awards include the Jean Hunter prize from the Royal College of Physicians (2014), the Royal College of Psychiatry President's Medal (2017), the Ted Burns Humanism in Neurology Award from the American Brain Foundation (2020) and the John Walton Lecture Award from the Association of British Neurologists (2022).

Weblinks

www.neurosymptoms.org;

www.fndsociey.org;

<https://www.ed.ac.uk/profile/dr-jon-stone>;

Twitter: @jonstoneneuro

Email: Jon.Stone@ed.ac.uk



PROF. UMAPATHI THIRUGNANAM

MBBS (SINGAPORE), MRCP

**ADJUNCT ASSOCIATE PROFESSOR,
SENIOR CONSULTANT NEUROLOGIST,
DEPARTMENT OF NEUROLOGY,
NATIONAL NEUROSCIENCE INSTITUTE,
SINGAPORE**

**ADJUNCT ASSOCIATE PROFESSOR, DUKE-
NUS MEDICAL SCHOOL**

**ADJUNCT ASSOCIATED PROFESSOR,
DEPARTMENT OF INTERNAL MEDICINE,
YONG LOO LIN MEDICAL SCHOOL,
NATIONAL UNIVERSITY OF SINGAPORE**

Prof. Umapathi N.Thirugnanam is the Senior Consultant in the Department of Neurology, at the National Neuroscience Institute, Singapore. His sub-specialty interests are Neuromuscular Disorders, Electrophysiology, Autonomic Nervous System, Eye movements, and Neuro-immunology. He has been awarded the NHG outstanding partner award for the contributions made to ED residency over several years, the NHG Internal medicine residency outstanding teaching award, the NUS YLL SOM Dean's award for teaching excellence, NUS YLL SOM Special Recognition award over numerous years including 2022, Duke-NUS outstanding faculty award over a number of years and Singhealth Rise award-over a number of years. Has numerous publications in peer-reviewed journals. His research interests are Guillain-Barré syndrome, diabetic neuropathy and other neuromuscular disorders.



PROF. BEJOY THOMAS

MD, DNB, PDCC

**PROFESSOR SENIOR GRADE,
DEPT. OF IMAGING SCIENCES AND
INTERVENTIONAL RADIOLOGY
SCTIMST, TRIVANDRUM, KERALA
INDIA.**

Prof Bejoy Thomas is a highly accomplished individual with a distinguished career in the field of Neuroradiology. He has an impressive record of 248 international publications, including 8 book chapters and 1 edited textbook. His dedication to research is further evidenced by his clinical fellowships in pediatric neuroradiology and functional neuroimaging and his numerous awards such as certificates of merit from RSNA and prestigious lectureships at ISMRM. Prof Thomas is also recognized as a leading researcher, included in Stanford's top 2% global scientists list for 2020. His expertise lies in advanced neuroimaging, particularly magnetic resonance imaging, and pediatric neuroimaging, and he actively engages in research and teaching. Beyond his professional pursuits, Prof Thomas also expresses himself creatively through oil and acrylic painting.

[drbejoy2002@gmail.com.](mailto:drbejoy2002@gmail.com)



DR DEEPTI VIBHA

DR DEEPTI VIBHA,
MD (MEDICINE), DM (NEUROLOGY),
MSC(CLINICAL EPIDEMIOLOGY)
**PROFESSOR, DEPARTMENT OF
NEUROLOGY,**
**ALL INDIA INSTITUTE OF MEDICAL
SCIENCES, NEW DELHI, INDIA**

I'm Dr Deepti Vibha, a Professor in the Department of Neurology at the All India Institute of Medical Sciences, New Delhi, India.

I did my graduation from the King Georges' Medical University, Lucknow India in 2002 after which I specialized in Internal Medicine and subsequently did my training in Neurology from All India Institute of Medical Sciences (AIIMS), New Delhi, India in 2009.

I have been a faculty member at AIIMS, New Delhi since 2012. My specific areas of research interest are stroke, neuroinfections, and headache.

I have published 142 papers in PubMed, various chapters in books, and abstract papers in journals.

In addition, I have done a Masters in Clinical Epidemiology from Erasmus MC, Netherlands and I'm a board certified expert in Neurosonology (Transcranial and Carotid doppler).



DR SANKALPA VITHANAGE

MBBS (COL), MD (ANAESTHESIOLOGY)
**CONSULTANT INTENSIVIST, TEACHING
HOSPITAL KARAPITIYA**

Dr. K. S. Vithanage is the consultant Intensivist at Teaching Hospital Karapitiya, who holds an MD in Anaesthesia and over a decade of experience in anaesthesia and critical care. Dr. Vithanage is dedicated to quality improvement in critical care through innovative ideas and implementation. Passionate about evidence-based patient management, incorporating cutting-edge technologies to improve patient outcomes.



PROF. JITHANGI WANIGASINGHE

MBBS, MD(PEDA NEUROLOGY), DCH,
MPHIL, FRCPCH

**PROFESSOR IN PAEDIATRIC NEUROLOGY,
UNIVERSITY OF COLOMBO
CHAIR, PAEDIATRIC TASK FORCE OF THE
ILAE-ASIA OCEANIAN REGION**

Jithangi Wanigasinghe is a Professor in paediatric neurology at the University of Colombo. Her main research interests are Infantile Epileptic Spasm syndrome and epilepsy epidemiology. She is the current chair of the paediatric task force of the ILAE-Asia Oceanian region and the chair of the long-term development committee of the Asia-Oceanian Child Neurology Association. She has authored 105 publications in local and international peer reviewed journals.



DR SAJITHA WEERASINGHE

MBBS, MD MEDICINE (COLOMBO),
MRCP (UK), FRCP (LONDON), FRCP
(EDINBURGH), SCE NEUROLOGY (UK),
DIPUKMP (UK)

**CONSULTANT NEUROLOGIST,
TEACHING HOSPITAL PERADENIYA.**

Dr Sajitha Weerasinghe obtained his MBBS from the University of Peradeniya and MD from the University of Colombo. He is a fellow in the Royal Colleges of Physicians London and Edinburgh. He has a speciality certificate in Neurology UK and a Diploma in UK Medical practice from Liverpool School of Tropical Medicine UK. He had his overseas training in LEEDs UK. He was awarded the presidential research award for his research publications and he has a book chapter and local and international publications in peer-reviewed journals. He has presented at local and international conferences. He is a reviewer for local and international peer-reviewed journals. He has a special interest in AI in neuroimaging.

DR J B PEIRIS ORATION



WORLD FEDERATION
OF NEUROLOGY

ASNAC 2024

Association of Sri Lankan Neurologists

17th Annual Congress

CONNECTOMES IN NEUROLOGY
NETWORKING FOR PRECISION CARE



International Parkinson and
Movement Disorder Society



PROF. K D PATHIRANA

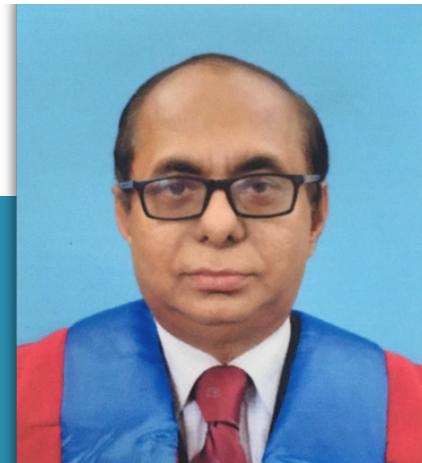
MBBS (PERADENIYA) MD (COLOMBO) FCCP (SL)

HONORARY CONSULTANT NEUROLOGIST

PROFESSOR IN MEDICINE

HEAD, DEPARTMENT OF MEDICINE,

FACULTY OF MEDICINE, UNIVERSITY OF RUHUNA



Professor K.D. Pathirana is a senior Neurologist with extensive experience in the field of Neurology, Neurophysiology and Medical Education. He graduated from the University of Peradeniya with second class honors and completed his MD from the Postgraduate Institute of Medicine, University of Colombo. He pursued further training in neurology at the Institute of Neurology, National Hospital of Sri Lanka under Dr J B Peiris, and at the Southampton General Hospital, United Kingdom.

With his board certification, in 1995, Professor Pathirana played a pivotal role in reactivating and developing the Neurology Unit at Teaching Hospital Kurunegala, where he worked as a specialist for three years. Since 1999, he has served as senior lecturer, professor and a senior professor at the Department of Medicine, Faculty of Medicine, University of Ruhuna, Galle, and currently holds the position of Senior Professor. He contributes his clinical expertise to The University Medical Unit, Teaching Hospital Galle as a Neurologist.

In addition to his teaching commitments, Professor Pathirana has served in various leadership roles within the Faculty of Medicine, University of Ruhuna as clinical coordinator, head of Department of Medicine, Information Technology Unit and

Medical Education Unit as well as chairperson and member of many committees.

He has also received training in Teaching in Higher Education and holds accreditations from the University of Colombo and SEDA (UK) in Teaching in Higher Education. He was conferred with the FCCP from the Ceylon College of Physicians in 2008. Professor Pathirana has a diverse range of research interests, including neurotoxicology, neurophysiology, stroke, epilepsy, aphasia, neurorehabilitation, and cognitive dysfunction. He has published extensively and presented his work at national and international conferences. He has received recognition for his research, including a Presidential Award and merit rewards. Beyond his academic and research pursuits, Professor Pathirana has served as the President of the Galle Medical Association in 2008 and the Association of Sri Lankan Neurologists in 2013.

His recent collaborations with Dr Noeline Prins, Senior Lecturer in Biomedical Engineering, in brain-computer interfaces for stroke rehabilitation highlight his commitment to innovative approaches in patient care.

IS SILENCE ALWAYS GOLDEN? SINGING SNAKE BITE VICTIM THAT LED TO RESEARCH IN SELF-THERAPY FOR APHASIA

PROF. K D PATHIRANA MBBS (PERADENIYA) MD (COLOMBO) FCCP (SL)

Aphasia is a language disorder that affects the ability to understand and express verbal or written language. Typically, the speech area is located in the left hemisphere of the brain in right-handed individuals and about 80% of left-handed individuals.

We encountered a unique case of a left-handed female who experienced a cerebral infarct in the right hemisphere, resulting in complete expressive aphasia following a Russel Viper bite. Interestingly, she was still able to sing when prompted. She could not write, although she could copy words written by others perfectly. This case inspired us to further study cognitive functions and explore the use of singing as a potential intervention for improving aphasia, as suggested by a senior consultant. This led to the development of various studies and interventions aimed at enhancing the quality of life for patients with aphasia.

To assess the prevalence of aphasia in stroke patients, a group of healthcare professionals, including nursing teachers, speech and language therapists, linguistic experts, neurologists, and bilingual non-health professionals, we validated the Sinhala version of the Mississippi Aphasia Screening Test (MAST).

Using the validated MAST scale, we found that approximately 32% of stroke victims had some form of aphasia, with 16% experiencing global aphasia, 13.5% having pure expressive aphasia, and 2.3% having pure receptive aphasia in a cohort of 257 patients. Using the Sinhala version of World Health Organization's Stroke-Aphasia Quality of Life (SAQOL 39 BREF) which was

validated in a similar manner to validation of MAST, we assessed the quality of life of patients with aphasia compared to those without. We found that patients with aphasia had significantly lower overall quality of life scores, as well as lower scores in the physical, communication, and psychological subdomains.

The type of stroke (whether it is an infarct or a haemorrhage), side of the lesion, Barthel index, and MAST score were all significantly different between the two groups as expected, with age showing a negative correlation with quality of life. Factors such as male sex, married status, higher education level, higher income, self-employment, and retired status were associated with higher quality of life scores indicating the importance of attending to language rehabilitation in these patients.

One of the main obstacles to proper rehabilitation for these patients is the lack of an adequate number of language therapists. Therefore, we propose exploring the potential of developing an app for speech therapy that can be tailored to local languages to address this issue. Additionally, we have successfully used brain-computer interface technology for motor rehabilitation in stroke survivors and aim to apply this approach to aphasic patients, although it presents more complexities. With the collaboration of experts in a wide variety of different fields, we are determined to realize this vision of rehabilitating patients with aphasia, which was initially inspired by the forward-thinking ideas of Dr. J.B. Peris.

ABSTRACTS OF PLENARIES AND SYMPOSIA



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08:30 - 09:00**Neuroprotection in intensive care****Dr Sankalpa Vithanage**

Neuroprotection in the intensive care unit (ICU) remains a paramount concern in optimizing outcomes for critically ill patients with neurological conditions. This presentation delves into the latest advancements and approaches in neuroprotection tailored specifically for critically ill patients. From pharmacological interventions to targeted temperature management and emerging neuroprotective agents, this explores the multifaceted strategies aimed at mitigating secondary injury mechanisms and preserving neurological function. By elucidating the intricate interplay between systemic insults and neurological vulnerability, this underscores the importance of a proactive and multidisciplinary approach in safeguarding brain health within the ICU environment. Through the integration of cutting-edge research and clinical insights, this presentation aims to elucidate promising avenues for enhancing neuroprotection and improving outcomes in critically ill neurological patients.

whether iatrogenic or HIV-related is critical in determining management strategies. The role of adjunctive steroids in management of acute bacterial meningitis in immunocompetent adults will be reviewed.

The talk will stress early involvement of critical care in the management of patients with suspected brain infections in order to manage associated sepsis, fluid management, fluctuating consciousness and airway protection, and intracranial pressure.

09:30 - 10:00**Status epilepticus in the ICU****Prof. Andrew Bleasel**

For the intensivist the clinical spectrum of status epilepticus includes convulsive, non-convulsive status epilepticus and refractory status epilepticus. The management of convulsive status epilepticus should begin in the ambulance and the emergency department with well-established protocols for intravenous antiseizure medication. The aetiology is the major determinant for outcome.

For the neurologist non-convulsive status epilepticus (NCSE) presents a diagnostic challenge due to its subtle clinical manifestations and varied aetiologies. NCSE is increasingly recognized in critically ill patients, with reported prevalence ranging from 8% to 20%. Aetiologies include traumatic brain injury, subarachnoid haemorrhage, ischemic stroke, hypoxic brain injury, encephalitis, and post convulsive status epilepticus. Clinical suspicion for NCSE should be maintained in patients with altered mental status, especially those with a history of epilepsy, acute brain insults, or focal cerebral pathology. Continuous EEG monitoring is advocated for optimal

09:00 - 09:30**Emergency care of central nervous system infections****Dr Nicholas Davies**

This talk will focus on early recognition and management of the syndromes of suspected meningitis and encephalitis in adults. Both conditions are associated with significant mortality and morbidity – early recognition and treatment are key management goals.

Early recognition of immunosuppression

detection, although it may not always be feasible due to resource constraints.

Refractory status epilepticus includes seizures that fail to respond to initial treatment or seizures that recur following an initial response. Titration of intravenous anaesthetic agents with continuous hemodynamic and neurologic monitoring remain the mainstay of treatment. Incorporation of the ketogenic diet, immunotherapy, and neurostimulation techniques is common.

10:30 - 11:00

Movement disorder emergencies

Prof. Shen-Yang Lim

Although "movement disorder emergencies" can be defined as conditions where patients develop a movement disorder over hours to several days, I will use "emergencies" to denote disorders in which failure to promptly and accurately diagnose and manage the patient may result in significant morbidity or mortality (on the other hand, prompt diagnosis and management can make a major difference to patient outcome).

There is thus a long list of conditions that can present as movement disorder emergencies.

In this lecture, I will focus more on hypokinetic emergencies. These can usefully be categorized under the following headings: withdrawal of antiparkinsonian treatment in a patient with a known parkinsonian disorder; drug-induced parkinsonism (neuroleptic malignant syndrome [NMS] constituting the more severe end of this spectrum); toxic-metabolic conditions; genetic disorders; infections; structural causes; and finally "miscellaneous related disorders" including catatonia.

I will show numerous videos to highlight the above conditions, including: drug-induced parkinsonism; deep brain stimulation (DBS)

failure; osmotic demyelination syndrome (ODS) with central pontine myelinolysis (CPM) and/or extra-pontine myelinolysis (EPM); Wilson's disease; carbon monoxide poisoning; structural causes such as subdural haematoma; parkinsonism in the context of HIV infection; and rapid-onset dystonia parkinsonism.

I will also briefly show examples of emergent conditions and "mimics" that can occur in patients with known parkinsonian disorders, such as stridor in patients with multiple system atrophy (MSA); and purposeless groaning in progressive supranuclear palsy (PSP) that could be misinterpreted as being due to pain.

11:00 - 11:30

Neurovascular emergencies

Prof. Udaya Ranawaka

Neurovascular emergencies remain an important cause of global mortality, morbidity and disability. Ischaemic stroke and intracerebral haemorrhage are the most important of these and have received much attention in recent times. However, there are several less common neurovascular emergencies that have not been adequately studied, and their causation, pathophysiology and management remain unclear to varying degrees. This talk will be a brief update on the current knowledge and understanding of some of these disorders.

Subarachnoid haemorrhage (SAH) is the least common stroke subtype, and continues to be a devastating disease with one-month mortality up to 35%. Recent trials have provided some evidence to guide the interventional management of ruptured aneurysms and hydrocephalus secondary to SAH. However, several trials on acute medical interventions have yielded negative

or conflicting results, and many of the treatments used are not supported by evidence. Cervical artery dissection is an important cause of ischaemic stroke, accounting for up to 25% of ischaemic strokes in young adults. Its pathophysiology is incompletely understood, and is believed to be multifactorial with environmental factors serving as possible triggers in individuals with a genetic predisposition to arterial dissection. Its clinical and radiological diagnosis is challenging. Recent evidence suggests that both antiplatelet and anticoagulant therapy provide similar benefits in stroke prevention after cervical artery dissection. Cerebral venous thrombosis (CVT) is a rare cause of stroke, but an important cause in young patients and women of reproductive age. Predisposing factors vary in different geographical regions, with infections being an important cause in resource-limited settings. Diagnosis depends on MR or CT venography, and acute and long-term anticoagulation is the mainstay of treatment. There is limited data on treatment approaches such as thrombolysis and endovascular treatment.

11:30 - 12:00

Functional neurological disorders presenting as emergencies

Prof. Jon Stone

FND is one of the most common reasons for neurological symptoms in the emergency department(1,2). A recent study in Scotland showed that it was the top reason for a request for neurological opinion in the general hospital (FND) (3). In this talk I will discuss:

- How to make a diagnosis of inclusion of FND with positive signs like Hoover's sign, the tremor entrainment test and

typical features of functional seizures.

- Diagnostic pitfalls both including missing FND, overdiagnosis of FND and consideration of feigning.
- What neurologists can do during the assessment to consider other neurological, functional and psychiatric comorbidities
- Sharing the diagnosis with patient and family and immediate management

1 Hallett M, Aybek S, Dworetzky BA, McWhirter L, Staab JP, Stone J. Functional neurological disorder: new subtypes and shared mechanisms. Lancet Neurol 2022; 21: 537–50.

2 Finkelstein SA, Cortel-LeBlanc MA, Cortel-LeBlanc A, Stone J. Functional neurological disorder in the emergency department. Acad Emerg Med 2021; 28: 685–96.

3 Ramsay N, Stone J, Fadiloglu K, et al. Functional neurological disorder: A common reason for a neurology inpatient referral. Eur J Neurol 2023; 30: 3886–9.

12:00 - 12:30

Neuromuscular emergencies

Prof. Thashi Chang

Neuromuscular emergencies are critical conditions that demand prompt recognition and immediate intervention, particularly when they lead to respiratory failure. Respiratory muscle paralysis can arise from dysfunctions at various levels of the neuromuscular system: the anterior horn cells (motor neuron disease), the peripheral nerve (Guillain-Barre syndrome), the neuromuscular junction (myasthenia gravis) or the muscle (muscular dystrophies). These conditions may stem from a diverse array of causes - autoimmune, inflammatory, toxic, metabolic, degenerative, or genetic. Each aetiology disrupts normal

neuromuscular function through different pathophysiological mechanisms.

Understanding these mechanisms is crucial in developing targeted treatment strategies.

The initial segment of the talk will focus on the recognition of impending respiratory failure in neuromuscular disorders. It will highlight the critical signs and symptoms that signify deteriorating respiratory function, emphasizing the importance of early detection to prevent catastrophic outcomes. Subsequently, the discussion will transition to the common neuromuscular causes of respiratory failure, focusing on myasthenia gravis and Guillain-Barré syndrome, providing insights into their pathogenesis, key clinical features, diagnostic challenges, and current therapeutic strategies. Finally, the presentation will conclude with a brief look at future directions in the management of neuromuscular emergencies.

09:00 - 09:30

Tuberculosis and the central nervous system

Dr Nicholas Davies

Mycobacterium tuberculosis (MTB) can affect the central nervous system in a myriad of ways: from asymptomatic tuberculomata to the most feared complication – meningitis.

This talk will use cases to illustrate neurological presentations related to CNS MTB and its complications. The speaker will draw upon his experience over 15 years of management of CNS TB in an European setting, acknowledging that the audience to which he speaks possess much larger wealth of practical experience in this field.

The advances in diagnostics of MTB infection, in particular molecular tests, will be discussed although the paramount importance of MTB culture will be stressed.

The therapeutics of management of MTB meningitis will be reviewed focussing upon the key areas of anti-mycobacterial drug treatment (and debate over drug doses), management of granulomatous inflammation, and prevention of ischaemic stroke.

Discussion of multidrug-resistant CNS TB infections or HIV / MTB coinfection will be beyond the scope of this talk.

09:30 - 10:00

Functional neurological disorders, advances in management

Prof. Jon Stone

In this talk I will focus on how the neurologist can become involved in the

management of FND and why it is no longer just a case of 'refer to the psychiatrist'(1).

The talk will cover

- Using the assessment as treatment and to tailor explanations of the diagnosis individually and appropriate use of self-help resources. (2,3)
- Developing neurology skills in triage and follow up – who may benefit from FND focused physio, who needs psychotherapy, who shouldn't be referred for treatment.
- Multidisciplinary therapy and how these differ from other neurological conditions. Physiotherapy (4), Psychological Therapy (5), Occupation and Speech therapy (6,7), new treatments including TMS and sedation.

1 Hallett M, Aybek S, Dworetzky BA, McWhirter L, Staab JP, Stone J. Functional neurological disorder: new subtypes and shared mechanisms. Lancet Neurol 2022; 21: 537–50.

2 Carson A, Hallett M, Stone J. Assessment of patients with functional neurologic disorders. In: Handbook of Clinical Neurology. 2016: 169–88.

3 Stone J, Carson A, Hallett M. Explanation as treatment for functional neurologic disorders. In: Handbook of Clinical Neurology. 2016: 543–53.

4 Nielsen G, Stone J, Matthews A, et al. Physiotherapy for functional motor disorders: a consensus recommendation. J Neurol Neurosurg Psychiatry 2015; 86: 1113–9.

5 Gutkin M, McLean L, Brown R, Kanaan RA. Systematic review of psychotherapy for adults with functional neurological disorder. J Neurol Neurosurg Psychiatry 2021; 92: 36–44.

6 Nicholson C, Edwards MJ, Carson AJ, et al. Occupational therapy consensus recommendations for functional neurological

disorder. *J Neurol Neurosurg Psychiatry* 2020; 91: 1037–45.

7 Baker J, Barnett C, Cavalli L, et al. Management of functional communication, swallowing, cough and related disorders: Consensus recommendations for speech and language therapy. *J Neurol Neurosurg Psychiatry* 2021; 92: 1112–25.

10:00 - 10:30

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), an overview

Prof. Russell Dale

Before the discovery of autoantibody biomarkers, most monophasic demyelination was inappropriately termed acute disseminated encephalomyelitis (ADEM), and relapsing demyelination was termed multiple sclerosis.

Due to the discovery of key autoantibody biomarkers in demyelination, we now recognise that there are key and distinct autoimmune demyelination syndromes, defined by their specific autoantibody. The first was neuromyelitis optica syndrome, defined by the presence of 'neuromyelitis optica IgG' which are aquaporin-4 autoantibodies'. The second was myelin oligodendrocyte glycoprotein associated disorder, defined by the presence of 'myelin oligodendrocyte glycoprotein autoantibody'.

We now know that MOGAD constitutes about 50% of all new onset demyelination syndromes in children, about 20% in adolescents, and ~2% in adults. In adults, multiple sclerosis remains much more common than NMOSD and MOGAD. However, the therapeutic pathway is different for MOGAD compared to MS, hence the recognition of the clinico-radiological

phenotype of MOGAD is important.

The talk will discuss the history of MOGAD, and the detection methodology (fixed versus live cell-based assays), but then focus on clinical and radiological features and therapy.

Approximately 50% of all ADEM cases have MOG autoantibodies, and MOG-ADEM has different features to non-MOG ADEM. The most important clinical syndrome of MOGAD is optic neuritis, and optic neuritis in MOGAD has differing features to optic neuritis in MS. ON in MOGAD tends to be bilateral (as opposed to unilateral), and often the ON is 'anterior' resulting in optic nerve head swelling, observable on fundoscopy. MRI reveals enhancing and swollen optic nerves which are 'longitudinally extensive', meaning the ON involves large rather than short segments of the optic nerve. Often the rest of the brain is unaffected (unlike MS).

The other important MOGAD clinical syndrome is transverse myelitis- again the clinical and radiological features of TM in MOGAD are distinguishing from NMOSD and MS.

These clinical and radiological characteristics can improve diagnostic suspicion and allow targeted autoantibody testing (where available).

The treatment of acute episodes involves corticosteroids, and if needed, intravenous immunoglobulin or plasma exchange. As many patients with MOGAD will only have one attack and not relapse, it is not recommended to start chronic immune therapy after the first attack of MOGAD (unlike in NMOSD or MS). In patients with relapsing MOGAD, the options include ongoing intravenous immunoglobulin, rituximab, tocilizumab or azathioprine /mycophenolate. The use of a steroid taper for 3-4 months after the first episode to reduce an early relapse will be discussed but is not yet established practise.

11:00 - 11:30

Parkinson's disease: What is new?

Prof. Shen-Yang Lim

This topic could potentially cover many interesting areas. In this lecture, I will focus on 4 main areas:

- Advances in genetics, and implications for diagnosis and prognostication ("natural" history, and also response to treatment), and ultimately translating improved understanding of disease pathogenesis to the advent of disease-modifying therapies
- The broad array of available tools to enhance care - from simple "pen-and-paper" rating scales like the CISI-PD, to technological advances including sensors for earlier diagnosis and management
- Continuous "dopaminergic" stimulation, particularly early application of DBS (also briefly infusions, and stem cell therapy)
- The "new" biological approach to PD emphasizing molecular biomarkers

12:00 - 12:30

Temporal lobe epilepsies: Epileptogenic zones to networks

Prof. Kurupath Radhakrishnan

The International League Against Epilepsy (ILAE) 1998 Classification of Epileptic seizures and Epilepsies distinguished two subtypes of temporal lobe epilepsies (TLE) – mesial (MTLE) and lateral (neocortical) TLE based on the electroclinical evidence for localization of the seizure onset zones (SOZ). In recent years, there has been a change in the concept of seizure genesis from zones to epileptogenic networks. In accordance with the network concept, the electroclinical

characteristics of TLE could occur with seizure genesis within the network in which temporal lobe is one of the principal nodes (but need not be generator). Thus, seizures originating from brain regions adjacent (but not within) the temporal lobe or at times even from remote areas may masquerade as TLE. Based on this hypothesis, in addition to mesial and lateral TLEs, several other TLEs are recognized. They are

- Temporal plus syndromes: SOZ in the orbito-frontal region, operculo-insular region and posterior cortex region;
- Bilateral TLE: bilateral independent temporal SOZs;
- Syndrome of dual pathology: hippocampal sclerosis with a second lesion adjacent to (but not within) the temporal lobe;
- Pseudo-temporal epilepsy: SOZ in remote lesions such as hypothalamic hamartoma or periventricular nodular heterotopia with propagation to temporal lobe; and
- Pseudo-extra temporal lobe epilepsy: SOZ within temporal lobe, but electroclinical features denoting an extratemporal region to where the seizure has propagated.

Failure to understand the heterogeneity of TLE is a common cause of surgical failure. The importance of identifying electro-clinical "red flags" in patients who superficially may mimic classical MTLE cannot be overemphasized. These red flags are absence of febrile seizures (and presence of trauma, meningitis, or encephalitis) as the antecedent, high seizure burden, frequent tendency for seizures to become bilateral, auras other than epigastric such as sensory, gustatory, auditory, and visual, extratemporal interictal epileptiform EEG discharges, and normal or atypical MRI and/or PET findings.

12:30 - 13:00

Brain networks and neuropsychiatric disorders

Prof. Ed Bullmore

In this talk, I will review recent advances in brain network analysis, or connectomics, that are relevant to understanding normal structure and development of large-scale human brain networks, with a focus on their topological properties as measured by graph theoretical analysis of cortical similarity matrices. I will rehearse some of the prior evidence that neuropsychiatric disorders generally impact on the most highly connected hubs of brain networks. Focusing on schizophrenia as a heritable, neurodevelopmental disorder of brain connectivity, I will report results providing new support for the concept that specific and generic genetic risks for schizophrenia (compared to Alzheimer's disease and bipolar disorder) are pleiotropically associated with brain structure at hubs of the normative connectome.

lethal.

In this presentation, we will review the progress of evidence on reperfusing large core leading to the most recent seminal trials and come up with recommendations whereby such therapy can be safely used especially in stroke services where the economic use of resources is paramount.

14:30 - 15:00

Viral Encephalitis and its mimics

Prof. Nicholas Davies

Viral encephalitis is a feared syndrome afflicting all ages including those who are immunocompetent as well as immunosuppressed. It is associated with significant mortality and amongst survivors acquired brain injury.

The causes of viral encephalitis vary and can be grouped into sporadic and epidemic causes. Distinguishing viral encephalitis from its mimics requires astute clinical judgement. Whilst ultimately diagnosis of aetiology is dependent upon identifying a virus by molecular methods or a virus-specific immune response, it is the clinician who delineates the syndrome through assiduous clinical assessment. Exposure through geographic, occupational or other factors are key history points. Some viruses have specific neurotropisms, which give aetiological clues.

Mimics of viral encephalitis are multitudinous: this talk will briefly highlight non-infectious mimics as the subsequent talk will cover in more depth other infection-triggered encephalopathies.

14:00 - 14:30

Management of large infarct core

Prof. Andrew Lee

Reperfusion therapy in acute ischaemic stroke has moved in leaps and bounds since the seminal publication of the NINDS/trial followed up by ECASS studies and then by the advent of combining endovascular therapy with thrombolytic. One of the key issues is whether large strokes can be safely reperfused. On 1 hand, the natural history of a large infarct core is poor. On the other hand, the risk of reperfusion haemorrhage is high whereby an intracranial bleed as a result of reperfusion is almost universally

15:00 - 15:30

Infection triggered encephalopathy syndromes

Prof. Russell Dale

The most common causes of acquired brain injury associated with infection is meningitis and encephalitis. Encephalitis involves invasion of the brain by infection, or invasion of the brain by inflammatory cells. The symptoms of encephalitis are due to a combination of infection and inflammation.

However, there are several acquired encephalopathy syndromes, which are triggered by fever or infection, which do not fulfil criteria of encephalitis. Some of these syndromes are termed 'Infection-triggered encephalopathy syndromes' and include specific clinico-radiological phenotypes. These syndromes are more commonly seen in Japan and East Asia but affect children and adults throughout the world.

All ITES syndromes have in common: ethnic and presumed genetic predisposition, age predisposition, a triggering infection (influenza being the most important), and evidence of CNS inflammation but without infiltration of the brain by inflammatory cells.

The more important syndromes are: Acute necrotising encephalopathy affects all ages, but pre-school children are most commonly affected. Early CT can be suggestive with bithalamic swelling and signal change. The MRI findings are characteristic with symmetrical bithalamic swelling, and sometimes involvement of the brainstem, putamen, cerebellum and white matter. The imaging is always symmetric with diffusion weighted imaging abnormalities, and sometimes complicated by necrosis. Other than encephalopathy, seizures and motor symptoms are common. There is often changes in liver enzymes and thrombocytopenia.

Acute encephalopathy with biphasic seizures and diffusion restriction (AESD) is an important and poorly recognised entity, characterised by an initial episode of status epilepticus following by partial recovery but residual encephalopathy for 3 days, and then onset of frequent focal seizures on days 4-5 onwards. The initial MRI is normal, but the MRI on days 4 onwards shows diffusion restriction in the white matter with a 'Christmas tree' appearance. Other than seizures and encephalopathy, dyskinesia is common.

Febrile-infection related epilepsy syndrome (FIREs) is recognised by some as an epilepsy syndrome, and others as an ITES. FIREs has similar features to other ITES syndromes and has clear evidence of neuroinflammation. FIREs is a devastating epilepsy syndrome, with uncontrolled and refractory, often focal, status epilepticus, often for weeks, with chronic epilepsy outcome, cognitive deficits and cerebral atrophy.

Mild encephalopathy with reversible splenial lesion (MERS) affects children and adults, and is a common cause of transient infection associated encephalopathy, and has a nearly universal good outcome. The MRI is infiltration of the brain by inflammatory cells.

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gastrointestinal tract and heart, indicating the systemic nature of this disease. I endeavor to discuss spectrum of clinical features, classification, a summary of the pathology, and investigations with an update of treatments available for IIMs.

15:30 - 16:00

Inflammatory muscle disorders

Dr Channa Hewamadduma

The idiopathic inflammatory myopathies (IIMs), known collectively as myositis, constitute a large spectrum of clinical phenotypes. As indicated by the name, the classical clinical manifestations of IIMs, such as muscle weakness, relate to chronic inflammation in skeletal muscles. This inflammation frequently affects other organs, including the skin, joints, lungs,

09:00 - 09:30**Use of rituximab in neurological disorders****Prof. Anu Jacob**

Rituximab is a B-cell-depleting monoclonal antibody and as a with growing evidence of efficacy and tolerability in several neuroinflammatory disorders. It is an attractive alternative to conventional immunomodulatory medications. The talk will outline principles of B-cell depletion with therapeutic monoclonal antibodies. It will provide evidence for using rituximab in neurological diseases, and describe the practical aspects of prescribing, including dosing, monitoring, safety, treatment failure and its use in special circumstances such as coexisting viral hepatitis, pregnancy and lactation. It will also suggest monitoring and risk reduction strategies.

09:30 - 10:00**Paradigm shift in the practice of medicine due to artificial intelligence: What a neurologist needs to know****Dr Yudara Kularathne**

This presentation delves into the pivotal role of Artificial Intelligence (AI) in reshaping contemporary medical practices, with a specific focus on its implications in the field of neurology. The talk begins with an introductory overview of AI, elucidating its fundamental concepts and explaining why it represents a game-changer in the healthcare industry.

The core of the discussion pivots to a detailed exploration of AI's diverse applications in neurology. It presents various case studies and examples to illustrate how AI is currently being utilized in the diagnosis, management, and treatment of neurological disorders. A significant emphasis is placed

on how AI influences patient experiences, particularly those grappling with neurological issues. This includes an in-depth look at AI's role in emergency neurology, especially in diagnostic imaging techniques like MRI and CT scans.

Furthermore, the talk extends to discuss AI's increasing involvement in the continuous management of neurological conditions. This section covers its utility both in hospital settings and in remote patient care at home, highlighting how AI tools and systems are revolutionizing patient monitoring and ongoing treatment strategies.

Looking towards the horizon, the presentation ventures into predictions and possibilities for AI in neurology. It offers insights into emerging technologies, ongoing research, and potential future applications that could further transform the field.

Finally, the talk concludes with a critical section dedicated to neurologists. It provides guidance on what neurologists need to know to stay ahead in this rapidly evolving landscape, emphasizing the importance of integrating AI knowledge and skills into their practice.

10:00 - 10:30**Neuroimaging in the era of AI: Our experience****Dr Sajitha Weerasinghe**

This talk explores the intersection of neuroimaging and artificial intelligence (AI). It delves into how AI, particularly machine learning, is revolutionizing neuroimaging by enhancing the analysis of complex neurological data. The discussion further illuminates how AI-driven neuroimaging can aid in diagnosis and treatment of neurological disorders based on local research findings.

11:00 - 11:30

An update on idiopathic generalized epilepsy spectrum disorders

Prof. Andrew Bleasel

Idiopathic generalised epilepsy (IGE) disappeared briefly as a diagnosis until it became clear how uncomfortable clinicians were with the term genetic generalised epilepsy. The key syndromes within IGE include childhood absence epilepsy, juvenile absence epilepsy, juvenile myoclonic epilepsy, and generalized tonic-clonic seizures alone. This update reviews the classification, epidemiology, clinical features, and treatment strategies for IGE. While we believe in a molecular basis for IGE decades of genetic research has not provided us with diagnostic tools or therapies. Specific clinical and electroencephalographic features can aid in determining prognosis for remission. Identification of permissible focal features in the seizure semiology and EEG have been helpful. The choice of antiseizure medications has expanded in the last 20 years and safer alternatives exist for management in pregnancy. Management of refractory seizures in patients with IGE can be as challenging as in focal epilepsy.

11:30 - 12:00

Emerging advances in the treatment of epilepsies

Prof. Kurupath Radhakrishnan

Even though few new antiseizure medicines (ASM) have been marketed in the last 2 years and few more are in the pipeline, none of them have significantly impacted the outcome of drug-resistant epilepsies (DRE). Cenobamate and cannabidiol (in children with developmental and epileptic encephalopathies) are claimed to be better

than other newer ASMs, however, pharmacotherapy of DRE continues to be disappointing. The tremendous advances that have happened in the fields of genomics, innovations in structural and functional neuroimaging that enable us to delineate the epileptogenic and functional networks involved in the pathogenesis of not only the epileptic seizures but also the associated comorbidities afflicting speech, cognition, and behavior, artificial intelligence (AI) and machine learning using large volume of data have made precision therapy in epilepsy no longer hype but a reality. The classification of epileptic seizures and epilepsy syndromes are soon going to be genome-connectome based, the selection of ASMs will be influenced by pharmacogenomics, and we will be able to reliably predict the response, adverse effects, and resistance to ASMs. Furthermore, we will be able to select more ideal candidates for epilepsy surgery and predict their long-term postoperative outcome. The resective epilepsy surgery is soon going to be outdated and replaced by more precise and targeted minimally invasive approaches such as laser interstitial thermocoagulation. Modulation of brain networks through both invasive and noninvasive neuromodulation devices will replace resective and ablative surgical procedures. Perhaps the most disabling aspect of epileptic seizures from the patients' perspective is its unpredictable occurrence. However, data gathered for several years through implanted devices such as responsive neurostimulator (RNS) have shown that seizures occur in cycles or "rhythms" that repeat every several days to weeks, called 'multidien rhythms'. Based on this information and utilizing AI and machine learning, seizure forecasting devices will soon be available that can warn the patients hours or days in advance the oncoming seizure, which could be aborted by extra ASM or by invasive or noninvasive

neuromodulation strategies.

12:00 - 12:30

Genetic diagnosis for precision therapy in epilepsy

Prof. Deepak Gill

The term “Developmental and Epileptic encephalopathy” (DEE), highlights the deleterious effect of severe infant onset epilepsy on the developing brain, where the epilepsy itself and epileptiform activity is associated with cognitive slowing and associated behavioural and psychiatric consequences. Once thought to be mostly related to birth injury or other prenatal or perinatal factors, many DEEs through gene discovery, are now known to have a genetic basis. Structural causes, such as malformation or cortical development also account for a significant proportion of cases. With more genes being discovered for DEE, the genetic diagnostic testing is now being increasingly utilised and becoming the standard of care, with up to 50% of DEE patients having a genetic cause elucidated, but many cases do not fit into clear epilepsy syndromes. Combined Next Generation Sequencing data, shows that 12 genes account for almost 2/3 of all monogenic cases. SCN1A, KCNQ2, CDKL5, SCN2A , PRRT2 and STXBP1 are the 6 of the most common genes. As new genes are discovered there remains a lag in the understanding of the natural history of many DEEs, as well as the full extent of the comorbidities associated with each condition. A framework where access to genetic investigations, in the same way patients can have access to EEG and MRI is the ideal model. As genetic testing becomes more readily available, many challenges with genetic testing remain. There is a gap in the knowledge, or the ‘genetic literacy’ of

neurologists, with the International League Against Epilepsy (ILAE) Genetics Commission, aiming to help close this gap. The translation of molecular genetics findings to therapeutics in DEE is continuing at a rapid pace with many genes being the subject of phase 1 studies and early phase 2 studies using precision therapies with gene-targeted treatment such as Anti-sense Oligonucleotide Therapy (ASOT). Further challenges are that access to genetic testing to people with epilepsy on a global perspective remains out of reach.

12:30 - 13:00

Concurrent use of anti-platelets and anticoagulants in acute ischaemic stroke

Prof. Andrew Lee

Antiplatelet therapy has been a mainstay for the prevention of acute ischaemic stroke since the early 1990s. Aspirin, clopidogrel and ticagrelor monotherapy as well as in combination with one another has been used for ischaemic stroke prevention. However the effect size has been modest at best, unless combined with other secondary prevention measures. These include the revascularisation of the symptomatic carotid artery, the deployment of antihypertensives and statins as well as monitoring for atrial fibrillation to convert an antiplatelet to an anticoagulant.

The advent of the direct acting oral anticoagulants over the last 10 years have revolutionised treatment. In particular, the risk of haemorrhagic stroke is much less compared to vitamin K antagonists.

Recent data has suggested that examination of aspirin with low dose effective than aspirin alone in the prevention of acute ischaemic stroke. This presentation will review the evidence for this and potential mechanisms as well as introduce a rapid way of imaging

both cerebral as well as coronary arteries using CT coronary angiography.

14:00 - 14:30

Metabolic disorders for the neurologist

Prof. Jithangi Wanigasingh

Neurometabolic disorders are rare and are almost all genetic in origin. Though frequently present in very early childhood, they are known to present in adulthood as well. The clinical manifestations are numerous and varied but neurology is a frequent association.

Major advances in the understanding of the genetics of these diseases has facilitated understanding of molecular defects identified in many of the disorders. Advanced neuro-imaging techniques, next-generation sequencing and next-generation metabolic screening have increased the speed and yield of the diagnostic process in neurometabolic disorders. This includes mandatory newborn screening in developed settings. On the contrary, in resource limited settings like ours, diagnosis is a great challenge. Clinical suspicion, detailed patient evaluation, family history and limited biochemistry are the main tools available for diagnosis.

Treatment is available for only a few of these diseases. In a majority, its management is mostly symptomatic. However, early suspicion and appropriate simple treatment strategies for some of these metabolic disorders can be lifesaving and or relieving of life-long morbidity. Disease-specific treatment strategies are available for very few and visible on the horizon for some.

14:30 - 15:00

An update on neurosarcoidosis

Prof. Anu Jacob

Neurosarcoidosis, is an uncommon neuroinflammatory disease with protean manifestations. The talk will focus on the pathology clinical and laboratory features diagnostic criteria and advances in treatments of neurosarcoidosis. It will also provide an approach to differential diagnosis, diagnostic approach of CNS inflammation, and treatment strategies.

Sarcoidosis: Sarcoidosis a systemic granulomatous disorder can uncommonly affect any part of the nervous system with varying severity. It forms part of the differential diagnosis in inflammatory, infective, neoplastic and degenerative neurological diseases and may be very difficult to diagnose without histological confirmation. There are many treatment options. The talk will give an overview of sarcoidosis and its treatment with the help of cases.

Rituximab treatment: Rituximab is a widely used B-cell-depleting monoclonal antibody. Though unlicensed for use in neurological disorders there is growing evidence of efficacy and tolerability in several neuroinflammatory disorders and it is an attractive alternative to conventional immunomodulatory medications. The talk will describe evidence for using rituximab in neurological diseases, the practical aspects of prescribing, including dosing, monitoring, safety, treatment failure and its use in special circumstances such as coexisting viral hepatitis, pregnancy and lactation.

ABSTRACTS OF ORAL & POSTER PRESENTATIONS



WORLD FEDERATION
OF NEUROLOGY

ASNAC 2024

Association of Sri Lankan Neurologists

17th Annual Congress

CONNECTOMES IN NEUROLOGY
NETWORKING FOR PRECISION CARE



International Parkinson and
Movement Disorder Society



OP:01**CLINICAL AND EEG CHARACTERISTICS OF CHILDREN WITH FIRST EPISODE OF SELF-LIMITED EPILEPSY WITH CENTROTEMPORAL SPIKES (SLECTS) IN A TERTIARY CARE PAEDIATRIC NEUROLOGY CENTRE IN SRI LANKA****Rupasinghe JPN 1, Galhenage JS 2, Ratnayake PD 3, Wanigasinghe J 4, Padeniya AB3**

1 Sirimawo Bandranayake Specialized Children Hospital-Peradeniya and National Hospital-Kandy, Kandy, Sri Lanka

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Introduction: Self-Limited Epilepsy with Centro-temporal Spikes (SLECTS) is the commonest childhood epileptic syndrome. Behavioural and neuropsychological deficits rarely emerge or worsen during the active phase of SLECTS although seizures usually resolve by puberty.

Objectives: To describe the clinical & electroencephalographic (EEG) characteristics, behavioural and emotional problems of children presenting with the first episode of seizures to the Lady Ridgeway Hospital.

Methods: A retrospective cross-sectional study was carried out at three paediatric neurology units. Children with the first episode of SLECTS were identified by screening all digital EEGs performed in 2019. Clinical details were obtained by contacting the parents. Electroencephalographic features were analysed using Nihon Kohden neurofax EEG-2100 and NicoletOne EEG systems. The child's current emotional and behavioural problems were identified using a validated version of Strength and Difficulties Questionnaire(SDQ). Data was analysed using SPSS 20.

Results: A total of 4756 EEGs were screened and 44 children were identified. The majority

were males; mean age was 7.9 (SD=2.6) years. Mean age at first seizure was 7.9 (SD=2.6) years. Family history of epilepsy was found in 6.8% and 9.1% gave a history of febrile seizures. Hemiclonic focal seizures (68.2%), unilateral facial sensory-motor seizures (54.5%), speech arrest (47.7%), hyper salivation (34.1%) and oro-pharyngolaryngeal seizures (22.7%) were the key manifestations; 65% had seizures during sleep. EEG findings were typical spikes and waves in 93.2% and, 52% were in the centro temporal region. One or more 'abnormal' scores were identified among 15 (34%) either in emotional, conduct, hyperactivity and peer problem subscales. Ten parents (22.7%) reported having learning deficits and 32% were concerned about the impact of their epilepsy. Bilateral EEG spikes were significantly associated with high impact scores ($p=0.008$).

Conclusions: The majority of children were males. Typical centro-temporal spikes and waves were identified in nearly all. Around one third of the children had some form of emotional and/or behavioural problems even at the onset of their epilepsy.

OP:02**THE CLINICAL AND GENETIC SPECTRUM OF ATAXIA AND MOVEMENT DISORDERS IN A COHORT OF SRI LANKAN PATIENTS**

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Introduction: Ataxia and movement disorders are common presentations of neurogenetic diseases. Next Generation sequencing based whole exome sequencing (WES) has revolutionized the diagnosis and management of them. However, data is lacking in under-represented populations. This study describes the clinical and genetic spectrum of ataxia and movement disorders in a cohort of Sri Lankan patients.

Methods: Data of 28 patients presented with ataxia and movement disorders above 14 years with suspected genetic aetiology but not confirmed with other investigations who underwent WES between January/2015 and January/2023 was maintained prospectively and analyzed retrospectively. Patients had undergone fragment analysis for Huntington's disease and spinocerebellar ataxia types 1,2,3,6,7,8 prior to WES if clinically indicated and found to be negative. WES data was generated using Illumina HiSeq platform with target sequencing coverage of 100X. Copy number variations and deep intronic variations were not captured. Patients were categorized based on the predominant phenotype according to clinical presentation.

Results: Mean age of the cohort was 33 years. Majority 57.1% (16/28) were males. Predominant phenotype was ataxia in 28.6%

(8/28) and movement disorder in 71.4% (20/28). Genetic variants were detected in 57.1% (16/28) comprising of 87.5% (14/16) pathogenic /likely pathogenic (P/LP) and 12.5% (2/16) variants of uncertain significance. The diagnostic yield of the cohort was 50% (14/28).

The predominant phenotypes, associated genes with P/LP variants and their respective frequencies were: Ataxia 28.6%(8/28)-ATM(12.5%,1/8), ELOVL5(12.5%,1/8), PRNP(12.5%,1/8), VPS13D(12.5%,1/8); Tremor 25%(7/28)-ATP7B(42.9%,3/7); Atypical parkinsonism 17.6%(5/28)-ATP7B(20%,1/5), SPTBN2(20%,1/5), PINK1(20%,1/5); Dystonia 10.7%(3/28)-TOR1A(33.3%,1/3), PANK2(33.3%,1/3); Chorea 10.7%(3/28)-VPS13A(33.3%,1/3) and Parkinsonism with dystonia 7.1%(2/28)-PARK7(50%,1/2).

Family members with similar phenotype was noted in 39.3% (11/28) and 10.7% (3/28) were products of consanguineous marriages.

Conclusions: The diagnostic yield of WES was 50% emphasizing its utility in genetic confirmation of hereditary ataxia and movement disorders which implicates in treatment, and prognostication.

OP:03**FEASIBILITY STUDY: USING BRAIN-COMPUTER INTERFACES IN STROKE REHABILITATION**

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Introduction: Physiotherapy is a major component in the rehabilitation of stroke survivors. Functional Electrical Stimulation (FES) is used as a component of physiotherapy. A Brain-computer interface (BCI) interprets the user's intent into actions.

Objectives: The objective was to explore the feasibility of implementing a BCI in subjects with upper extremity weakness due to stroke. We hypothesized that BCI augmented FES will enhance recovery of stroke by enhancing plasticity.

Methods: Six subjects were recruited, while results from subjects who completed the study are reported. BCI+FES system was developed with the power of different EEG frequency bands and using support vector machine and random forest algorithms, with accuracies of $52\% \pm 5.2$, $54\% \pm 7.5$ and $51\% \pm 3.9$ for each subject. For all 3 subjects the following functional tests were done pre-BCI and post-BCI: Fugl-Meyer Assessment (FMA), Barthel Index (BI) and Chedoke Arm and Hand Activity Inventory (CAHAI). For subjects P2 and P3, electromyography (EMG) were recorded from the extensor compartment of the forearm, while for subject P3, electroencephalography (EEG) was also recorded.

Results: All 3 subjects showed improvement in the functional scores. The total FMA score improved from 72.67 ± 21.502 to 94.0 ± 31.607 . The BI score improved from 68.33 ± 14.434 to 76.7 ± 20.207 while the CAHAI score improved from 13.33 ± 9.292 to 20.3 ± 13.796 . Subject P3 showed most improvement (36%, 18% and 50% improvement for FMA, BI and CAHAI respectively) while subject P2 showed the least improvement (24%, 8% and 43% improvement for FMA, BI and CAHAI respectively). EMG showed 133% improvement in P2 and 15% in P3. EEG power in mu and beta bands showed improvement of 0.5% and 0.9%.

Conclusions: In this feasibility study we found that BCI improved electrophysiological changes of acute stroke more than chronic stroke and it is a feasible add-on option to conventional physiotherapy. The data shows promising results as an exploratory study on the feasibility of implementing BCI for stroke rehabilitation.

OP:04**EFFICACY OF THALIDOMIDE IN THE TREATMENT OF TUBERCULOUS
OPTOCHIASMATIC ARACHNOIDITIS: INSIGHTS FROM A CASE SERIES**

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Background: Optochiasmatic arachnoiditis (OCA) results from accumulation of tuberculous exudates or tuberculomas in the interpeduncular, suprasellar and Sylvian cisterns. Although there is no treatment with proven efficacy, immunomodulation has been utilized in its management. Thalidomide, which inhibits tumour necrosis factor-alpha (TNF- α) synthesis has been used for its anti-inflammatory potential. We report our experience of thalidomide in the treatment of OCA.

Case Presentation: Five patients with OCA without HIV co-infection were treated with thalidomide after multidisciplinary discussions. This included three re-treatment patients and two new patients (Table 1). At the time of thalidomide initiation, all except one patient had raised cerebrospinal fluid (CSF) protein. Two patients had grossly elevated protein, lymphocytic pleocytosis and CSF/blood sugar ratio <0.5. Considered treatment failure, the latter two patients were on second line antituberculous therapy (ATT). All patients received intravenous dexamethasone prior to thalidomide. One patient received pulsed methylprednisolone, followed by three doses of intravenous infliximab 300 mg at 0, 2 and 6 weeks, with suboptimal response.

The average time from start of ATT to thalidomide initiation was 6.6 months (range 5-11 months). The median thalidomide dose was 2.3 mg/kg/day with treatment durations ranging from 3 to 12 months. Thalidomide led to clinical improvement in two patients, without significant visual recovery. Increased meningeal enhancement, enlarging tuberculoma and perilesional edema were noted in 4/5 patients (Figure 1). One patient continued to progress, necessitating surgical decompression. One patient died one month after starting thalidomide due to myocardial infarction in a background of known ischemic heart disease. This patient had transaminitis as a consequence of thalidomide. Rashes, cytopenia and neuropathy were not observed in any patients.

Discussion: Thalidomide may be of clinical benefit in selected patients with OCA despite paradoxical radiological worsening. Further studies are warranted to identify the optimal timing, dose, and duration of thalidomide.

OP:05**EVALUATION OF THE TREATMENT GAP IN MANAGEMENT OF CONVULSIVE STATUS EPILEPTICUS, IN CHILDREN AT THE LADY RIDGEWAY HOSPITAL, SRI LANKA****Hamid AA 1, Wanigasinghe J 1,2***1 Lady Ridgeway Hospital for Children, Colombo, Sri Lanka**2 Faculty of Medicine, University of Colombo, Colombo, Sri Lanka*

Objectives: To evaluate the treatment gap for convulsive status epilepticus (CSE) in children admitted to the premier children's hospital in the country and identify possible aetiologies, and compare the treatment gap with that described in Low and Middle-Income Countries.

Methods: This cross-sectional descriptive study with an analytical component investigated the clinical characteristics, management, and deviations from protocols in children presenting with CSE to the Preliminary Care Unit (PCU) of Lady Ridgeway Hospital for Children, over a three-month period in 2023. All children aged 3 months to 15 years who presented with seizures fulfilling the International League against Epilepsy (ILAE) definition of established SE, were included. Data were gathered related to patient characteristics, aetiology, pre-hospital care, management at PCU, description of the status, and deviations from ILAE protocols.

Results: This study analyzed 386 cases of seizures in children. The majority were 1-2 years old (28.0%) and male (59%). Most were simple febrile convulsions (68.7%). Pre-hospital care for the seizure was limited, with only 42.5% receiving any intervention. Among them was positioning (26.8%) and only two individuals received pre-hospital anti-seizure medications in the oral formulation. Total number of patients who

experienced established status was 177 (45.9%) however in 148 of them the status aborted before arrival at the hospital. Febrile seizures were the commonest aetiology (67.5%).

Thirty-nine arrived seizing in status epilepticus. Response to first-line medications was observed in 75%. ICU care was required for 3 patients, but only 1 received it immediately. Recognition of seizure onset was a major challenge, affecting 77.2% of cases. Education level of both parents was significantly associated with poor recognition of seizure ($p < 0.01$). Transport issues (38.3%), expert unavailability (9.1%), and drug availability (2.8%) were challenges hindering optimal management. Irregularities in medications/dosing and notes were found in 8 cases (2.1%).

Conclusions: Pre-hospital care with anti-seizure medication is underutilized. Poor recognition of the onset of seizures was associated with low parental education. Despite most cases being simple febrile seizures, many completed the diagnosis of established status epilepticus. Response to first-line treatments was frequently adequate. There was no treatment gap observed in hospital care during the study period.

OP:06**THE ROLE OF CYTOKINE-MEDIATED INFLAMMATION IN FEBRILE SEIZURES**

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Objectives: Immune mediated inflammation is believed to play a role in the pathogenesis of febrile seizures (FS). This study aims to compare the association of pro-inflammatory (IL-1beta, IL-6) vs anti-inflammatory (IL-4 and IL-10) cytokines between FS (cases) and non-seizure febrile illnesses (controls) in children aged 6 months to 6 years.

Methods: Cases (n=25) and controls (n=25) including 12 simple FS (SFS) and 13 complex FS (CFS) were recruited from the Lady Ridgeway Hospital, Colombo between January and October 2023. Serum cytokine levels were measured using Enzyme-Linked Immunosorbent Assays (ELISA).

Results: The levels of IL-1beta (64.38 ± 8.25 pg/ml vs. 63.27 ± 5.61 pg/ml) and IL-4 (1.21 ± 1.36 pg/ml vs. 0.52 ± 0.45 pg/ml) were elevated while IL-6 (59.73 ± 46.89 pg/ml vs 72.68 ± 62.27 pg/ml) and IL-10 (21.43 ± 11.21 pg/ml vs 23.22 ± 13.65 pg/ml) were decreased in cases compared to controls ($p > 0.05$). The levels of pro-inflammatory cytokines were higher than anti-inflammatory cytokines in cases (IL-1beta vs. IL-4: 64.38 ± 8.25 pg/ml vs. 1.21 ± 1.36 pg/ml; IL-6 vs. IL-4: 59.73 ± 46.89 pg/ml vs. 1.21 ± 1.36 pg/ml; $p < 0.05$). Serum IL-1beta was elevated in CFS compared to SFS (IL-1beta: 68.06 ± 9.41 pg/ml vs. 60.4 ± 4.3 pg/ml; $p = 0.024$). Positive correlations were identified between IL-1beta and IL-4 in CFS

($r = 0.637$; $p = 0.019$) and IL-1beta and IL-6 ($r = 0.643$; $p = 0.024$) in SFS.

Conclusions: A pro-inflammatory cytokine-biased immune response may contribute to the pathogenesis of FS. A higher concentration of IL-1beta can be observed in CFS compared to SFS.

Disclosures – This work was funded by the Association of Sri Lankan Neurologists' Annual Research Grant (Grant no. ASN/2023/04/02/01)

OP:07**REVIEW OF CLINICAL AND WHOLE EXOME SEQUENCING RESULTS FROM PATIENTS WITH DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHY WITH OR WITHOUT DRUG RESISTANCE (DEE +/- DRE) ATTENDING THE NATIONAL EPILEPSY CENTRE OF SRI LANKA.****Dantanarayana C 1, Weerapperuma G 1, Nissanka J 2, Padeniya P 3, Fernando S 1,4, Silva DD 2***1 Department of Paediatric Neurology, Colombo North Teaching Hospital, Ragama, Sri Lanka**2 Department of Physiology, Faculty of Medicine, University of Kelaniya, Kelaniya, Sri Lanka**3 Department of Anatomy, Faculty of Medicine, University of Kelaniya, Kelaniya, Sri Lanka**4 The National Epilepsy Centre of Sri Lanka, Colombo, Sri Lanka*

Objectives: Describe socio-demographic, clinical, and molecular findings of a consecutive series of patients attending the National Epilepsy Centre with developmental and epileptic encephalopathy and investigated using whole exome sequencing (WES).

Methods: Fifteen consecutive children with DEE+/-DRE who were investigated using WES were identified using case records maintained by a paediatric neurologist and clinical geneticist. Demographic, clinical, and genetic data were analysed using descriptive statistics.

Results: The fifteen patients included eight (53%) males. The average age was 1.81 ± 2.84 and 4.20 ± 3.07 years respectively for disease onset and WES testing. There were 4 (27%) fathers' and 2 (13%) mothers between 36-40 years at patients' birth. Patients were from ten districts. In 6 (40%) and 7 (47%) patients respectively, parental income was between Rs. 100,000-200,000 and >Rs. 200,000. Epilepsy-protocol-MRI brain scans were normal in 10 (67%) while interictal-EEG remained abnormal in 11 (73%). Seizure types included infantile spasms (7; 46.7%), generalized-motor seizures (4; 26.7%), and two each of focal-motor and focal-non-motor seizures. Eleven (73.3%) had global

developmental delay. Antenatal problems and term deliveries by Caesarean sections were reported in 8 (53.3%) and 13 (87%) respectively. None had significant perinatal insults. Mean birth weight was $2.95 \text{ kg} \pm 0.63$. In eight (50%), a diagnosis was made by WES including two SCN1A and one each of SCN2A, MECP2, NALCN, CACNA1D and PACS2 pathogenic/ likely pathogenic variants.

A clinically suspected Dravet-syndrome patient tested negative. In all WES diagnosed patients, there was a positive impact on management. Six had variants of uncertain significance (VUS).

Conclusions: Molecular diagnosis is helpful in determining the genetic aetiology and improves patient management. In Sri Lanka, costs and limited access to familial segregation or functional studies limits delineation of VUS. This series illustrates the cost implications of genetic testing as most families able to get testing were high income earners.

OP:08**MEETING THE TIME TARGETS IN ACUTE STROKE THROMBOLYSIS- A CLINICAL SURVEY AT DISTRICT GENERAL HOSPITAL EMBILIPITIYA (DGHE)****Vidanagamage AS 1, Palihawadana CNH 1, Gamage S 1, Kumara YU 1***1 District General Hospital Embilipitiya, Embilipitiya, Sri Lanka*

Objectives: Time is brain. It is vital to adhere to standard time targets in stroke thrombolysis. According to the internationally accepted time targets, it is aimed to achieve a door- to-needle time (DNT) within 60 minutes in 75% or more and a DNT of within 45 minutes in 50% or more of acute ischemic stroke patients treated with intravenous thrombolysis. A door-to-CT time (DTC) of 25 minutes or less is considered the best practice. A stroke thrombolysis service was established in December 2022 in DGHE following a series of targeted awareness program.

Methods: A printed protocol was utilized to document evaluation of every hyperacute stroke admission, triaged for thrombolysis. Demographic details, NIHSS score, and time taken at each step were documented. Data from December 2022 to November 2023 were collected.

Results: There were 22 patients who presented within the thrombolysis window, who underwent evaluation in the hyperacute stroke pathway. There were 15 males (68%). Eleven patients were thrombolysed after fulfilling the eligibility criteria. Out of them, 8 had moderate (NIHSS 5-15), one had moderate to severe (NIHSS 16-20) and 2 had severe (NIHSS 21-42) strokes. Out of the thrombolysed, the mean DTC time was 45 minutes, ($SD= 46.48$) with a range of 100

minutes (15minutes to 115 minutes). The mean DNT was 84 minutes, ($SD = 41.04$) with a range of 113 (42 minutes to 155 minutes). Only 37.5 % were thrombolysed within 60 minutes.

Conclusions: DGHE did not meet the standard goals of thrombolysis time windows which is comparable to other published data within Sri Lanka. Measures should be taken to minimize these time windows. Restructuring the thrombolysis flow within the hospital and continuous thrombolysis training for health care workers would be of potential benefit.

OP:09**EFFECTS OF A MEDITATION-BASED INTERVENTION ON MOTOR MANIFESTATIONS OF PARKINSON'S DISEASE: A RANDOMIZED CONTROLLED CLINICAL TRIAL****Vithanage KK 1, Dissanayake DWN 1, Chang T 2***1 Department of Physiology, Faculty of Medicine, University of Colombo, Colombo, Sri Lanka**2 Department of Clinical Medicine, Faculty of Medicine, University of Colombo, Colombo, Sri Lanka*

Objectives: Parkinson disease (PD) is the most common neurodegenerative disorder that leads to an akinetic-rigid syndrome. Although dopaminergic therapy remains the most effective treatment for PD, it often fails to provide optimal symptom control. Thus, we explored the efficacy of an adjunct meditation-based intervention (MBI) on the motor manifestations of PD in a randomized controlled trial.

Methods: Forty-six patients with PD (H&Y 1-3) were selected using convenient sampling and randomized to an interventional group (IG) and a usual-care-alone group (UC). IG underwent eight weeks of MBI in addition to their routine treatment. Motor manifestations were assessed via three tests: Short Parkinson's Evaluation Scale/Scales for Outcomes in Parkinson's Disease-motor function (SPES/SCOPA-Motor), Timed-Up-and-Go (TUG) test and Tibial nerve-nerve conduction study (NCS). SPES/SCOPA-Motor tool assessed motor evaluation (ME), activities of daily living (ADL) and motor complications (MC). TUG evaluated functional mobility and NCS evaluated neural processing and transmission. All tests were performed before and after intervention. Data was analyzed using SPSS-29 software. Non-parametric tests were used to assess outcome significance.

Results: Twenty-three PD patients each were randomized to IG (M:F=14:9; mean

(M:F=13:10; mean age=66.1, SD=6.6 years) and to UC SD=6.7 years). Baseline characteristics of the two groups did not differ significantly. IG group had a significant improvement in results following the intervention.

Post-intervention results between the 2 groups are; SPES/SCOPA-Motor [ME mean-rank: IG=12.4, UC=32 (SE=41, p<0.001), ADL mean-rank: IG=16, UC=28.2 (SE=40.9, p=0.001), MC mean-rank: IG=18.7, UC=25.5 (SE=39.3, p=0.06)], TUG test mean-rank: IG=18.4s, UC=25.3s (SE=40.5, p=0.05) and NCS, conduction velocity mean-rank: right side IG=28.9m/s, UC=14.7m/s (SE=41.1, p<0.001) and left side IG=28.2m/s, UC=15.5m/s (SE=41.1, p<0.001) while amplitude mean-rank: right side IG=31.1mV, UC=12.5mV (SE=41.1, p<0.001) and on left IG=31.4mV, UC=12.1mV (SE=41.1, p<0.001).

Conclusions: MBI is an effective adjunct in improving the motor manifestations of PD.

PP:01**UNVEILING THE MITOCHONDRIAL MYSTERY: A CASE OF MELAS****Weerasinghe NT¹, Weerasinghe WGNM¹, Dissanayake LK²1 Hordagoda HL¹, Peiris PJP¹***¹ The National Hospital Kandy, Kandy, Sri Lanka*

Background: Neurological disorders with mitochondrial inheritance, are a group of rare diagnoses which require a high index of suspicion. Among these, mitochondrial encephalopathy with lactic acidosis and stroke-like episodes (MELAS) is extremely rare and no cases have been reported from our part of the world.

Case Presentation: We came across a 38-year-old female patient with difficulty in walking and intermittent focal onset seizures with preserved awareness. She had been on treatment for epilepsy with multiple antiseizure medications since the age of 20 and had an episode of status epilepticus at the age of 25. Examination revealed nystagmus and other peripheral cerebellar signs. Lower limbs were spastic with up-going plantar responses bilaterally. An arterial blood gas revealed a raised lactate level of 3 mmol/l. Magnetic Resonance Imaging (MRI) scan of the brain revealed multiple cortical infarcts involving right temporal and parietal lobes and new T2 signal abnormalities involving old infarcts in the right occipital lobe and both cerebellar hemispheres. There was evidence of mineralization of the globus pallidus with cerebral atrophy. MR spectroscopy of the brain showed evidence of a lactate peak in the right signal region and in the cerebrospinal fluid of the lateral ventricles. Her cerebrospinal fluid lactate level was also elevated.

Her anti-seizure medications were reviewed as sodium valproate is contraindicated in mitochondrial disorders and she was started on coenzyme Q10. Her seizures settled and she was referred for physiotherapy.

Discussion: Mitochondrial encephalopathy is an important differential diagnosis to consider in refractory epilepsy as well as in strokes among young patients. This case is a good example of how neuroimaging could play a pivotal role for the diagnosis of MELAS in the absence of genetic studies due to limited resources.

PP:02**BRAIN IN BRAIN MALFORMATION, A RARE VARIANT OF MALFORMATION OF CORTICAL DEVELOPMENT****Wanasinghe WAKI 1, Ratnayake P 1, Chandrakumara A 2, Fernando S 3***1 Department of Paediatric Neurology, Lady Ridgeway Hospital for Children, Colombo, Sri Lanka**2 Department of Neuroradiology, National Hospital of Sri Lanka, Colombo, Sri Lanka**3 Colombo North Teaching Hospital Ragama and the National Epilepsy Centre of Sri Lanka, Colombo, Sri Lanka*

Background: Brain-in-brain malformation (BBM) is a rare malformation of cortical development (MCD), noted in patients with Drug-Resistant Epilepsy (DRE). So far, this condition has not been recognized as a specific entity due to its rarity.

Two cases (C1 and C2) with BBM are reported herein.

Case Presentation: Both patients started getting seizures during their early infancy (C1 – two months and C2 – six months), first seizure semiologies in both were - left focal motor seizure to generalized tonic-clonic seizures (GTCS). Semiology has evolved to asymmetric epileptic spasms (C1), focal motor status epilepticus to focal non convulsive status epilepticus (C2) since the onset. Currently both have Drug Resistant Epilepsy (DRE), with epileptic tonic drop attacks (C1). C2 gets focal to GTCS, atonic drop attacks and atypical absence seizures. Both get daily multiple events. The family history, antenatal and perinatal histories were unremarkable. Both are delayed in development (all domains), with low IQ and issues in Strength and Difficulty analysis. Clinical examinations were unremarkable. Inter-ictal EEGs were compatible with epileptic encephalopathy with some focal changes. 3Tesla - MRI scans of brain revealed the midline – to left conglomerate masses of dysplastic brain tissues

representing miniature brains with deeply infolded tissue representing gyral folds with grey matter lined cortical tissues. With incomplete lobar separation, the absence of corpus callosum are in keeping with the interhemispheric variant of holoprosencephaly. Polymicrogyria with grey matter heterotopia are noted at different foci of the rest of the brain. C2 had periventricular band heterotopia at the left temporal region.

Discussion: Brain in brain cortical malformations are rare entities of MCD, may represent a severe form of subcortical grey matter and or neuro-glial heterotopia. However, the exact aetiopathogenesis and the classification are yet to be elucidated. We suggest multicentre data collection for further delineation of Brain in Brain Cortical malformation.

PP:03

EPILEPSY IN INFANCY WITH MIGRATING FOCAL SEIZURES WITH MUTATION IN KCNAB1 SUCCESSFULLY TREATED WITH TOPIRAMATE**Vinushiya KG 1, Ratnayake P 1, Gunapala R 1, Anandagoda G 2, Dissanayake VHW 2**

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Background: Genetic factors are a major contributor to drug resistant epilepsy. KCNAB1 mutation causes epilepsy with temporal spikes but has not been reported to cause epilepsy in infancy with migrating focal seizures (EIMFS). We report a case of KCNAB1 associated EIMFS with migrating focal seizures controlled by topiramate.

Case Presentation: A Sri Lankan boy presented with refractory seizures since day 2 of life. Semiology was an abnormal cry followed by clonic movements. By 5 months, he had multiple daily seizures going up to twenty events a day and had global developmental delay. He was the 1st child born to nonconsanguineous parents without any antenatal or perinatal complications. His maternal grandmother was treated for epilepsy but didn't have any apparent developmental concerns. His electroencephalography (EEG) showed slowing over the left temporal region and migrating focal seizures of temporal origin. Extensive metabolic screening and imaging didn't reveal any aetiology. Whole exome sequencing showed him to have a heterozygous missense variant (c.1043C>T (p.Ala348Val) in the KCNAB1 gene, known to be associated with susceptibility to various forms of epilepsy with temporal spikes on EEG. Parental screening for a similar mutation was negative. His seizures remitted after adding topiramate at 5 months. He was started on early developmental intervention. Currently the

child is 2 years and 8 months and is seizure free and has achieved appropriate developmental milestones excepting expressive language skills.

Discussion: Here we report the first case of epilepsy in infancy with migrating focal seizures with KCNAB1 mutation and its successful treatment with topiramate.

PP:04**A CASE OF LENNOX-GASTAUT SYNDROME (LGS) FOLLOWING AUTOIMMUNE ENCEPHALITIS RESPONDING TO THE KETOGENIC DIET**

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Background: Epileptic encephalopathies are a group of disorders that lead to unrelenting seizures associated with cognitive and behavioural decline; Lennox-Gastaut Syndrome (LGS) represents a specific epileptic encephalopathy, displaying diverse seizure patterns and a distinctive electroencephalogram (EEG). The ideal management of LGS following autoimmune encephalitis (AIE) remains a puzzle as it is a rare entity, and the pathophysiology is uncertain. We report a case of post-encephalitis LGS responding to a ketogenic diet.

Case Presentation: A 5-year-old boy was diagnosed with AIE when he presented with typical EEG and clinical features without evidence of central nervous system infection. He developed super refractory status epilepticus and was managed with methylprednisolone pulses, intravenous immunoglobulins, plasmapheresis, and rituximab. He recovered with a modified Rankins score of 2 but was nonverbal and continued to have frequent atonic convulsions, progressive cognitive decline, and hyperactivity. 9-months post initial insult, the EEG was typical of LGS. There was no response to antiseizure drug adjustments, and methylprednisolone pulses. A ketogenic diet was initiated and after 5 months on the diet,

the patient showed no clinical seizures, no epileptiform activity on EEG, and notable improvement in cognitive and speech ability and behaviour.

Discussion: The case expands the literature regarding the development of LGS following AIE and highlights the possibility of successful remission with ketogenic diet even in those unresponsive to anti-seizure medications and immunotherapy.

PP:05**UNUSUAL MANIFESTATIONS OF JUVENILE DERMATOMYOSITIS (JDM)**

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Background: Juvenile dermatomyositis (JDM) is an inflammatory disorder characterised by muscle and dermatological manifestations. This report details the case of a 7-year-old girl with an unusual presentation of JDM.

Case Presentation: A 7-year-old girl, who had been experiencing excessive hair growth two months, along with a history suggestive of proximal muscle weakness for one month, presented with a sudden onset of right-sided hemiplegia. Dermatological signs included Gottron's papules, nail fold capillary abnormalities and an uncommon occurrence of hirsutism. Creatine Kinase level was elevated at 2397 U/L (normal range: 30 – 150 U/L). A Magnetic Resonance Imaging (MRI) scan of the brain, along with MR angiogram, revealed an infarction in the left middle cerebral artery (MCA) territory, accompanied by vessel narrowing indicative of vasculitis. Antinuclear antibody (ANA) was >1:80, and the Extractable Nuclear Antigen Antibodies (ENA) panel was negative. Treatment involved the administration of methylprednisolone, aspirin, azathioprine, and cyclophosphamide. 3 months later with the prescribed management and subsequent rehabilitation, the child showed improvement and is back to her normal activity without any further clinical events.

Discussion: JDM is a multisystem disorder where the pathology is a micro vasculitis.

Medium / large vessel vasculitis is an uncommon but documented characteristic associated with JDM. On rare occasions, JDM patients may exhibit focal weakness warranting consideration of cerebral vasculitis in their diagnostic evaluation. Cerebral vasculitis is rare with no clear guidelines for management of this complication. Hirsutism is a rare manifestation of JDM that can act as a sentinel sign.

PP:06**CDKL5 DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHY PATIENT PRESENTING WITH INFANTILE SPASMS****Prasadani TGM 1, Ratnayake P 1***1 Neurology Unit, Lady Ridgeway Hospital for Children, Colombo, Sri Lanka*

Introduction: CDKL5 developmental and epileptic encephalopathy (DEE) is a recently described genetic DEE of infantile onset, due to the mutation in the cyclin-dependent-kinase-5 gene.

With the advancement of genetic epilepsy panels, this is now a separate entity, characterized with early onset epilepsy, dysmorphism, movement disorders, hypotonia and corticovisual impairment (CVI).

We describe a patient who presented to us with infantile spasms with a classical clinical phenotype, who is genetically confirmed.

Case Presentation: A 4-month-old baby, first born to non-consanguineous parents with normal antenatal and birth history presented to us with infantile spasms. His developmental age was less than 6 weeks. He had had infantile tonic seizures started from 6 weeks onwards, which had gone unnoticed. His EEG was hypsarrhythmic and, he was commenced on the UKIESS regimen of prednisolone with minimal response. There after vigabatrin, topiramate, lamotrigine and levetiracetam were prescribed, with unsatisfactory seizure control. Later, the ketogenic diet was commenced, and he responded well. He had severe developmental delay, hypotonia and CVI phase 1 as associated factors. His metabolic workup and MRI brain was normal. He was genetically confirmed to have a CDKL5 gene mutation.

Discussion: CDKL5 genetic DEE is a separate disease entity with unique features. Epilepsy predominates the clinical phenotype starting as very early onset infantile tonic or tonic clonic seizures, as the first stage. Infantile spasms are the second stage, followed by myoclonic, tonic, absence, and atonic seizures. Seizures are usually pharmacoresistant and suggested drugs are topiramate, lamotrigine, valproate, vigabatrin and zonisamide. Other modalities of treatment include the ketogenic diet, vagal nerve stimulation and ACTH. Upcoming interventions in clinical trials includes cannabidiol and fefluramine.

Other associated features aiding diagnosis include dysmorphism, CVI, movement disorders, severe development delay and hypotonia.

Timely identification of this specific genetic DEE will guide precision care, and hence good epilepsy and developmental outcomes.

PP:07

SCN8A MUTATION-RELATED EPILEPTIC ENCEPHALOPATHY AND MULTIPLE BONE FRACTURES**Thennakoon MSBTMMN 1, Wanigasinghe J 2**

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Background: SCN8A associated epileptic encephalopathy is one of the treatable causes of intractable epilepsy in infancy. SCN8A mutation is associated with a spectrum of clinical phenotypes, including cognitive deficits, movement disorders, muscle atrophy, bone loss, and fractures. Severe bone loss has been previously described in a child and also demonstrated in mouse models of SCN8A mutation. The pathophysiology of SCN8A mutations related to skeletal complications is still not clearly known.

Case Presentation: These monochorionic diamniotic (MCDA) twin children were born to consanguineous parents without perinatal complications. They were developmentally normal until 6 months of age when they developed focal tonic and sequential seizures; followed by development of epileptic spasms. The clinical phenotypes of both children were the same, with tonic seizures followed by developmental regression. The focal tonic seizures did not respond to a combination of sodium valproate and clonazepam. However, treatment with phenytoin loading dose resolved the tonic seizures. The spasms were treated with oral prednisolone and seizures settled for the next 5 months. However, recurrence of seizures resulted in regression.

At 1.5 years, they presented with spontaneous bone fractures involving bilateral femurs. Clinical examination

revealed blue sclera with dental enamel hypoplasia in both twins suggesting a diagnosis of osteogenesis imperfecta. The serum Calcium was low; Phosphate level was elevated. ALP 247 U/L(122-470U/L) Vitamin D 24 ng/mL(>20ng/mL), PTH 35pg/ml(10-55pg/ml). The skeletal survey suggested osteogenesis imperfecta. The DEXA scan revealed combined trabecular and cortical bone loss. IV bisphosphonate, calcium, and vitamin D supplements stopped further fractures. Whole exome sequencing in one of the twins revealed a pathogenic variant in exon 27 of the SCN8A gene as a missense mutation, denoted as C.5615G>A at the cDNA level. At last review, they remained seizure free on sodium valproate and topiramate.

Discussion: SCN8A gene mutation in DEE 13 is associated with epileptic encephalopathy. Skeletal complications, and systemic involvement warrant active assessment for bone fractures and osteopenia.

PP:08

INCIDENTAL DETECTION OF TWO CASES OF PYRIMIDINE DEGRADATION PATHWAY DISORDERS BY URINE ORGANIC ACID ANALYSIS USING GAS CHROMATOGRAPHY MASS SPECTROMETRY

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Background: Dihydropyrimidine dehydrogenase (DPD) and dihydropyrimidinase (DHP) deficiencies are two rare congenital disorders affecting degradation of pyrimidines, uracil and thymine, with a varied clinical spectrum from asymptomatic cases to neurological manifestations. We report two cases of DPD and DHP, presenting with neurological manifestations, detected incidentally by urine organic acid assay using gas chromatography mass spectrometry (GC-MS) in the department of chemical pathology, Lady Ridgeway Hospital for Children. GC-MS technique for urine organic acid assay though primarily used to detect organic acidurias, can identify some polar compounds indicating pyrimidine degradation pathway defects.

Case Presentation:

Case 1: A 7-month-old baby girl born to consanguineous parents with a low birth weight (1.76 kg, <-3SD) and microcephaly (28 cm, <- 3SD) was investigated for global developmental delay and hypertonia. MRI brain revealed generalized severe cerebral atrophy while the EEG was normal. High uracil and thymine with no dihydrouracil and dihydrothymine in the urine organic acid profile confirmed the diagnosis of DPD deficiency.

Case 2: A 15-day-old boy of consanguineous

parents with poor sucking and hypotonia developed frequent apnoeic attacks since the 4th day of life. His urine organic acid profile revealed uracil, thymine and dihydrouracil confirming DHP deficiency supported by a missense homozygous likely pathogenic variant in the DPYS gene.

Discussion: There is limited awareness about inborn errors of pyrimidine pathway defects (PPD). Urine organic acid analysis should be requested in patients with unexplained neurological presentations. GC-MS technology plays a minor role in the diagnosis of purine degradation pathway defects hence a laboratory identified as a centre for rare disease detection should be armed with equipment to detect more PPD.

PP:09

TETRABENAZINE RESPONSIVE MYOCLONUS DYSTONIA DUE TO DYT 11 MUTATION**Munasighe H 1, Sanjaya Fernando 1,2**

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Background: Myoclonus-dystonia is a movement disorder that typically affects the neck, torso, and arms. Individuals with this condition experience quick, involuntary muscle jerks or twitches (myoclonus). About half of individuals with myoclonus-dystonia develop dystonia, which is involuntary tensing of various muscles that causes unusual positioning. In myoclonus-dystonia, dystonia often affects one or both hands, causing writer's cramp, or the neck, causing the head to turn (torticollis).

Case Presentation: A 3-year-old male child presented with abnormal jerky movements in both upper limbs and difficulty in walking for 4 months. He was the 1st child of non-consanguineous healthy parents. Antenatal history was uneventful and he was delivered by elective Caesarean section at 38 weeks of gestation with 2.5kg of birth weight, and had no perinatal or post natal complications.

He was developmentally normal up to 2 years and 2 months of age then developed recurrent falls due to left side lower limb weakness. Then gradually developed jerky movements in bilateral upper limbs affecting fine movements.

Neurological examination was clinically normal. His brain imaging, electroencephalogram (EEG), nerve conduction studies and basic metabolic

studies were normal. These movements responded poorly to Syndopa and clonazepam. Whole exome sequencing revealed a DYT11 mutation in the SGCE(epsilon-sarcoglycan) gene. Then tetrabenazine was started and jerky movements were significantly improved.

Discussion: This case history is important due to the clinical identification of the DYT11 mutation and guidance on effective management strategies in the context of dystonia.

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PP:10**A RARE VITAMIN-RESPONSIVE DEVELOPMENTAL AND EPILEPTIC ENCEPHALOPATHY DUE TO PACS 2 MUTATION****Munasinghe H 1, Sanjaya Fernando 1,2**

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Background: PACS2 (phosphofuran acidic cluster sorting protein 2) gene variation is inherited in an autosomal dominant manner. It often begins with epilepsy within 1 week after birth. It is accompanied by facial abnormalities and varying degrees of developmental delay. PACS2 is involved in membrane trafficking, apoptosis and autophagy.

Here we describe a case history of drug resistant epilepsy which was caused by PACS2 gene variation.

Case Presentation: A 12-year-old child presented with multi drug resistant epilepsy. He was the 1st child of nonconsanguineous healthy parents. The antenatal period was unremarkable. He was delivered by an elective Caesarean section at 40 weeks of gestation, and had no perinatal or postnatal complications.

He was well up to 2 months of age and then developed right sided tonic clonic convulsions lasting 1 minute, which were followed by post-ictal drowsiness for 45 minutes. Seizure burden was 1-2 episodes per day which was complicated with global developmental delay. EEG revealed epileptic encephalopathy. MRI did not reveal an abnormality. He was treated with multiple antiepileptics including steroids for 11 years but had poor response. Whole exome sequencing revealed PACS2 gene mutation. Then folinic acid was added with pyridoxine.

After that seizure burden was decreased by 50%. and quality of life improved significantly according to QOLCE-55 (Quality of life in Childhood Epilepsy Questionnaire).

Discussion: This case history is important for clinical identification of the PACS2 gene mutation and guiding effective management strategies in the context of drug resistant epilepsy.

PP:11

PARANEOPLASTIC MYELOPATHY: A DIAGNOSTIC DILEMMA IN LONG SEGMENT TRANSVERSE MYELITIS.**Wanasinghe WAK 1, Ranasinghe KMIU 2, Fernando A 2**

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Background: Non-classic manifestation of paraneoplastic myelitis is rare compared to the classic paraneoplastic neurological symptoms, and even more rarely presents as long segment transverse myelitis. The aim of presenting this case is to discuss the diagnostic dilemma that occurs due to paraneoplastic myelopathy when it presents in the early stages of a tumour.

Case Presentation: 41-year-old lady with diabetes for 6yrs ,presented with back pain for 2 weeks followed by bilateral lower limb numbness, weakness, urinary retention, and bowel involvement over 1 day duration. She had a history of low-grade fever over 2 months duration prior to the onset of symptoms. Examination revealed bilateral lower limb spastic paralysis with a positive Babinski sign and a sensory level at T10. Long segment transverse myelitis involving C6 to D6 was noted on Magnetic Resonance Imaging (MRI) with associated right mediastinal lymphadenopathy. Other investigations are as follows: slightly elevated inflammatory markers with negative screening for autoimmune and infectious aetiologies. Cerebrospinal fluid analysis revealed protein 81mg/dL, lymphocytes 65, with normal cytology. The lymph node biopsy showed reactive nodes and the bone marrow study was normal.

She was mobile with improvement in bladder and bowel function following 5 doses of

intravenous methylprednisolone. On follow-up, 2 months later she complained of evening pyrexia and abdominal pain. Reimaging with contrast enhanced computed tomography (CECT) abdomen revealed enlarged previously noted lymph nodes with hepatomegaly and splenic lesions. Her erythrocyte sedimentation rate (ESR) was elevated and repeat lymph node biopsy confirmed nodular lymphocyte predominant Hodgkin's lymphoma with possible transformation into diffuse large cell lymphoma.

Discussion: Paraneoplastic myelitis can be overlooked during evaluation due to its rarity and the nonspecific nature of the investigation findings. This case underscores the significance of considering rare possibilities when faced with a diagnostic dilemma and emphasizes the importance of close monitoring for the emergence of new symptoms, even after initial clinical improvement.

PP:12**UNILATERAL 7TH NERVE PALSY AS THE CLINICAL PRESENTATION OF MALIGNANT HYPERTENSION: THE IMPORTANCE OF BLOOD PRESSURE MONITORING IN PAEDIATRIC POPULATION WITH NEUROLOGICAL SYMPTOMS.****Wanasinghe WAK 1, Govinda DPS 1, Gunasekara V 1, Jagoda J 1, Weerasekara K 1***1 Lady Ridgeway Hospital for Children, Colombo, Sri Lanka*

Background: Lower motor type 7th nerve palsy is a common clinical presentation in paediatric population and about 80% present with Bell's palsy with unknown etiology. Malignant hypertension can account up to about 8% as an etiology, secondary to renovascular stenosis, congenital malformations of the urinary system, coarctation of the aorta, or endocrinopathies. In those cases, the child might only have the 7th nerve palsy without any other symptoms or signs of secondary etiology.

Case Presentation: A 4-year-old was admitted with left-sided lower motor type facial nerve palsy. On the day of admission, he developed epistaxis. Child had frequent hospital visits for recurrent episcleritis and had constitutional symptoms over a 6-month duration. On examination, the child had stage 2 hypertension with a blood pressure reading of 220/180 mmHg. There was no pulse difference in all four limbs. The cardiovascular system and eye examination were normal. The Renal Doppler study revealed renal artery stenosis in the right kidney and upper pole of the left kidney, confirmed by a computer tomography renal angiogram. Other vessel involvement was excluded through MRA, MRV and angiogram of thoracic vessels. Inflammatory markers were normal, and infections were ruled out.

The diagnosis of Takayasu arteritis was made with radiological findings and

managed with methotrexate, intravenous methylprednisolone pulses, and later with infliximab. Blood pressure control was achieved with four types of antihypertensives.

Discussion: In our patient, the key indicator for blood pressure monitoring was epistaxis, which facilitated the early diagnosis of malignant hypertension. While arterial hypertension is a known cause of lower motor 7th nerve palsy, the literature indicates a median delay in diagnosis of approximately 45 days. Another concern is that in cases of misdiagnosis, a child might be prescribed steroids as a treatment for Bell's palsy, potentially exacerbating hypertension and leading to a catastrophic event.

PP:13

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME (PRES) DUE TO TWO UNUSUAL CAUSES

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Background: Posterior reversible encephalopathy syndrome (PRES) is a syndrome of heterogeneous aetiologies characterised by altered mental status, headache, seizures, and visual changes with white matter vasogenic oedema in the posterior cerebral hemispheres on neuroimaging. We present two cases of PRES with two unusual aetiological factors.

Case Presentation:

Case 1: A 42-year-old female on treatment for uterine fibroids with Gonadotrophin Releasing Hormone (GnRH) agonists (Goserelin) presented with acute onset, thunderclap headache one week after the sixth dose of GnRH agonist. On admission, she developed a generalised tonic-clonic seizure. Blood pressure was 228/119mmHg. Magnetic Resonance Imaging (MRI) of the brain showed bilateral symmetrical T2/FLAIR hyperintensity white matter changes in the occipital lobes and minimal similar small areas in the posterior parietal lobe white matter bilaterally.

Case 2: A 43-year-old female with menorrhagia and uterine fibroids presented with a haemoglobin of 5.3 g/dL. Three blood transfusions were given over three consecutive days.

One and a half hours after the final transfusion, the patient developed a headache. Blood pressure was 150/100mmHg with bibasal crepitations in the lungs. The patient was managed as transfusion-associated circulatory overload with diuretics. Five days after the initial onset of the headache she developed blurring of vision, worsening headache, and three episodes of generalized tonic-clonic seizures. MRI brain showed bilateral symmetrical T2 high signal intensity involving subcortical and deep white matter of occipital, parietal & paracentral gyri indicative of PRES.

Discussion: GnRH agonist and blood transfusion related PRES is a very rare presentation which clinicians need to be aware of.

PP:14

ACUTE ISCHAEMIC STROKE FOLLOWING HYMENOPTERA STINGS**Palliyaguruge RC 1, Weerasinghe NT 1, Kumara UGC 1, Hordagoda HL 1, Peiris PJP 1***1 The National Hospital Kandy, Kandy, Sri Lanka*

Background: Wasps and honey bees belong to the order Hymenoptera and their stings are commonly encountered in the daily life of a Sri Lankan villager. Although allergic reactions are well documented, neurological complications such as acute ischaemic stroke (AIS) are exceptionally rare. This case series reports two distinct cases of AIS following hymenoptera stings, highlighting the potential for this severe complication and the need for further investigation into its underlying mechanisms.

Case Presentation:

Case 1: A 58-year-old non-smoking gentleman developed sudden right-sided hemiplegia and aphasia within one hour of receiving stings from about 100 wasps in the jungle. Imaging confirmed acute infarctions in the external border zone areas on the left side of the brain. Extensive investigations revealed no pre-existing risk factors for stroke and the patient received standard stroke therapy with rehabilitation.

Case 2: A 64-year-old tea-plucker presented with sudden onset left-sided hemiplegia and dysarthria following stinging of approximately 20 honey bees. She had angioedema of the face with possible laryngeal oedema requiring supplemental oxygen in the initial few hours following admission. A non-contrast computed tomography (NCCT) scan of her brain

revealed a right-sided lenticular striate artery territory infarction. She had no prior history of stroke or any other risk factors. She was given steroids in addition to standard stroke care.

Discussion: The exact mechanism behind the development of ischaemic stroke following hymenoptera stings is poorly understood. Several potential pathways have been described which include; direct vascular toxicity of venom components inducing vascular inflammation, vasoconstriction and platelet aggregation, immune-mediated endothelial dysfunction and vascular damage and venom-induced dysrhythmias contributing to stroke development. This case highlights the need to increase awareness among physicians to consider the possibility of AIS in the setting of neurological deficits following a hymenoptera sting even in the absence of traditional risk factors and the importance of rapid recognition and timely intervention to improve outcomes in AIS.

PP:15

ACUTE MOTOR AND SENSORY AXONAL NEUROPATHY (AMsan) PRESENTING WITH T8 SENSORY LEVEL – A RARE CASE REPORT

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Background: Guillain-Barre Syndrome (GBS) is an acute immune-mediated disorder affecting the peripheral nervous system, causing acute flaccid paralysis. While classically recognized by ascending motor weakness, GBS rarely presents atypically with profound sensory level involvement, challenging diagnostic norms, and leading to diagnostic and management delays. The Acute Motor Sensory Axonal Neuropathy (AMsan) variant of GBS, presenting with a sensory level, is an extremely rare occurrence..

Case Presentation: A 63-year-old Sri Lankan female, diagnosed with hypertension and ischaemic heart disease, was admitted with progressively worsening lower limb numbness and weakness over 6 days. By the 5th day, she developed bilateral hand numbness and urinary retention. There was no preceding history of infection. Neurological examination revealed bilateral flaccid paraparesis, generalized areflexia, and a sensory level at T8. The rest of the neurological examination and other system examinations were normal. The nerve conduction study demonstrated intact bilateral sural nerve sensory responses with absent median and ulnar responses. There were absent common peroneal motor responses and F-wave abnormalities supporting a diagnosis of AMsan. Cerebrospinal fluid revealed albuminocytological dissociation. Magnetic

Resonance Imaging (MRI) of the whole spine was unremarkable except for L1-L5 diffuse disc bulging causing mild canal stenosis. The patient showed a dramatic response to intravenous immunoglobulin (IVIg) and further improvement with physiotherapy, experiencing mild residual motor weakness in bilateral lower limbs.

Discussion: In AMsan, as in AMAN, the pathology is consistent with immune-mediated primary axonal injury at the node of Ranvier, with the difference being that both the dorsal and ventral roots are affected. It can be hypothesized that the initial inflammation at the nerve roots up to the level of T8 could have caused the symmetric T8 sensory loss in our patient clinically. This case emphasizes the need for awareness of atypical GBS presentations to improve patient care, highlighting clinical presentation, diagnostic challenges, and treatment outcomes.

PP:16**WHEN VISION TOOK A DIVE DUE TO A MISSION IN THE SINUSES- A CASE OF BILATERAL RHINOGENIC OPTIC NEURITIS CAUSED BY A SPHENOIDAL MUCOCELE PRESENTING AS AN ALTITUDINAL FIELD LOSS****Palliyaguruge RC 1, Hettige H 2, Pathirana G 2, Mohideen S 1**

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Background: Sphenoidal mucocoeles are rare complications of chronic sinusitis, that rarely can cause vision loss through optic nerve compression. Early diagnosis and intervention are crucial to prevent permanent impairment.

Case Presentation: A 64-year-old man presented with progressive bilateral vision loss, severe headache, and ptosis in the right eye for two months. Examination revealed complete vision loss in the right eye and limited vision in the left. He had ophthalmoplegia and an inferior altitudinal visual field defect. Fundus examination showed optic disc pallor, and magnetic resonance imaging (MRI) confirmed bilateral sphenoidal mucocoeles compressing the optic nerves with underlying bone expansion and remodeling.

Discussion: The patient underwent endoscopic sinus surgery, successfully draining and removing the mucoceles. Postoperatively, his vision improved significantly in both eyes, with headache relief and reduced optic disc pallor. Histopathological analysis confirmed chronic inflammation in the mucoceles.

This case highlights the importance of considering sinus pathology in individuals with sudden vision loss and headache, especially with atypical presentations like altitudinal field defects. Prompt collaboration between otorhinolaryngologists and ophthalmologists is crucial for optimal management and vision preservation.

PP:17

CONGENITAL MYASTHENIC SYNDROME (CMS) - COLQ MUTATION IN A CHILD**Selvakumar L 1, Sajeemala WG 1, Ganesan S 1, Wijesekara DS 1,2**

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Background: Congenital Myasthenic Syndromes (CMS) are a genotypically and phenotypically heterogeneous group of neuromuscular disorders, due to mutations of proteins in the motor end plate. Mutations occur de novo or can be inherited. They are characterized by fatigability and weakness. The onset varies from intrauterine to adolescence. The prevalence is not known as CMS is rare compared to the acquired type. In Sri Lanka, there are few reported cases in adults. We report the first case of COLQ-CMS diagnosed at three years of age in the Sri Lankan context.

Case Presentation: A 3-year-old girl presented with respiratory arrest, following a respiratory tract infection which required ventilatory support. Atracurium was used as a muscle relaxant. She was born to nonconsanguineous parents; but, isolated gross motor delay and poor weight gain were noted at six months. Stridor and swallowing difficulty were detected for which nasogastric tube feeding and physiotherapy were started. She was able to walk and take oral feeds at two years of age. She had ptosis, external ophthalmoplegia, and unequal pupil sizes. There was central hypotonia, fatigable proximal muscle weakness, reduced deep tendon reflexes with normal gait. The spine and other systems were normal. Sustained upward gaze and single breath count to twenty demonstrated fatigability.

Electromyogram (EMG) showed a decremental pattern. Pyridostigmine worsened the weakness and was replaced by salbutamol to which she responded. Whole Exon Sequencing (WES) revealed CMS with a COLQ mutation.

Discussion: Twenty two CMS disease genes have been identified to date. Mutations in the acetylcholinesterase (AChE) collagen-like subunit gene, COLQ, are the most common causes of synaptic CMS. This leads to deficient AChE. Most patients are disabled early with respiratory difficulties and axial muscle weakness. Common triggers for relapses are infection, esterase inhibitors and puberty. DOK7 and slow channel CMS may mimic COLQ-CMS. Muscle relaxants are used cautiously as the action of AChE is unpredictable. Atracurium is used in children due to its noncumulative metabolism. Succinylcholine is contraindicated.

Genetic diagnosis is essential for appropriate drug selection. Pyridostigmine worsens symptoms in COLQ CMS, as in our patient. Clinical improvement has been reported with ephedrine and/or salbutamol. Our patient responded to salbutamol with improved exercise tolerance, but external ophthalmoplegia remained with minimal disturbance to activities of daily living.

PP:18

SUBACUTE COMBINED DEGENERATION OF THE SPINAL CORD PRESENTING AS A GUILLAIN-BARRE SYNDROME (GBS) MIMIC.**Prasadani TGM 1, Ratnayake P 1***1 Neurology Unit, Lady Ridgeway Hospital for Children, Colombo, Sri Lanka*

Background: Subacute combined degeneration of the spinal cord (SACDS) is a disease affecting the lateral and posterior columns of the spinal cord, primarily due to demyelination. It is a neurological complication of vitamin B12 deficiency. We present a nine-year-old girl who presented with ascending motor paralysis and sensory symptoms, who responded to treatment as for GBS and diagnosed as SACDS subsequently.

Case Presentation: A nine-year-old apparently healthy girl presented with ascending type motor weakness over ten days. Her examination confirmed lower limb more than upper limb proximal more than distal weakness with absent reflexes. Her initial sensory examination was normal. Nerve conduction studies showed only abnormal F waves in motor recordings.

She developed inability to stand without support and was treated for GBS with intravenous immunoglobulin (IVIg) and improved markedly, becoming ambulatory without assistance. Over the next five days, she rapidly deteriorated and plasmapheresis was undertaken, with no improvement. By day twenty, her clinical picture evolved, and she developed paraesthesia involving lower limbs, her gait was ataxic, and her joint position sensation became impaired. On inquiry, she was a vegan for several years and started consuming animal products a week prior to the onset of weakness. Her

vitamin B12 level was 101.7pg/ml (200-800). Her MRI spine confirmed the diagnosis of SACDS. There was no hematological involvement.

We prescribed, intramuscular vitamin B12 100micrograms weekly injections for a month and once a month thereafter with dietary management, and she recovered fully by four months.

Discussion: SACDS, is a rare diagnosis and its presentation can vary. This case was unusual for the acute onset of isolated motor manifestations with areflexia which is attributable to motor neuropathy at presentation and the initial response to IVIg which may point to an inflammatory accompaniment of B12 deficiency-associated neuropathy.

PP:19

LONGITUDINALLY EXTENSIVE TRANSVERSE MYELITIS (LETM) AS THE SOLE MANIFESTATION OF *Neisseria meningitidis* INFECTION**Gunawardane IKPS 1, Pathirana KD 1,2, Piyasiri DLB 1, De Zoysa WD 1,2, Kehelovitagama KOV 1**

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Background: Acute myelopathy secondary to meningococcal meningitis is considered a rare complication that is limited to a few reported cases all of which occurred in the presence of typical symptoms of meningitis and signs of meningeal irritation. In contrast, myelopathy appearing as the sole manifestation of *Neisseria* infection is extremely rare.

Case Presentation: A 70-year-old previously healthy male presented with fever for three days and acute onset lower limb weakness with urinary retention. On examination, he was febrile, with stable vital parameters. Neurology examination revealed flaccid paraparesis with a sensory level at T2 which progressed to C4 level within 12 hours from the onset. Magnetic Resonance Imaging (MRI) revealed features of longitudinally extensive transverse myelitis extending from the cranivertebral junction to the T4 spinal level. His cerebrospinal fluid (CSF) showed neutrophilic pleocytosis (2560) with an elevated protein level. Both gram stain and culture of CSF were normal, but CSF was positive for *Neisseria meningitidis* antigen. He was treated with IV Ceftriaxone and a course of IV methylprednisolone followed by 4 cycles of plasma exchange with marked improvement. Unfortunately, he succumbed to death on day 12 of hospital admission due to hospital-acquired pneumonia.

Discussion: *Neisseria meningitidis* is considered one of the most frequent pathogens causing bacterial meningitis which may typically present with fever, headache, vomiting, and neck stiffness. Without timely treatment, it can result in complications, including meningococcal sepsis, obstructive hydrocephalus, or cranial nerve palsies. However acute spinal cord dysfunction secondary to *Neisseria* infection is considered rare. Most of such reported cases have first had meningitis then spinal cord dysfunction as a sequel. Up to now, only one case has been reported in the literature to have acute myelitis as the sole manifestation of *Neisseria* reported by Ibrahim et al. in Doha, Qatar.

PP:20

NEUROLEPTIC MALIGNANT SYNDROME IN ANTI-N-METHYL-D-ASPARTATE RECEPTOR ENCEPHALITIS – A CASE REPORT

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Background: Anti-N-methyl-D-aspartate receptor (anti-NMDAR) encephalitis is a difficult diagnosis presenting with neuropsychiatric manifestations. Neuroleptic malignant syndrome (NMS) not only share most of the features with anti-NMDAR encephalitis, but also it can develop as a consequence of antipsychotic use in these patients.

Case Presentation: We describe a 30-year-old male with no previous psychiatric history, who initially presented to the Psychiatry unit (PU) with aggressive behaviour and altered level of consciousness (LOC) and was given antipsychotics. He developed one generalised tonic clonic seizure and reduced LOC and was transferred to our neurology unit. High serum creatinine phosphokinase (CPK) and creatinine were detected at the PU before the seizure. On admission, he had a high fever, generalised muscle rigidity, and hyperreflexia. He was managed at the intensive care unit with respiratory support, intravenous meropenem, and aciclovir and treated as for NMS with hydration and bromocriptine. Further history revealed that he initially had a low-grade fever and headache with behavioural abnormalities. Despite the initial management, he had persistent fever with reduced LOC and developed orofacial dyskinesia. There was an ill defined T2 hyperintensity in the left insular cortex and external capsule with no contrast

enhancement or diffusion restriction in his initial magnetic resonance imaging (MRI) of the brain which was reduced in size and hyperintensity in the repeat MRI. His cerebrospinal fluid anti-NMDAR antibodies became positive. He had a marked improvement following initial immunotherapy and is currently doing well.

Discussion: As patients with autoimmune encephalitis with psychiatric symptoms are initially managed with antipsychotics they have a high chance of developing NMS. This further complicates the disease process leading to diagnostic difficulty and treatment delay. Timely identification and immunotherapy lead to better outcome. More studies are warranted to differentiate whether NMS is caused by the antipsychotics given or if it could be a feature of anti-NMDAR encephalitis.

PP:21

CRYPTOCOCCAL MENINGITIS IN IMMUNOCOMPETENT YOUNG ADULTS**Uvaim AAM 1, Fernando A 1, Jayakody JAA 1, Thineshan P 1**

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Background: Cryptococcal meningitis (CM) is a relatively rare infection with high mortality and morbidity. It is considered as an opportunistic fungal infection, classically described in immunocompromised patients. Cryptococcosis has been very rarely reported in young and otherwise healthy patients. Here we report 2 cases of CM in young healthy females.

Case Presentation:

Case 1 - A 24-year-old lady presented with fever, headache, altered behavior for 4 days duration. On examination, she had low GCS. Initially, she was managed for possible viral encephalitis. With poor response and cerebrospinal fluid (CSF), findings suggest pyogenic or tuberculous (TB) meningitis, antibiotics and anti-TB regimen were commenced. Her initial Magnetic Resonance Imaging (MRI) Brain was normal and her repeat MRI Brain showed extensive cerebral venous sinus thrombosis and bilateral infarcts. Despite ongoing treatment, she had a continuing fever with headache and a persistent CSF sugar drop. Her Cryptococcal antigen of CSF as well as of blood were positive. She was treated with amphotericin followed by oral fluconazole. She had improved with treatment and was investigated for an immunocompromised state which eventually became negative.

Case 2 - A 22-year-old lady presented with fever, headache, photophobia, and diplopia for 3 days duration. On examination, she

had a left 6th cranial nerve palsy. With herMRI and CSF findings, she was managed for pyogenic meningitis. Later on she developed seizures with a reduced level of consciousness. Her repeat CSF study showed a persistent sugar drop. An anti-TB regimen was commenced. Despite treatment, she had a continuing fever. She also was found to have hydrocephalus for which an external ventricular drain (EVD) was inserted. Her CSF for Cryptococcal antigen was positive and she was commenced on amphotericin. Her GCS had improved and her fever settled. She later on had obstruction of the EVD and had multi-drug resistant (MDR) acinetobacter and MDR enterococci in the CSF as well as blood cultures. Her repeat MRI had shown basal ganglia and large middle cerebral artery (MCA) infarcts with hydrocephalus. Despite aggressive treatment for sepsis she succumbed.

Discussion: The most common manifestation of cryptococcal infection in an immunocompromised patient is central nervous system (CNS) involvement. Cryptococcal meningitis in young, previously healthy patients is very uncommon. The diagnosis of CM is determined if the patient met any one of the following: CSF-positive culture, positive India ink, and positive cryptococcal antigen. Lack of awareness among treating clinicians and low index of suspicion leads to the diagnosis often being delayed or missed, resulting in poor prognosis with severe consequences.

PP:22**VALIDATION OF THE PROGRESSIVE PLANNING TEST (PPT) FOR USE IN THE ASSESSMENT OF CHILDREN WITH NEURODEVELOPMENTAL DISORDERS IN SRI LANKA.****Gunawardena AR 1, Dass G 1, Kodituwakku PW 2, Wanigasinghe J 3***1 Child, Adolescent and Family Services, Sri Lanka**2 Department of Paediatrics, University of New Mexico School of Medicine, USA**3 University of Colombo, Colombo, Sri Lanka*

Objectives: The Progressive Planning Test (PPT) is a “look-ahead puzzle” developed to assess cognitive planning. The main objectives of the current research were to test the following hypotheses: 1. The test performance increases linearly with age until adolescence; 2. The effect of age varies by the difficulty level of the problems, with only the problems of higher levels (levels 2 and 3) showing age differences; and 3. Solving level 2 and 3 problems involves the dynamic interplay between motor and higher cognitive processes (frontal).

Methods: Participants: Sixty-seven children, 33 girls and 34 boys ranging in age from 6 to 17 years, participated. They were typically developing children who met the following exclusionary criteria: known neurological illness, traumatic brain injury, neurodevelopmental disorders, and significant psychiatric problems.

The PPT was administered individually as part of a larger neuropsychological test battery. In this test, a participant was asked to solve 10 planning problems of graded difficulty progressing through 3 levels.

Results: The participants were grouped into 3 age categories and the planning problems, into 3 levels. Oneway ANOVA showed that average total score earned by the participants increased with age linearly, F

$(2,65) = 12.24$, $p < .001$. Repeated measures factorial ANOVA revealed a significant interaction between age and level of difficulty, $F (2,64) = 5.97$, $p = .004$, indicating only level 2 and 3 problems discriminated between the 3 age groups, confirming hypothesis 2. The third hypothesis was also confirmed, indicating the influence of motor habits in cognitive planning.

Conclusions: The current findings on the PPT are in line with the results obtained in other countries, thus attesting to its construct validity. Therefore, the PPT can be used to assess the executive functioning of children in Sri Lanka. This test can also be used to assess adults with various neurological conditions like leukoencephalopathy and Parkinson disease.

PP:23**QUALITY OF CARE & LEVEL OF SATISFACTION AMONG CAREGIVERS OF CHILDREN ATTENDING PAEDIATRIC NEUROLOGY CLINICS IN A TERTIARY CARE HOSPITAL IN COLOMBO, SRI LANKA****Shahnaz MAF 1,Wijesekara DS 2 Madhubhashini JASD 1***1 Colombo South Teaching Hospital, Kalubowila, Sri Lanka**2 Department of Paediatrics, Faculty of Medical Sciences, University of Sri Jayewardenepura, Sri Lanka*

Background: The Colombo South Teaching Hospital (CSTH) is a leading tertiary care center with two crucial clinics - Multidisciplinary Neurodevelopmental Clinic and Paediatric Neurology Clinic. Despite limited resources and time, both clinics serve a large number of patients. Thus, it is essential to assess caregiver satisfaction for improved care quality.

Objectives: To evaluate the quality of care and satisfaction level of caregivers of children who attend the Multidisciplinary Neurodevelopmental Clinic and Paediatric Neurology Clinic.

Methods: Interviewer administered questionnaires were given to caregivers attending the above clinics at CSTH over 2 months.

Results: Fifty carers were interviewed. Most children were boys (68.8%) and aged 5-10 years & 1-2 years. The primary caregivers are mothers. who accompany children for early intervention due to prematurity, complicated neonatal period (32%), cerebral palsy (18%), and epilepsy (24%). Parents expressed high satisfaction levels during registration (65%), with supporting staff (65%), nursing staff (71%), and doctors (79%), despite waiting for 30 minutes to an hour. The study highlighted the provision of excellent services through the best counselling

with a good insight at 64.6% & timely referrals for evaluation of vision (84.8%), hearing (77.3%), swallowing assessment (40%), and oral-hygiene (31%). Satisfaction rates for physiotherapy (88%), speech and language therapy (80%), and occupational therapy (79%) emphasizes the top-notched care.

Conclusions: Based on the study, parents reported that they were more satisfied with the quality of care provided by both clinics. It also highlighted the importance of extending high-quality care, including counseling, complication assessment, appropriate interventions, and effective therapies. Compassionate care and efficient therapies have played a vital role in minimizing complications, allowing children to maintain their age-appropriate abilities, thus making early intervention more successful.

PP:24

COMPARISON OF URINE GLYCOSAMINOGLYCAN EXCRETION BETWEEN CHILDREN WITH AUTISM SPECTRUM DISORDER AND TYPICALLY DEVELOPED CHILDREN

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Objectives: The objective of the present study was to compare urine excretion of sulfated glycosaminoglycans (GAG) in children with autism spectrum disorder, with neurotypical controls.

Methods: Random urine samples were collected from children with autism spectrum disorder ($n=61$) between the ages of 2 and 6 years, and age- and sex-matched neurotypical controls. Urine glycosaminoglycan levels were quantified by the dimethylmethylen blue (DMMB) dye-binding assay, using a microplate reader.

Results: Urine GAG levels are significantly higher ($p = 0.026$) in the ASD group when covariates such as age, weight, urinary creatinine, and height are taken into consideration by ANCOVA. In neurotypical subjects, the urine glycosaminoglycan levels appear to decline with age, height, and weight while this trend was not apparent in subjects with autism spectrum disorder.

Conclusions: Children with ASD exhibit higher urine GAG excretion. Further research is needed to explore the molecular basis.

PP:25**USE AND MISUSE OF URGENT EEGS IN PAEDIATRIC PRACTICE; AN AUDIT DONE IN A TERTIARY CARE CHILD NEUROLOGY CENTRE IN SRI LANKA****Dantanarayana C 1, Dharmadasa T 1, Weerapperuma G 1, Nandasiri S 1, Lakmal P 1, Fernando S 1,2**

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Introduction: Electroencephalography (EEG) is a non-invasive investigation done to analyze the electrical activity of the brain. This audit aimed to investigate the true necessity of urgent EEG (uEEG) in paediatric patients at a tertiary care centre in Sri Lanka. The uEEG guidelines in this centre were formulated based on the 'EEG in acute Neurology' guideline of Harvard Medical School – MGH epilepsy services. Altered level of consciousness of different strata was taken as the prime indication to perform an uEEG in this guideline.

Methods: A retrospective cohort analysis was conducted on paediatric uEEG requests. uEEG request forms and reports were examined by two researchers who were blinded. The deviations from the predefined guideline were assessed.

Results: A total number of 812 EEGs were done during a period of 12 months, out of which 402 (49.5%) were uEEGs. The mean age for uEEG was 7.59 years (\pm 4.58 years). Clinical indications ranged from analysis of the first event 154 (38.3%), altered level of consciousness 103 (25.6%), breakthrough events 77 (19.2%), to seizure with fever 57 (14.2%). Abnormalities were detected in 122 (30.3%) of uEEGs; focal-interictal discharges 52 (46.4%), and epileptic-encephalopathy 23 (20.5%), were the commonest. uEEG done for breakthrough events had the highest

percentage of abnormal EEGs 37/77 (48.1%). Out of the nonurgent EEGs, 107 (26.1%) had abnormal reports. There was no significant difference between the percentages of EEG abnormality between uEEG versus nonurgent EEGs ($p = 0.18$).

Conclusions: Nearly half of the EEGs were done on urgent EEG requests. Assessment of the first event was the commonest indication. Altered level of consciousness was the indication only in a quarter and aligned with the guideline. Positive rates between EEGs done on urgent versus non-urgent basis did not show a significant difference. This audit, highlights the value of reiterating the EEG guideline among requestees to refine the requesting methodology.

PP:26

USE OF SCALP EEG IN CHILDREN WITH "FEVER PLUS SEIZURES", A SINGLE CENTRE RETROSPECTIVE COHORT STUDY**Dharmadasa T 1, Dantanarayana C 1, Weerapperuma G 1, Lakmal P 1, Fernando S 1,2**

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Introduction: The co-association of "Fever-Plus-Seizures" included febrile seizures, Central Nervous-System (CNS) infections and first epileptic seizure or a relapsed epileptic seizure triggered by fever. A correct differential diagnosis of "Fever-Plus-Seizures" leads to appropriate management. Scalp electroencephalogram (EEG) is one investigation which is being requested in this clinical context. Current study was done to determine the use of scalp EEG records in children with "Fever-Plus-Seizures".

Methods: A retrospective collection of data was done, from November-2022 to December-2023. EEGs requested for "Fever-Plus-Seizures" between the ages of 1month – 16 years were included. EEG request forms and reports were independently analysed by the researchers.

Results: A total number of 139 EEGs were done for "Fever-Plus-Seizures" over a period of 12-months, 97/139 (69.8%) were done on urgent requests. The mean age 5.3 years (± 3.6) and males 89 (64%). The indications were febrile-seizures in 119 (85.6%), out of which 49 (41.2%) had complex-febrile seizures, 36 (30.2%) had simple-febrile-seizures and 34 (28.5%) had late-onset-febrile-seizures (febrile-seizures occurring after an age of 5 years). In the febrile seizure group EEG abnormalities were noted in 11 (9.2%) of which 06 had postictal background

slowing. 08 (16.3%) patients with complex-febrile-seizures had EEG abnormalities, (interictal-epileptiform-abnormalities = 04). One with late-onset-febrile-seizures had interictal-epileptiform-abnormalities.

Epileptic seizure triggered by fever is the indication in 11 (7.9%), with EEG abnormalities reported in 27.2%, (postictal-slowing = 01, interictal-epileptiform-abnormalities = 2). 9 (6.5%) scalp EEGs were done for clinically suspected CNS infection and abnormalities noted in 22.2%.

Conclusions: In a wide majority with "Fever-Plus-Seizures" indication for scalp EEG was febrile seizures, and a quarter of the sample had simple-febrile seizures. Only a minority of the EEGs done for febrile seizures had abnormalities. Very few of the complex-febrile-seizure group had interictal epileptiform-abnormalities. Scalp EEGs requested for the analysis of CNS infection and for seizures triggered by fever are minimal.

PP:27**A STUDY ON SLEEP DISTURBANCES IN CHILDREN WITH CEREBRAL PALSY****Fernando S 1, Nimalratne U 2, Hewage NN 3, Sirisena D 1***1 Department of Neurology, Colombo North Teaching Hospital, Ragama, Sri Lanka**2 Department of Paediatric Neurology, Teaching Hospital Kurunegala, Sri Lanka**3 Department of Paediatric Neurology, Teaching Hospital Anuradhapura, Sri Lanka*

Introduction: According to evidence children-with-cerebral-palsy (CWCP) are at a risk of having sleep disturbances. However, this has not been considered a sinister health issue up-to-date. Therefore, we intended to determine the frequency, types and associations of sleep-disturbances in CWCP.

Methods: Descriptive-cross-sectional study method was used; Sleep- Disturbance-Scale-for-Children (SDSC) was used as the tool, which is a 26-itemed parent reported questionnaire with a five-point scale (SDSC, T-score of >70 was taken as pathological). Trained doctor did rest of the assessments using validated tools.

Results: Sample comprised 400-CWCP; mean age 5.9years (+/-3.3year), males-229 (57.25%). Quadriplegic-Cerebral-Palsy (CP) n=163 (14.75%), Diplegic-CP n=130(32.5%), Hemiplegic-CP n=89 (22.25%). Out of the sample 181(45.25%) had at least one pathological sleep disturbance. Disorders-of-Initiating-Maintaining-Sleep(DIMS) is the commonest n=91(50.27%), which is identified more in 2-6 years (n=65, p=0.0105) versus >6years, Quadriplegic-CP (n=60, p=0.0001), Patients-with-Epilepsy (n=43, p=0.0001), higher Gross-Motor-Function-Classification-Scale ($p<0.0001$) higher Manual-Ability-Classification-Scale ($p<0.0001$), and higher Communication Function-Classification-System ($p<0.0001$).

Out of the cohort, n=133 were on antiseizure medication, with higher DIMS scores (n=48, $p = < 0.0001$). Sleep-Hyperhidrosis is seen in 60(33.15%), Sleep-Breathing-Disorders in 49(27.07%), Disorders-of-Arousal in 31(17.13%), Sleep Wake-Transition-Disorders in 30(16.57%), and Disorders-of-Excessive-Somnolence in 23 (12.70%).

Of the sample, 388(97.00%) co-share the bed with a family member, and sleep has not been discussed as a medical issue in 301 (75.25%). However, only a minority 66 (16.5%) considered sleep difficulties to have a negative impact on the quality of life of the family.

Conclusions: Nearly half of the cohort had at least a single sleep disturbance. Disorders-of-Initiating-Maintaining-Sleep is the commonest disorder, followed by Sleep-Breathing-Disorders and Disorders-of-Arousal. Sleep disturbance has not been discussed as a medical issue in a majority

PP:28**EPIDEMIOLOGY, AETIOLOGY AND VISUAL OUTCOME OF CHILDREN RECEIVING REHABILITATION SERVICES FOR CEREBRAL VISUAL IMPAIRMENT IN A TERTIARY CARE HOSPITAL**

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Introduction: Cerebral Visual Impairment (CVI) is the loss of visual function resulting from damage to structures in the posterior visual pathway (beyond the lateral geniculate body).

Our centre has been carrying out rehabilitation services for children with CVI for over 12 years. The analysis of the characteristics of the population and the outcome of intervention is undertaken as this information is essential to improved detection and service delivery.

Methods: A retrospective observational study was conducted to identify the epidemiology, aetiology, and visual outcomes of patients followed up at the CVI intervention clinic at the Lady Ridgeway Hospital for Children.

Results: Data was available for 485 patients. 56.4% of them were males. 36.1% were detected at the screening when they presented to the early intervention clinic at an age of less than 6 months. 65.6% were reported to have a perinatal insult with 56.1% having radiological evidence of hypoxic ischaemic encephalopathy (HIE). 50.6% had respiratory distress syndrome. 64.6% of the cohort had global developmental delay and 35.6% had an abnormal electroencephalogram (EEG).

Data for improvement following interventions was available in 216. Median improvement was 39.28%. Respective improvements of 25-

50%, >50-75%, and >75-100% were seen in (67) 31%, (49) 22.7% and (36) 16.6% of the cohort. The level of CVI at the first visit was associated with maturity (p value = 0.037), presence of HIE (p value = 0.037) & and age at the first visit (p < 0.005). The CVI score at the first visit predicted the outcome of interventions with a sensitivity of 90% and specificity of 80% (area under the curve 0.9, 95% CI 0.93-0.98, p <0.0001).

Conclusions: The dominant and potentially preventable cause for CVI was HIE. Neurological co-morbidities were seen in the majority which could point to CVI association that justifies screening. The usefulness of intervention was demonstrated with an improvement of 50% or more of the CVI score in more than one-third of the population, with this number increasing to more than two-thirds for an improvement of 25% or more.

PP:29

A DETAILED ANALYSIS OF PARENTAL PERCEPTIONS OF AUTISM SPECTRUM DISORDER (ASD): A TERTIARY CARE EXPERIENCE IN SRI LANKA

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Objectives: Raising a child with ASD can be an overwhelming experience for parents and families as Autism Spectrum Disorder (ASD) is characterized by poor social communication, repetitive behaviours and restricted interests. The objective of this study is to identify associations of parental perceptions of children with ASD, as it is of utmost importance to provide individualized parent and child-centered therapy to achieve a better outcome.

Methods: A descriptive cross-sectional study was conducted among 100 children with ASD meeting DSM V criteria (aged 2-17) at Teaching Hospital Kurunegala following ethical clearance from the Sri Lanka College of Paediatricians. Data was collected through an interviewer-administered questionnaire on parental perceptions using the Likert scale.

Results: The population comprised a mean and median age of 6.88 and 6 years respectively, with a male majority (76.4%). Parental perception of aetiology, social stigma, schooling, prospects and therapy were assessed. 63.5% of parents believed it is a disorder of the brain and 84% disagreed that it's familial in etiology. With regards to social stigma, 51% found it difficult to take their child to public places, and only 11% felt embarrassed. 76.5% and 65.3% of parents believed that their child can join school and communicate with others respectively. Only

16.5% of parents feared the child would never be able to find a job compared to 48.3% who thought otherwise. A majority (81.1%) of parents believed that doctors can improve the child's condition, of which 75.3% thought the available therapies are beneficial, but only 51.8% believed doing therapy at home was effective.

Conclusions: Getting a better understanding of parental perceptions will facilitate clinicians to deliver interventions that would yield higher parent satisfaction.

PP:30**EFFECTS OF A MEDITATION-BASED INTERVENTION ON NON-MOTOR SYMPTOMS OF PARKINSON'S DISEASE: A RANDOMISED CONTROLLED CLINICAL TRIAL****Vithanage KK 1, Dissanayake DWN 1, Chang T***1 Faculty of Medicine, University of Colombo, Colombo, Sri Lanka*

Objectives: Parkinson's disease (PD) is characterized by motor and non-motor symptoms (NMS). There is no disease-modifying therapy yet available for PD. Currently available symptomatic therapy often fail to optimally control NMS.

Thus, we explored the efficacy of an adjunct meditation-based intervention (MBI) on the NMS of PD in a randomized controlled trial.

Methods: Forty-six patients with PD (H&Y 1-3) were selected on convenient sampling and randomized to an interventional group (IG) and a usual-care-alone group (UC). IG underwent eight weeks of MBI in addition to their routine treatment. NMS were assessed in the two groups pre and postintervention using the SENS-PD (SEverity of Nondopaminergic Symptoms in Parkinson's Disease) scale. The SENS-PD assessed cognitive functioning (CF), psychotic symptoms (PSY), postural instability and gait difficulty (PIGD), excessive daytime sleepiness (EDS), autonomic dysfunction (ANSD), and depressive symptoms (DEP). (Data was analyzed using SPSS-29 software. Non-parametric tests were used to assess outcome significance

Results: In the IG there were 14 men and 9 women (mean age of 63.9; SD=6.6) years. In the UC group, there were 13 men and 10 women (mean age of 66.1, SD=6.7) years. The baseline characteristics of the two groups

did not differ significantly. In IG, SENS-PD scores showed a significant improvement postintervention in all assessed parameters except psychotic features [CF mean-rank IG=16.1, UC=28.2, (SE-40.8, p=0.002), PSY mean-rank IG=22.1, UC=21.9, (SE-39.7, p=0.98), PIGD meanrank IG=14.6, UC=29.8, (SE-40.4, p<0.001), EDS meanrank IG=18, UC=26.2, (SE-38.8, p=0.024), ANSD mean-rank IG=16.1, UC=28.2, (SE-40.2, p=0.001), DEP mean-rank IG=17.4, UC=26.8, (SE- 40.6, p=0.013)].

Conclusions: MBI is an effective adjunct in improving the NMS of PD.

PP:31

PREVALENCE OF PARKINSON'S DISEASE SYMPTOMS AND ITS EFFECT ON PATIENTS IN A DEVELOPING SOUTH ASIAN COUNTRY - A PATIENT'S PERSPECTIVE

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Objectives: Parkinson's disease is a neurodegenerative condition with a range of causes and clinical presentations. Though the initial observations were limited to motor manifestations, it became apparent that there are a whole array of non-motor symptoms, which significantly affect the quality of life of Parkinson's patients affecting individuals to varying degrees. This study aims to describe the prevalence of the motor and non-motor symptoms of Parkinson disease and how the patients quality of life (QOL) and activities of daily living (ADL) are being affected.

Methods: This is a descriptive cross-sectional study with an interviewer-administered questionnaire for patients diagnosed with Parkinson's disease according to the UK Brain Bank criteria attending the neurology clinic at the National Hospital of Sri Lanka for one year duration. Patients diagnosed with major psychiatric illness were excluded. Patients were asked to rank the impact of symptoms on their quality of life.

Results: Of the 192 patients, the majority were males (58.3%; n=112). Mean age of the population was 64.09 ± 8.69 years. 92.7% (n=178) were married. 78.6% (n=151) had secondary education. 82.3% (n=158) rated that Parkinson's disease had

impact on employment and earning capacity. The mean age of onset of Parkinson's was 59.5 ± 9.87 years. The mean duration of Parkinson's disease was 4.62 ± 4.25 years. Of 192 patients, 97% (n=186) reported at least two non-motor symptoms, with fatigue (88%; n=169) and pain (74%; n=142) being identified as the most bothersome. About 83% (n=160) screened positive for anxiety, 40% (n=76) for depression. Daytime sleepiness was present in 68.7% (n=132) and insomnia in 65.1% (n=125)

Conclusions: Our findings provide evidence for the diversity of experience with both motor and non-motor symptoms affecting the patient's quality of life. This highlights the importance of managing both motor and non-motor symptoms to preserve the quality of life of patients with Parkinson's disease.

PP:32

A NOVEL GAME PLATFORM FOR IMPROVEMENT OF HAND FUNCTIONS IN STROKE REHABILITATION

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Introduction: Stroke is a debilitating condition. Traditional rehabilitation is often costly and geographically limited, prompting exploration into novel stroke rehabilitation methods.

Objectives: The objective is to create a game platform using motion tracking, tailored to individual patient needs and track the progress of the rehabilitation.

Methods: Here we integrated motion tracking technology, employing OpenCV and Python to capture and process patient hand movements. Hand gestures were integrated for control of three off-the-shelf games. These were transmitted to the Unity game engine, enabling the generation of realistic avatar movements mapped to the hand gestures. Visual feedback was enabled and different levels of difficulty used to track progression.

Results: Three distinct games were developed. Game 1 "rock pick up" requires the patient to sweep rocks on the screen using their palms. The difficulty is increased by placing rocks closer in proximity. Game 1 targets palm and wrist improvement across three escalating difficulty levels. Game 2 "running man" focuses on enhancing fingers and wrist movements. Improvements of each

of the fingers in both hands can be analysed. Once the finger digit is selected for playing, the game ignores other digits and only focuses on the specified digit. The difficulty can be changed by changing the speed of the avatar. In Game 3 "roll a ball", patients should virtually push the ball using the fist to collect gold and can stop the ball by squeezing the hand in a grip to avoid obstacles. Difficulty is increased by adding limitations for the game movements like adding more corners and moving obstacles. A secure login system connected to a local database grants exclusive access to patients, ensuring the therapy records.

Conclusions: This game platform, an innovative methodology leveraging motion tracking and personalised game experiences to enhance therapy for stroke patients shows great potential to use for rehabilitation.

PP:33**PREDICTING LIKELIHOOD OF IDIOPATHIC INTRACRANIAL HYPERTENSION FROM IMAGING: A RETROSPECTIVE STUDY****Herath HMMTB 1, Saleh M 2, Rodrigo C 3, Lutchman NG 1, Naidu L 2, Wimalaratna S 1, Wijayawardhana S 4***1 Department of Neurology, Kettering General Hospital, United Kingdom**2 Department of Radiology, Kettering General Hospital, United Kingdom**3 Department of Pathology, School of Medical Sciences, University of New South Wales Sydney, New South Wales, Australia**4 Faculty of Medicine, University of Kelaniya, Kelaniya, Sri Lanka*

Objectives: Idiopathic intracranial hypertension (IIH) is diagnosed by Dandy Walker criteria. Magnetic resonance imaging (MRI) is used to exclude secondary causes. The clinician's dilemma is 'to LP (Lumbar puncture) or not to LP' in an individual patient when the MRI is reported as consistent with IIH in patients who have undergone neuroimaging for a headache syndrome without clinical features of IIH. This retrospective study was carried out with the aim of identifying which MRI features are statistically significant in patients with suspected IIH.

Methods: MRI images of all patients diagnosed with IIH according to modified Dandy criteria and an age and gender-matched group of patients who had a diagnosis of migraine were re-reviewed by a neuroradiologist who was blinded to the diagnosis and clinical history.

Results: When each of the MRI features were considered separately (univariate analysis), seven features were statistically significantly associated with IIH ($p < 0.05$). However, after adjusting for multiple comparisons and excluding collinearity, only 2 features (optic nerve sheath distension, and right Meckel's cave anteroposterior diameter) were associated with a diagnosis of IIH (Bonferroni adjusted p value < 0.005). However, none

of these features were independently associated with IIH when combined in a logistic regression. Thus, should not be used singly.

Conclusions: We agree that patients who are reported by radiologists as likely IIH need further evaluation. While no individual feature could predict IIH, a combination of features had a good sensitivity, specificity, positive and negative likelihood ratios. Imaging features identified in this study as being associated with IIH may be potentially useful to train an artificial intelligence-based algorithm to predict the likelihood of IIH from MRI, which in turn may be independent of the experience of the interpreter.

PP:34

UTILIZATION OF TB GENEXPERT AND TB CULTURE IN CEREBROSPINAL FLUID IN THE NATIONAL HOSPITAL OF SRI LANKA: A CLINICAL AUDIT

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Objectives: To audit the process of cerebrospinal fluid (CSF) testing for tuberculosis (TB): CSF sampling, GeneXpert, TB culture.

Methods: A prospective clinical audit was carried out in 102 patients. The process of testing was analyzed. Focus was on strength of ground of suspecting TB- availability of baseline chest X-ray, at least a noncontrast computed tomography (NCCT) brain, CSF full reports, and the adequacy of sampled CSF volume.

Results: 96 patients were treated as a possible central nervous system (CNS) infection including viral, bacterial, fungal, or TB. 13 patients were on anti-tuberculosis treatment (ATT). Out of them, seven were for central nervous system tuberculosis (CNS TB), two for TB spine (TBS), and four for pulmonary TB (PTB). TB was eventually microbiologically confirmed in one PTB and one TBS patient.

All the patients except one, had undergone an NCCT before the lumbar puncture. 80% had been screened for PTB with a chest X-ray. Despite the availability of the CSF full report within 24 hours, TB GeneXpert and culture were sent before seeing the CSF full report.

All the samples requested TB GeneXpert to be performed. 78 samples proceeded to be used

for TB culture. 24% of samples were <0.5ml. 74% had ≥0.5-2ml (TB GeneXpert was positive in two). TB GeneXpert was negative in all four TB culture-positive patients.

Conclusions: The inadequacy of CSF sample volume is highlighted.

Inefficient utilization of TB GeneXpert and culture is apparent - i.e. not considering TB culture testing in patients who are likely to benefit and on the other hand, requesting the test for patients without strong clinical grounds.

Reference material on CSF sampling, for treating physicians and ground-level staff would be beneficial to fill this gap in communication. It will help to cost-effectively utilize the technical and human resources.

PP:35**A MULTICENTRE STUDY ON THE IMPACT OF EPILEPSY ON SCHOOLING IN CHILDREN**

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Introduction: In Sri Lanka, education is compulsory for children of the age group 5–16 years. Any child who leaves school before completing 16 years is considered a school-dropout. Epilepsy is the most common chronic neurological condition in children and neurocognitive and psychosocial problems associated with epilepsy may have an impact on schooling. Therefore, this study was aimed to determine the characteristics of school dropouts and to explore the factors leading to dropping out of school or never attending school in children with epilepsy (CWE).

Methods: A descriptive cross-sectional cohort study method was used. The study was conducted in three Teaching Hospitals of Sri Lanka. Children with epilepsy between 5–16 years were included.

Results: The sample comprised 207 CWE. The mean age was 11.4 ± 3.1 years, males 121 (58.5%). 129 (62.3%) had focal seizures, 78 (37.7%) had generalised seizures and 52 (25.1%) had epileptic encephalopathy. 03 (1.4%) children have never attended school and 34 (16.7%) dropped out. The mean age of dropping out is 11.1 ± 2.5 years. 167 (81.9%) were enrolled in mainstream school, 20 (9.8%) in special school and 17 (8.3%) in special education units of a mainstream school. Of those who were attending school, 98(57.6%)

had $> 75\%$ ($> 15/20$ days) school attendance, 38 (22.4%) had 50 – 75% and 34 (20%) had $< 50\%$ days of attendance. Disabilities of the child was a reason for dropping out in 33 (97.1%), mismatch/non-acceptance by the school in 18 (53.3%), and financial issues in 08 (23.5%). Dropping out rates were significantly higher with higher Global Assessment of the Severity of Epilepsy (GASE) scale scores ($p<0.05$), and had no significant relationship with the monthly family income.

Conclusions: Only a minority has never attended school, and the majority of children were attending a main stream school. Dropping out was positively associated with severity of epilepsy.

PP:36**PRELIMINARY ANALYSIS OF STUDY OF LONG-TERM OUTCOMES OF A COHORT OF PATIENTS THROMBOLYSED FOLLOWING ACUTE ISCHAEMIC STROKE AT A SINGLE ERTIARY CARE CENTRE IN SRI LANKA****Rajaratnam A 1, Samarasiri U 1, Senanayake B 1***1 Institute of Neurology, National Hospital of Sri Lanka, Colombo, Sri Lanka*

Introduction: Data on post-thrombolysis outcomes of Sri Lankan stroke patients is limited.

Objectives: The study aimed to describe the National Institutes of Health Stroke Scale (NIHSS), modified Rankin Scale (mRS) and Barthel Index (BI) scores at 30 and at 90 days post-thrombolysis, and to explore the potential factors affecting them.

Methods: This prospective study from July to October 2023, enrolled consecutive patients thrombolysed according to standard protocols, at our neurology unit.

Results: Data of 37 patients (males – 24, 64.8%; age - 61.6 years \pm 22) was analyzed. Time from symptom onset to reperfusion was 209 min \pm 101.6. Only 13 (35.1%) were thrombolysed within 3 hours, and rest between 3 and 4.5 hours. The majority (94.5%) received rTPA. Major complications included fatal ICH (2), non-fatal symptomatic ICH (5) and severe COPD exacerbation (1). 13 (35.1%) at admission, 27 (72.9 %) at 30 days and 31 (83.7 %) at 90 days had a NIHSS of 5 or below. 11 (29.7%) at 30 days, and 13 (35.1%) at 90 days were functionally independent (mRS of 0-2). 27 (72.9%) at 30 days, and 29 (78.3%) at 90 days were considered to have moderately/mildly severe disability (BI over 60). Two tailed Wilcoxon signed rank test

proved statistical significant ($p<0.05$) improvements in median values of NIHSS at admission, at 30 days and at 90 days ($Z = -3.93, -2.84$), mRS at admission, at 30 days and at 90 days ($Z = -4.05, -2.66$), BI at admission, at 30 days and at 90 days ($Z = -3.91, -2.51$). Those who had a Large Vessel Occlusion (LVO) had higher odds ($OR=25.6, p<0.01$) of being functionally dependent ($mRS >2$) at 90 days, whereas being male, having diabetes, hypertension, heart disease, atrial fibrillation, prior stroke or TIA, poorer social support, or presence of major complications did not have significant odds of having $mRS >2$ at 90 days.

Conclusions: The studied cohort demonstrated improvements in NIHSS, MRS and BI at 30 days compared to admission values, and at 90 days compared to 30-day values. Of the studied variables, the presence of LVO had higher odds of being functionally dependent at 90 days.

PP:37

IMPULSE CONTROL DISORDERS AND OTHER NON-MOTOR SYMPTOMS IN SRI LANKAN PATIENS WITH PARKINSON'S DISEASE

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Objectives: The impact of non-motor symptoms is often overlooked in favour of the motor symptoms when managing Parkinson's disease resulting in suboptimal patient outcomes. This study aimed to characterise the impulse control disorders and other compulsive behaviours (ICDs-CB) of Parkinson's patients in Sri Lanka.

Methods: All patients with idiopathic Parkinson's disease followed up at the National Hospital of Colombo, Sri Lanka were included. An interviewer-administered questionnaire was used. Symptoms of anxiety and depression were assessed with the Hamilton Anxiety and Depression scales. Presence of ICDs-CB was assessed with the questionnaire for Impulsive-Compulsive Disorders in Parkinson's Disease.

Results: Of 192 patients, only one patient (0.5%) reported being aware of having a compulsive behaviour, while all the others had never been screened for or informed about ICDs-CB. According to QUIP results, 32 patients (16.7%, 32/192) screened positive for at least one ICDs-CB. The subcategories were as follows: compulsive gambling (1.6%, 3/192), compulsive sexual behaviour (1%, 2/192), compulsive buying (4.2%, 8/192), compulsive eating (8.3%, 16/192), hobbyism (6.3%, 12/192),

punding (1%, 2/192), walkabout (10.9%, 21/192), compulsive medication use (0.5%, 1/192). Among these 32 patients, 15 (46.9%) had two or more ICDs-CB (2 ICDs-CB: 4 patients; 3 ICDs-CB: 5 patients; 4 ICDs-CB: 5 patients; 5 ICDs-CB: 1 patient). A lower Barthel index, history of past psychiatric disorders and family history of alcohol abuse were independent predictors of ICDs-CB in adjusted analysis by logistic regression.

Conclusions: Managing both motor and non-motor symptoms are important to preserve the quality of life of patients with Parkinson's disease. They should be screened for symptoms of anxiety and depression regularly during follow-up and educated about the possibility of ICDs-CB soon after diagnosis.

PP:38

IMPACT ON THE RANGE OF MOTION (ROM) OF THE SHOULDER JOINT FOLLOWING HEMIPLEGIC SHOULDER PAIN (HSP) AND ITS RELATIONSHIP BETWEEN DOMINANT SIDE AND NON-DOMINANT SIDE AMONG INDIVIDUALS WITH STROKE IN GAMPAHA DISTRICT

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Objectives: This study was conducted to assess the impact on range of motion (ROM) following hemiplegic shoulder pain (HSP) and its relationship between the dominant and non-dominant sides in individuals following a stroke in the Gampaha District.

Methods: A descriptive cross-sectional study was carried out within stroke survivors with HSP (n=263) in District General Hospital, Gampaha, Colombo North Teaching Hospital and Rehabilitation Hospital, Ragama, Sri Lanka. The socio-demographic data were collected by an interviewer administered questionnaire. Pain intensity, disability level and range of motion of the affected upper limb were measured using the Numerical Pain Scale, Disability of Arm, Shoulder and Hand (DASH) questionnaire and goniometer respectively.

Results: Prevalence of HSP were occurred within 7 days after the onset of the stroke was 29.3% (n=77). Comparing the mean of limited active ROMs (AROMs), the most affected AROM of the shoulder joint were shoulder flexion (89.35 ± 67.800) and external rotation (42.13 ± 32.170). Comparing the mean of limited passive ROMs (PROMs), the most affected PROMs of the shoulder joint were flexion (155.00 ± 29.450), abduction

(147.39 ± 31.620) and external rotation (71.41 ± 19.920). The AROM and PROM of the affected shoulder had a significant negative correlation with the aggravated pain severity. AROM and PROM in the affected shoulder were significantly associated with chronicity of stroke and also with DASH score categorization ($p<0.05$). Only the PROM in flexion, abduction and external rotation were significantly associated with the dominancy of the affected side ($p<0.05$).

Conclusions: According to its relationship with the dominant side and non-dominant side among individuals with stroke in the Gampaha District; the PROM in flexion, abduction and external rotation were significantly affected among participants with the non-dominant side affected compared to those whose with the dominant side affected, but the AROM was not significantly different between these two groups.

PP:39**CLINICAL PROFILE AND OUTCOMES OF CEREBRAL VENOUS SINUS THROMBOSIS IN A TERTIARY CARE SETTING IN SRI LANKA**

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Objectives: Cerebral venous sinus thrombosis (CVST) is a rare form of stroke affecting the young, with a broad aetiology. This study aims to assess the demographic and clinical profile, as well as the management and outcome and to determine the associated risk factors for thrombosis in CVST patients in Sri Lanka.

Methods: This is a prospective observational follow up study for patients with a radiologically confirmed diagnosis of Cerebral Venous Sinus Thrombosis. Demographic, clinical and management related details were collected and patients were followed up during a 6 to 12 month duration.

Results: Of the 28 patients, the majority (82.1%, n=23) were females with the median age of the population being 35 years. The commonest symptoms were headache at 89.3% (n=25), seizures and focal neurological deficits (42.9%, n=12 each). The commonest risk factors were oral contraceptive use in 25% (n= 7), local infections in 14.7% and anti-phospholipid syndrome (APLS) in 10.7%. Radiological features consisted of sinus hyperdensity in 28.6% (n=8), cerebral edema and empty delta sign at 14.3% each (n=4).

42.9% (n=12) had a single venous sinus involved. The commonest sinuses to be involved were the superior sagittal and transverse sinuses in 53.6% (n=15) each. Venous infarcts and hemorrhages accounted for 78.6 % (n=14). Isolated intracranial hypertension was detected in 32.1% (n=9), visual loss in 10.7% (n=3), and recurrent CVST and arterio-venous fistulas in 7.1% (n=2) each. All patients received anticoagulation. The majority (96.4%, n=27) recovered. During follow up magnetic resonance scanning, partial recanalization of sinuses was evident on 21.4% (n=6).

Conclusions: The Sri Lankan profile of CVST is mostly similar to regional and international studies, although there is a high rate of associated local infections and a hypercoagulable state. Pregnancy as an associated factor was lower in number. An increased frequency of isolated intracranial hypertension was detected. Comparatively, long-term visual complications were high. The majority had a good outcome from CVST.

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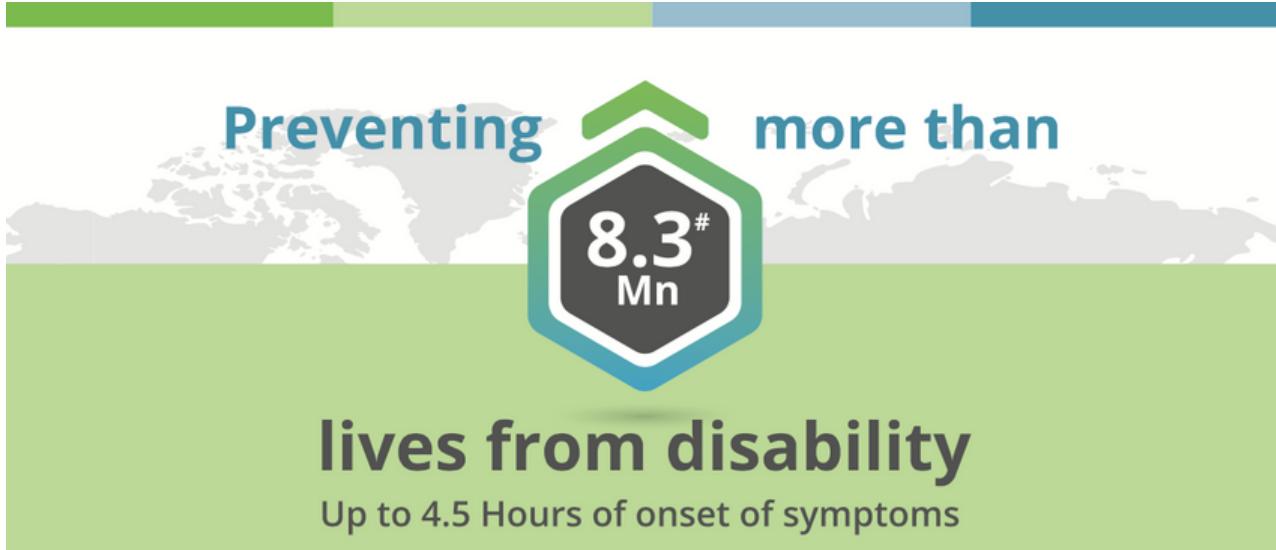


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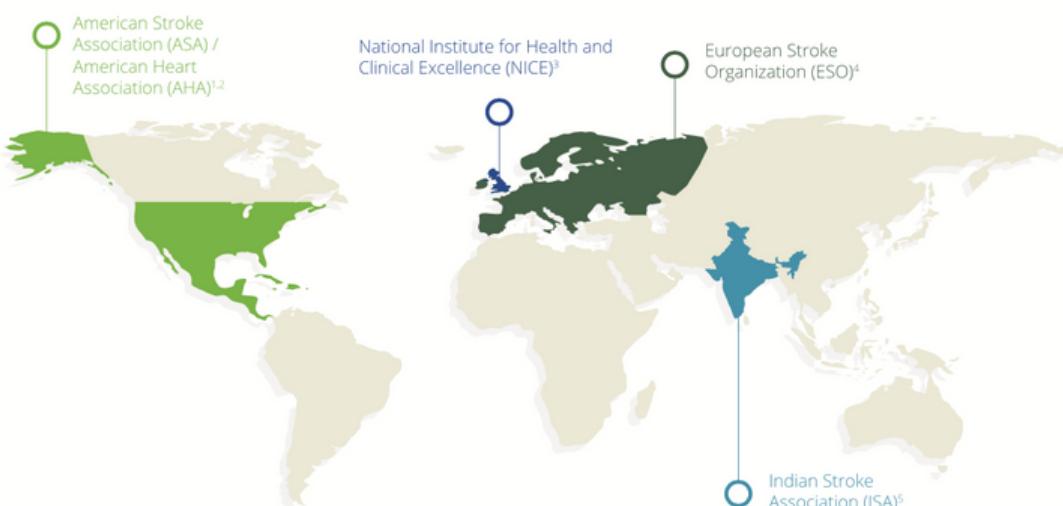
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