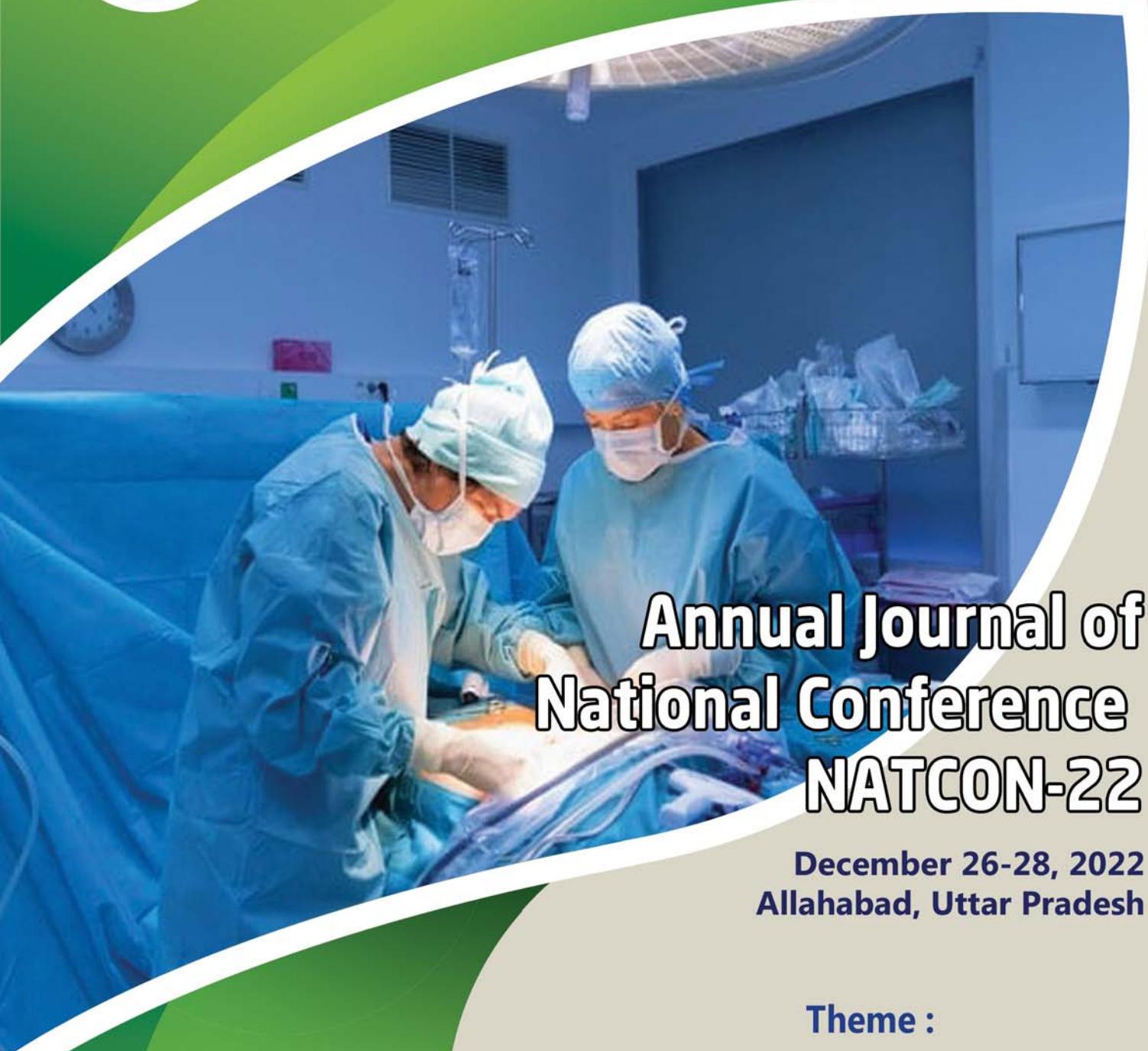




INDIAN MEDICAL ASSOCIATION ACADEMY OF MEDICAL SPECIALITIES (IMA AMS)

Head Quarters, Hyderabad, Telangana



Annual Journal of National Conference **NATCON-22**

December 26-28, 2022
Allahabad, Uttar Pradesh

Theme :
SURGICAL EMERGENCIES



IMA ACADEMY OF MEDICAL SPECIALITIES
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Past National President IMA Hqrs



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National President
IMA Hqrs



Dr. Sharad Kumar Aggarwal
National President Elect,
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Dr. Srirang Abkari
Hon. Editor (Annals),
IMA AMS Hqrs



INDIAN MEDICAL ASSOCIATION ACADEMY OF MEDICAL SPECIALITIES

HEADQUARTERS, HYDERABAD

ANNUAL JOURNAL OF NATIONAL CONFERENCE (NATCON-22)

on
26th, 27th & 28th December 2022
At Allahabad, Uttar Pradesh

“SURGICAL EMERGENCIES”

Dr. G. N. Prabhakara
Chairman, IMA AMS

Dr. Sanjeev Singh Yadav
Hony. Secretary, IMA AMS

Dr. Srirang Abkari
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National President, IMA

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Hony. Secretary General

Dr. Anil Goyal
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IMA PRAYER



May everybody be happy
May everybody be healthy
May everybody be free from pain
May everybody be free from sorrow
May we be the healing cure
Beyond every greed & lure

FLAG SALUTATION

We, the members of Indian Medical Association
Stand here to salute our National Flag.
Its honour and glory shall be our light and strength
And its course shall be our course.
We pledge our allegiance to it and realizing our responsibilities
As the accredited members of this National organization,
We swear we will dedicate everything in our power
To see it fly high in the comity of Nations.
Jai Hind!



From the Editor's Desk

As the world braces anxiously for another potential Covid-19 outbreak, we move ahead with our endeavor in empowering our members in managing issues of utmost importance. This Annals on Surgical Emergencies follows the previous issue of Annals on Medical Emergencies.

"To cut or not to cut" is the ultimate challenge for any surgeon confronted with a patient who suffers from unclear symptoms. The decision of initiating appropriate and timely care with a surgical blade, weighed against the risk of delayed or negligent care by choosing observation or non-operative treatment reflects the eternal "moment of truth" for any surgeon on call.

Diagnosing a surgical emergency is a huge challenge. It requires in-depth knowledge, years of experience, clinical skills at picking up findings and expertise to tackle unexpected events. The annals has articles contributed by eminent and experienced Surgeons on managing common emergencies.

It has been a great pleasure and delight to serve the IMA AMS as the Honorary Editor and I am indebted to our Patron and visionary leader Dr. Ketan Desai for his guidance and encouragement. My heartfelt thanks to Dr. Sahajanand Prasad Singh, National President, IMA for his affection and motivation to do well, Dr. Jayesh Lele, Honorary Secretary General, IMA for setting an example of hardwork and dedication and inspiring to excel in academics, Dr. E. Ravindra Reddy, National Vice-President for his infectious passion for IMA in general and medical ethics in particular, Dr. GN Prabhakara, Chairman IMA AMS for his dynamic leadership and whole-hearted support, Dr. Sanjeev Singh Yadav, Honorary Secretary IMA AMS for his tireless efforts for IMA AMS, for the trust and faith in me and in soliciting articles for this monograph in a very short period.

I am grateful for the immense support received from all the office bearers of IMA AMS, encouragement from the Past Chairmen and Past Secretaries of IMA AMS, the cooperation, enthusiasm and participation from all the State branches of IMA AMS.

I would like to place on record my sincere appreciation of Ms. Sarita, our office staff, for the hard and meticulous work rendered in coordinating with so many people to ensure timely publication of the annals. Mr. Kantilal Shah and Mr. Murali from Atlas Printers deserve accolades for their keen interest in all our publications and the wonderful designing of the annals.

My family deserves a special mention for the love, unflinching support and encouragement all through my life.

I hope this annals will help all our members in updating their knowledge and managing surgical emergencies in their clinical practice.

Long Live IMA!

Dr. Srirang Abkari

Honorary Editor
Annals, IMA AMS



NATCON - 2022

(Organised by: IMA UP State & IMA Prayagraj (Allahabad) Branch)



97th National Annual Conference of Indian Medical Association

83rd Annual Meeting of Central Council of IMA

228th Meeting of Central Working Committee of IMA

Dates :

26th, 27th & 28th
December 2022

Venue :

AMA Convention Centre,
Stanley Road,
Prayagraj (Allahabad)



Chandrashekhar
Azad Park



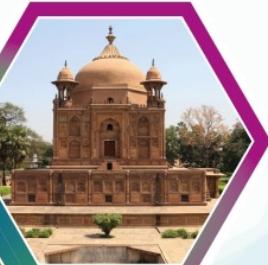
Yamuna Bridge



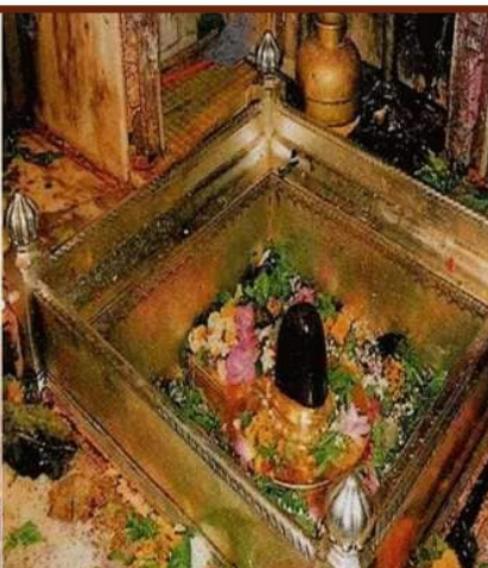
Hanuman
Mandir



Akshayavat



Khusro Bagh



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New Delhi

Message from National President, IMA

I am delighted to know that IMA Academy of Medical Specialities (AMS) is Publishing the prestigious Annals for “**SURGICAL EMERGENCIES**” for the year 2022 on 26th, 27th & 28th December 2022 at the National Conference (NATCON-22), Allahabad, Uttar Pradesh.

IMA Academy of Medical Specialities (AMS) is the one of the major wings of the Indian Medical Association (HQs). The Annual National Conference will enable our members to interact with each other and share their rich experiences of the Medical Practices.

I hope the scientific programmes of this conference will be great interest for our members.

I wish the organisers a great success.

Jai Hind Jai IMA

Sahajanand Prasad Singh

Dr. Sahajanand Prasad Singh
National President
Indian Medical Association (HQs.)

Priority in Rural Healthcare & Dignity of Profession

ग्रामीण स्वास्थ्य सेवा में प्राथमिकता एवं पेशे की गरिमा



Dr. Jayesh M. Lele

Hony. Secretary General
Indian Medical Association

Message

Greetings from Indian Medical Association!

A great pleasure to know that IMA Academy of Medical Specialties is releasing the prestigious Annals for "SURGICAL EMERGENCIES" for the year 2022 during the IMA NATCON 2022 the Annual National Conference of IMA which is going to organize by IMA Uttar Pradesh State Branch under the auspices of IMA HQs on 26th, 27th & 28th December at Allahabad, Uttar Pradesh.

IMA AMS is doing a great job by releasing Annals and I hope it will provide useful material to its members. I am sure, this will enhance the knowledge and expertise of its members in the latest advancements in medicine and medical technology.

IMA AMS is the only platform where all specialists meet with each other and can deliberate on issues which are relevant in providing holistic treatment to patients by discussing interdisciplinary management for better outcome.

Such Conferences provide an opportunity for the members of the Indian Medical Association to have a democratic, healthy and meaningful interaction on issues pertaining to the medical profession and the health-related problems of the people of our country.

I convey my best wishes to the Advisory Board of Annals and I hope it would provide useful material to its members.

I wish the Annual Conference a tremendous success.

Long Live IMA!

Dr. Jayesh Lele

Honorary Secretary General, IMA



Dr. E. Ravindra Reddy

National Vice President
Indian Medical Association

Message

It gives me immense pleasure to learn that IMA Academy of Medical Specialities (IMA AMS) is releasing the prestigious Annals for "SURGICAL EMERGENCIES" for the year 2022 during the IMA NATCON 2022 the Annual National Conference of IMA which is going to organize by IMA Uttar Pradesh State Branch under the auspices of IMA HQs on 26th, 27th & 28th December at Allahabad, Uttar Pradesh.

IMA AMS is doing a great job by releasing these Annals. I am sure; this will enhance the knowledge and expertise of our members in the latest advancements in medicine and medical technology.

I hope that this Conference will bring together research workers, academicians and practicing young doctors to a common platform so as to exchange ideas and stimulate discussion on current problems and develop strategies in the field of monitoring the performance of medical system. The deliberations at the conference will enhance the knowledge and expertise of the participants on latest advancements in medicine and medical technology.

CME is most essential for Medical practitioners as it keep them in touch with modern and latest treatment and diagnostic techniques in various medical fields. IMA has been promoting CME and conducting CME programmes for the last several decades and can look back with satisfaction and pride at this achievement. I am sure; participants will find the CME Programme highly enriching and beneficial.

I thank Our Dynamic Secretary Dr. Sanjeev Singh Yadav and Hon'ble Editor Dr Sriranga Abkari for selecting a wonderful topic.

Long Live IMA!

Dr. E. Ravindra Reddy
National Vice President
Indian Medical Association



Dr. Prabhakara G.N.

Chairman
IMA AMS -2021-2022

Message

Greetings from IMAAMS Head Quarters.

I am very happy that we are releasing the Annals for "SURGICAL EMERGENCIES" during IMA NATCON-2022 at Allahabad. I thank Our Dynamic Secretary and Hony Editor Dr Sriranga Abkari for selecting a wonderful topic and is need of the hour.

Any response to an emergency medical situation will depend strongly on the situation, the patient involved, and availability of resources to help them. It will also vary depending on whether the emergency occurs whilst in hospital under medical care, or outside medical care (for instance, in the street or alone at home).

Hope all these challenges will be clarified in this Annals. I welcome all the members to attend National conference IMA at Allahabad on 26th, 27th & 28th December 2022. I thank all CWC members, and the members who have cooperated with me. My special thanks to all the members who have contributed to Annals. Once again I thank Editor for bringing this issue.

Jai Hind - Jai IMA

Dr. G. N. Prabhakara

Chairman IMA AMS -2021-2022



Dr. Sanjeev Singh Yadav

National Secretary IMA AMS
Hony. Secretary, IMA AMS Hqrs

I am really honored as Secretary IMA AMS to communicate with you through these Annals. IMA Academy of Medical Specialities was established in the year 1979 with the following objectives:

- To provide a forum to Specialists and Super-specialties of all branches of Medicine to discuss multi-disciplinary matters of academic interest
- To promote and encourage unity among the members of IMA
- To enhance image of IMA
- To increase Life Membership and of Fellowship of IMA AMS
- To update all the members of IMA of the recent advances in the field of Medicine and allied subjects
- To conduct C.M.Es all over India
- To conduct various Specialty and sub-specialty courses

I am pleased to send this message through the Annals of IMA AMS, bought out for the convenience of the IMA Doctors. The Annals will provide a gist of the prevailing diseases for the benefit of better treatment to the patients and a comprehensive approach by the treating physicians.

Still a long way to go, the need to publish Annals every quarterly, must be the goal of this Academic Body. Being busy Doctors it has been difficult to get the Articles in time. I request all the Specialists to come forward and send the articles as soon as possible for publishing the same and making the AMS Annals a regular affair.

I would like to congratulate our Editor (Annals) Dr. Srirang Abkari for his tirelessly day and night effort, to bringing out **COVID-19, EPILEPSY, MEDICAL EMERGENCIES** and now **SURGICAL EMERGENCIES** Annals. Dr. Srirang Abkari will be taking charge as Hony. National Secretary of IMA AMS Hqrs along with Dr. Pankaj Mutneja, National Chairman Elect-2022-2023 IMA AMS Hqrs, Dr. Nomeeta Shiv Gupta, National Chairman Elect-2023-2024 IMA AMS Hqrs and Dr. Nibedita Pani, Vice Chairman Elect 2022-2024 IMA AMS Hqrs in December at Allahabad IMA National Conference. All E-Annals posted on the AMS website: <https://ima-ams.org>

AMS Statistics:

- 19 State Chapters
- 195 Branch Chapters
- 16446 Life Members
- 2582 Fellows as on the date

A. **The Academy shall have the following categories of membership:**

- 1. Life Members
- 2. Overseas Members
- 3. Honorary Members



B. The Academy shall have the following categories of Fellows:

1. Founder Fellows
2. Fellows
3. Honorary Fellows:

C. Honorary Professorship: IMA Professorships are now awarded only on approval by IMA Accreditation Council, New Delhi, given during Teacher's Day on 5th Sept of every year.

Increasing Memberships

As on 31 st Dec 2020 :	15395	Increased :	1051
As on 22 nd Nov 2022 :	16446		

Increasing Fellowships

As on 31 st Dec 2020 :	2516	Increased :	68
As on 22 nd Nov 2022 :	2582		

Uttar Pradesh State Chapter has opened "**Jhansi**" New Local Branch Chapter. Branch Chapter Inaugurated by Dr. Surya Kant, National Vice Chairman IMA AMS Hqrs.

The main Activities of IMA AMS during this financial year are as under.

1. **On 2nd April 2022**, as per the directions of IMA Hqrs, National IMA AMS Office Bearers are participated in "**IMA PROTEST DAY**". Justice for Dr. Archana Sharma.
2. **On 9th April 2022**, Dr. Sanjeev Singh Yadav, AMS and National IMA Senior Leaders are participated on "Ethical Issues faced by Medical Profession" organizing by IMA Hqrs on virtual way.
3. **On 16th April 2022**, submitted IMA AMS Activity Report on the occasion of Central Working Committee Meeting on 16th & 17th April 2022 at Chandigarh.
4. **On 22nd May 2022**, IMA National President Dr. Sahajanand Prasad Singh visited IMA AMS Head Office Hyderabad. All IMA & AMS Office Bearers are participated and Honored to National President.
5. **On 28th May 2022**, 36th Governing Council Meeting & IMA AMS National Regional Meeting held on 28th May 2022, at Hubli, Karnataka State, the meeting was presided over by Dr. G. N. Prabhakara National Chairman IMA AMS, Dr. Sahajanand Prasad Singh, National President IMA HQs, Dr. J. A. Jayalal, Imm. Past National President IMA HQs, Dr. Ravi Wankhedkar, Past National President, IMA HQs, Dr. E. Ravindra Reddy, National Vice President IMA HQs, Dr. J. A. Jayalal, Hony. Secretary General IMA HQs, Dr. Sanjeev Singh Yadav, Hony. Secretary IMA AMS Hqrs, Dr. B. N. Reddy, Joint Secretary, IMA AMS Hqrs, Dr. Kateel Suresh Kudwa, President IMA Karnataka, Dr. Ramalingappa Antarthani, Director KIMS Hubli, Dr. S. M. Prasad, Secretary IMA Karnataka, Dr. S. B. Lakkol, President Elect IMA Karnataka, Dr. S. Y. Mulkipatil, President, IMA Hubli, Dr. Ishwar Hosmani, Chairman IMA Annual CME-2022 and Dr. M. Sampath Rao, President IMA Telangana State and Other Governing Council members of IMA AMS from all over India are attended this meeting. The main agenda of conducting of meeting is how to improve our Membership, Courses of IMA AMS, Regional and Zonal Conferences of North, South, East, West and central hosting by IMA AMS, AMSCON-2022, and Fellowships of IMA AMS.
6. **On 1st July 2022**, IMA AMS Hqrs under the IMA Hqrs Conducted for **BLOOD DONATION CAMP** is associated with Abbott India Pvt Ltd at Abbott Office in Hyderabad. Dr. Jayesh Lele, Hony. Secretary IMA Hqrs initiative for the camp is appreciative and he has been doing it for the past few years. Shri Ajay Mishra, Chairman Red Cross Society inaugurated the camp on 1st July 2022 at 10:30 AM. Mrs. Mauha M from Abbott, Dr. Sanjeev Singh Yadav, Hony. Secretary AMS Hqrs, Dr. B.



Narender Reddy, Joint Secretary IMA AMS Hqrs, Dr. Gattu Srinivasulu, Finance Secretary IMA TS and other AMS Office Bearers Staff of AMS, Abbott and Red Cross Society are present. 52 donors participated in this camp.

7. **On 15th August 2022,** Dr. Sanjeev Singh Yadav, Hon. Secretary AMS Hqrs participated Independent Bharath 75 yrs of Mahotsav on 15th August 2022, at IMA Building, Hyderabad along with IMA Hyderabad City Branch and IMA Telangana State.
8. **On 17th Sept to 24th Sept 2022, National IMA AMS South Zone Conference-(AMSSZCON-2022)** (Hybrid Meeting) Inauguration held on 17th September 2022, AMSSZCON 2022- Indian Medical Association academy of medical specialised National AMS South zone conference 2022 was conducted by IMA Cuddalore Branch, Tamil Nadu State from 17/9/2022 to 24/9/2022. National Leaders and State Leaders are participated. The **inaugural function** was conducted from 3pm to 4pm 3pm, National and State IMA Leaders participated in the program.
9. **On 16th September 2022,** ASGE (The American Society for Gastrointestinal Endoscopy) conducted **Gastro Power Summit 2022** associated with IMA AMS Hqrs on 16th September 2022 on virtual way. Dr. Jayesh Lele, Hon. General Secretary IMA Hqrs, Dr. Suryakant, Vice Chairman IMA AMS Hqrs, Dr. Sanjeev Singh Yadav, Hon. Secretary IMA AMS Hqrs participated on behalf AMS Hqrs.
10. **On 9th October 2022, HYBIZ TV HEALTH CARE AWARDS** at HICC Novotel. Dr. Sanjeev Singh Yadav, Hon. Secretary IMA AMS Hqrs being felicitated and received Award from our Minister of Medical & Health , Govt. of Telangana Shri T. Harish Rao. It is well appreciated by our former Indian Cricket Captain Kapil Dev.
11. **On 5th Nov 2022:** IMA AMSCON 2022, the Annual National Conference of IMA Academy of Medical Specialties for the year 2022 was held on 5th Nov 2022, Saturday at the Calcutta Boating & Hotel Resorts, Kolkata, under the auspices of IMA AMS Hqrs and under the Chairmanship of Dr. Santanu Sen, Past National President and Hon. Sec. Bengal State. Academic session was organized from 10.00 am to 5:00 pm on 5th Nov 2022. The Inauguration function was held from 6:00 PM to 8:00 PM, Inauguration address by, Sri. Nirmal Maji, MLA, AITC (Bengal), Dr. Sahajanand Prasad Singh, National President IMA Hqrs, Dr. Sharad Kumar Agarwal, Elected National President-2022-2023, IMA Hqrs, Dr. R.V. Asokan, Elected National President-2023-2024 IMA Hqrs, Dr. E. Ravindra Reddy, National Vice President -2021-22, IMA Hqrs , Dr. Vinay Agarwal, Past National President IMA Hqrs, Dr. Ravi Wankhedkar, Past National President, Dr. G.N. Prabhakara, Chairman Elect IMA AMS Hqrs, Dr. D. Shree Hari Rao, Chairman IMA AMS Hqrs, Dr. Surya Kant, Vice Chairman IMA AMS Hqrs, Dr. Sanjeev Singh Yadav, Hon. Secretary IMA AMS Hqrs, Dr. Srirang Abkari, Hon. Editor (Annals) IMA AMS Hqrs, Dr. M A Kasem, State President, IMA Bengal State & Org. Chairman IMA AMSCON-2022, and Dr. Santanu Sen, MP, Org. Sec. IMA AMSCON-2022 and other Office Bearers and about 1500 delegates are attended this event. IMA AMS Annals released by Sri. Nirmal Maji, MLA AITC (Bengal) and also presented IMA AMS National Awards to Awardees.
12. **On 6th Nov 2022:** IMA AMS Convocation was held on Sunday, 6th Nov 2022 at the Calcutta Boating & Hotel Resorts, Kolkata. Dr. R. V. Asokan, National President Elect-2023-2024, IMA Hqrs delivered Convocation address and graced by Dr. Santanu Sen, Past National President, IMA Hqrs, Dr. E. Ravindra Reddy, National Vice President IMA Hqrs, Dr. G. N. Prabhakara, Chairman IMA AMS Hqrs, Dr. Sanjeev Singh Yadav, National Secretary IMA AMS Hqrs, Dr. Surya Kant, Vice Chairman IMA AMS Hqrs, Dr. D. Sreehari Rao, past National Chairman IMA AMS Hqrs, Dr. Srirang Abkari, Hon. Editor (Annals) IMA AMS Hqrs, Dr. Mohan Gupta, Past Secretary IMA AMS Hqrs awarded Fellowships to the doctors. A total number of 39 applicants were conferred fellowship at the convocation after administration of oath.

IMA Academy of Medical Specialties has proudly awarded Honorary Fellowships to Dr. Sahajanand Prasad Singh, National President IMA Hqrs, Dr. G. N. Prabhakara, National Chairman IMA AMS Hqrs, Dr. Mohan Gupta, Hony. Secretary IMA AMS Hqrs, and Dr. Santanu Sen, Past National President IMA Hqrs, and also awarded Scroll of Honour to Dr. R.V. Asokan, National President Elect-2023-2024, IMA Hqrs, Dr. E. Ravindra Reddy, National Vice President, IMA Hqrs, Dr. Dilip Bhanushali, Senior Member of IMA, and Dr. Srirang Abkari, Hony. Editor IMA AMS Hqrs.

13. **On 6th Nov 2022**, 37th Governing Council Meeting IMA AMS held on 6th Nov 2022, at Hotel Glorious, Kolkata, the meeting was presided over by Dr. Sharad Kumar Agarwal, National President Elect-2022-2023, IMA Hqrs, Chaired by Dr. G. N. Prabhakara, Chairman IMA AMS, Dr. E. Ravindra Reddy, National Vice President IMA HQs, Dr. Surya Kant, Vice Chairman IMA AMS Hqrs, Dr. Sanjeev Singh Yadav, Hony. Secretary IMA AMS Hqrs, and Other Governing Council members of IMA AMS from all over India are attended this meeting. The main agenda of conducting of meeting is how to improve our Membership, Courses of IMA AMS, Regional and Zonal Conferences of North, South, East, and West and hosting by IMA AMS, AMSCON-2022, and Fellowships of IMA AMS.
14. **On 1st Dec 2022:** Dr. Sanjeev Singh Yadav, National Secretary IMA AMS **inspected ESHA IVF Fertility Center** in Hyderabad on 1st Dec 2022 for permission to Conduct Fellowship Course in Infertility. Satisfied with their requirements for conducting Infertility Course. All documents pertaining to Faculty, Staff and Technical scrutinized
15. IMA AMS Kerala, Tamil Nadu, Karnataka, Maharashtra, Assam, Orissa, Bengal, Andhra Pradesh & Telangana States are conducted webinars with Associate of Various Specialties under the IMA AMS Hqrs. Dr. G.N. Prabhakara, Chairman and Dr. Sanjeev Singh Yadav, Hony. Secretary IMA AMS Hqrs and various State Chairman & Secretaries and IMA senior leaders are participated all webinars.

The Activities are as follows:

S.No	Particulars	Place
1	4 -Governing Council Meetings conducted from 1 st Jan 2021 to till date	Hyderabad, Tirupati, Hubli, Kolkata
2	2- National Conference's Conducted (AMSCON-2021 and AMSCON-2022)	Tirupati and Kolkata
3	6- Zonal Conference's Conducted from 1 st Jan 2021 to till date	Haryana-1, Tamil Nadu-2, Assam-1, Maharashtra-1, Kolkata-1
4	225- Zoom Meetings & CMES Conducted with Associate of Various Specialties under the IMA AMS Hqrs from 1 st Jan 2021 to till Date	Andhra Pradesh, Assam, Bengal, Bihar, Karnataka, Kerala, Maharashtra, Orissa, Tamil Nadu & Telangana States.

- **We IMA AMS HQrs providing free Zoom Link to conduct State/Branch webinars via IMA AMS Hqrs.**

Suggestions & Appeal:

1. Request to form a new IMA AMS State Chapter to the following States, few IMA States have not yet formed into the IMA AMS State Chapters, i.e. **ARUNACHAL PRADESH, CHANDIGARH,**



GOA, HIMACHAL PRADESH, MANIPUR, MEGHALAYA, MIZORAM, NAGALAND, SIKKIM, JAMMU & KASHMIR, PONDICHERRY and TRIPURA.

2. A State Chapter shall be established in each State, if there are 100 or more Life Members of the Academy in that State.
3. Request to State Presidents and Hon State Secretaries and to the Chairmen and State Secretaries of IMA AMS to increase membership and fellowships of IMA AMS.
4. State Chapters/Branch Chapters to conduct at least one activity in every month either CME or Webinar on behalf of IMA AMS in your respective State Chapters and their Branch Chapters involving basics Speciality. If you can conduct more than 3 to 4 webinars in a month. **We IMA AMS Hqrs providing free Zoom Link to conduct State/Branch webinars via IMA AMS Hqrs.** AMS State Chapter and National Chapter must be included in that programme. The IMA AMS Hqrs will recognize your services which will result in recognition of your efforts and you will be eligible for AMS National Awards in the National Conference (AMSCON) of IMA AMS.
5. As per the above membership list, if any branch membership is exceeding minimum 10 members, your branch is eligible for Share of LMS fee. So that increases membership to enroll all qualified doctors in IMA AMS.
6. And also we request to enroll Membership & Fellowships at least three as members of IMA AMS to strengthen our AMS which is working towards reaching new goals and Academic heights in the coming years. And also to encourage conducting Speciality Courses in your respective States.

My sincere thanks to our Chief Patron Dr. Ketan Desai, Dr. Sahajanand Prasad Singh, National President IMA Hqrs, Dr. J. A. Jayalal, Imm. Past National President, IMA Hqrs, Dr. Jayesh M Lele, Hon. Secretary General, IMA Hqrs, Dr. Anil Goyal, Finance Secretary IMA Hqrs, Dr. G. N. Prabhakara, National Chairman IMA AMS Hqrs, Dr. Suryakant, Vice Chairman, IMA AMS Hqrs, Dr. D. Sree Hari Rao, Imm. Past Chairman IMA AMS Hqrs for their valuable guidance and suggestions.

My Personal thanks to Dr. E. Ravindra Reddy, Vice-President IMA Hqrs 2021-2022 (from Telagnana) for constant guidance and also to the other Members of IMA National Body.

My sincere thanks to the Continues Guidance by Senior IMA luminaries in conducting the day to day affairs of AMS is always a gratitude and well wishes by our senior leaders, Dr. Vinay Agarwal, Dr. S. Arulrahj , Dr. G. Samaram, Dr. Vedprakash Mishra, Dr. Marthanda Pillai, Dr. K. Vijay Kumar, Dr. Ravi Wankhedkar, Dr. Shantanu Sen, Dr. Rajan Sharma, Dr. R. V. Asokan and all other senior IMA members And also thanks to, Joint Secretaries of IMA AMS Hqrs and Office Staff of IMA AMS Hqrs.

My Hearty wishes to Dr. Sharad Kumar Aggarwal, National President Elect-2022-2023, Dr. R.V. Asokan, National President Elect-2023-2024, Dr. Anilkumar J Nayak, Elect Hony. Secretary General Elect 2022-2024, IMA Hqrs and Dr. Shitij Bali, Finance Secretary Elect 2022-2024, IMA Hqrs.

Long Live IMA & IMA AMS

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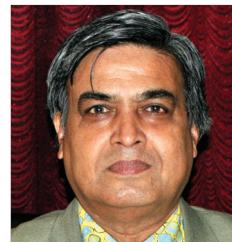
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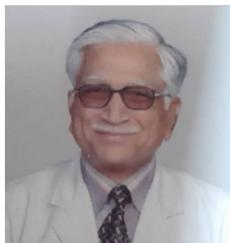
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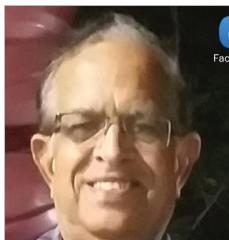
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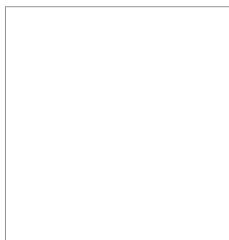
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Proctology/Anal canal Emergencies

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Proctologist Surgeon

Hony. Secretary, IMA AMS Hqrs

Abstract

Anal canal emergencies refer to anal disorders presenting with some alarming symptoms such as Acute Anal pain, Bleeding, foul smelling discharges, which require emergency intervention.

The topic is to understand the need to diagnose anal emergency disorder which may be as follows:

- A) Acutely thrombosed external hemorrhoid
- B) Thrombosed or strangulated internal hemorrhoid.
- C) Bleeding hemorrhoid.
- D) Bleeding anorectal varices.
- E) Anal fissure (Acute).
- F) Irreducible or strangulated rectal prolapse.
- G) Anal abscess.
- H) Perineal necrotizing fasciitis (Fournier gangrene),
- I) Foreign bodies(Inserted or Fecal)
- J) Obstructing anal malignancies.
- K) Sexually transmitted diseases as non-surgical emergencies.
- L) Anal emergencies in neonates.

The associated complications along with the above conditions may be acute urinary retention, bleeding, fecal impaction and anal sepsis.

Accurate diagnosis and treatment a challenge for clinicians and a detailed history taking, careful physical examination, digital rectal examination, video anoscopy is necessary to diagnose and plan and if necessary imaging with Endoanal USG and, MRI anal canal and USG abdomen are also Mandatory .

INTRODUCTION

Anal emergencies refer to anal canal disorders presenting with some alarming symptoms

- A. Anal pain
- B. Bleeding
- C. Swelling.

Most of these conditions may be life-threatening and may be successfully treated in an outpatient setting i.e. day care centers dedicated to Proctology an accurate diagnosis remains a challenging problem for physicians and surgeons.

Acute anal canal problem, patient should be handled with a careful clinical assessment since many of them are suffering from pain, discomfort and embarrassment.

If necessary, rectal examination may be performed under anesthesia. In some cases, some imaging examinations, such as endoanal ultrasonography and MRI scan, are required to confirm diagnosis.

A delay to diagnosis or appropriate treatment of these anal disorders was associated with poor outcomes.

ACUTELY THROMBOSED EXTERNAL HEMORRHOID # EXTERNAL CLOTS ARE ANAL VERGE PAINFUL SWELLING

Classic symptoms of this condition are acute anal pain with a newly enlarged or tender bluish lump at the anal verge.

Some patients may give a history of recent constipation or prolonged straining.

Severe pain, tender, swelling, discomfort on

defecation in the first couple of days. Pain will gradually subside. High pressure within the thrombus may cause the erosion of overlying skin and thus resulting in bleeding.

Acutely thrombosed external hemorrhoid must be differentiated from

1. Complicated internal hemorrhoids.
2. Pigmented melanoma.



Management

Initial conservative management, reassurance and if pain/swelling increases then the clot can be drained locally.

HEMORRHOID

Classified as **1)Thrombosed B)Bleeding c)Variceal**

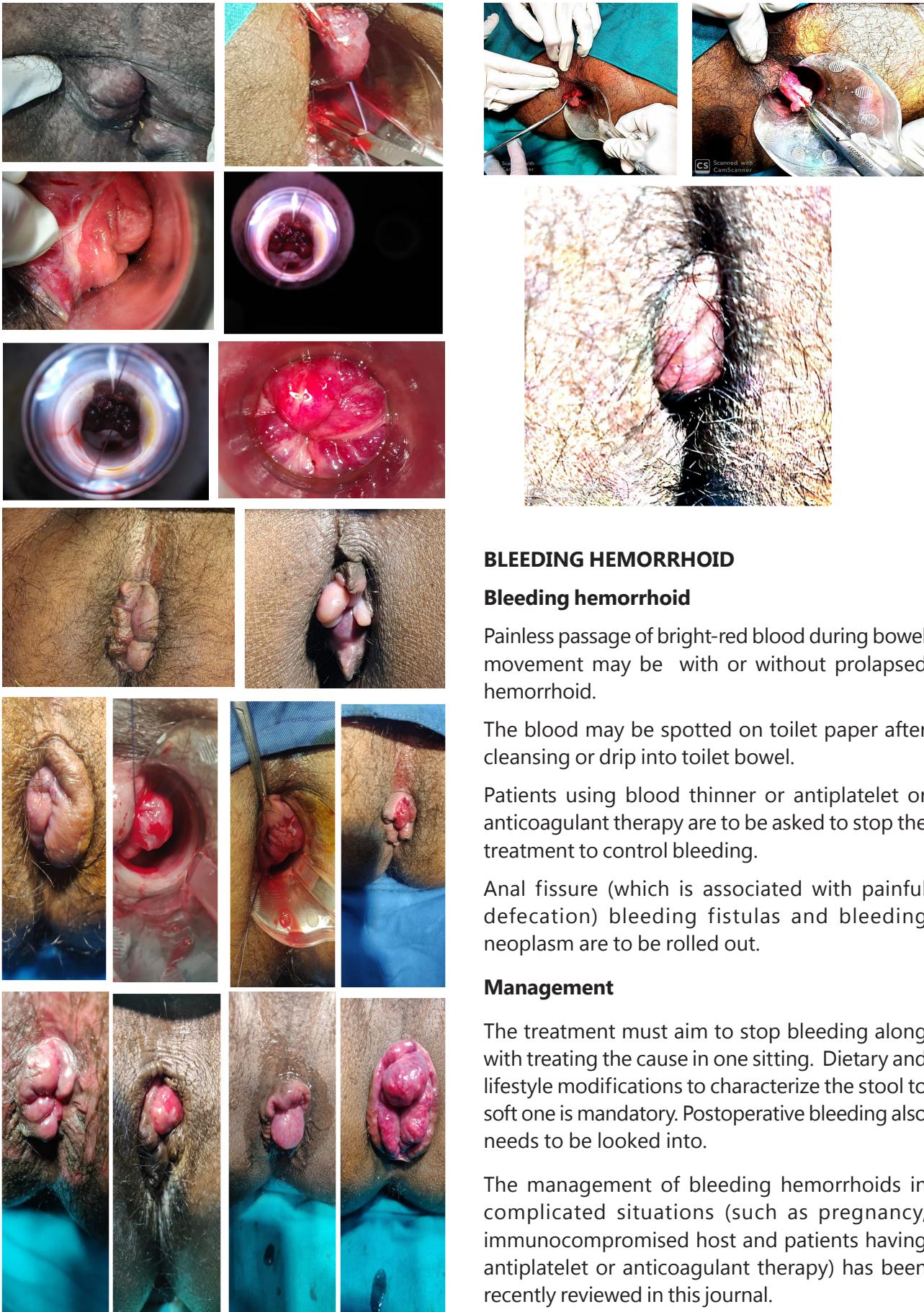
Thrombosed

Internal hemorrhoid become thrombosed, the prolapsed part is left protruded until vascular compromise or venous stasis occurs. Patients present with acute irreducible and painful hemorrhoid. Foul-smelling discharge may be seen in those with mucosal necrosis.

Management

Manual reduction of the hemorrhoid masses may help in reducing pain and tissue congestion. Use of Local Analgesia, Traction n Counter Traction, Magsulph Gauze dressing reduces Edema will help. Followed by Laser Surgery in Addition with Excision of the Thrombosed Mass will benefit.





BLEEDING HEMORRHOID

Bleeding hemorrhoid

Painless passage of bright-red blood during bowel movement may be with or without prolapsed hemorrhoid.

The blood may be spotted on toilet paper after cleansing or drip into toilet bowl.

Patients using blood thinner or antiplatelet or anticoagulant therapy are to be asked to stop the treatment to control bleeding.

Anal fissure (which is associated with painful defecation) bleeding fistulas and bleeding neoplasm are to be ruled out.

Management

The treatment must aim to stop bleeding along with treating the cause in one sitting. Dietary and lifestyle modifications to characterize the stool to soft one is mandatory. Postoperative bleeding also needs to be looked into.

The management of bleeding hemorrhoids in complicated situations (such as pregnancy, immunocompromised host and patients having antiplatelet or anticoagulant therapy) has been recently reviewed in this journal.

SURGICAL MANAGEMENT INCLUDES

Routine hemorrhoidectomy (painful and not result oriented)

DG HAL RAR i.e. Doppler Guided Hemorrhoid Artery Ligation along with Recto-Anal repair.

MIPH

Laser Hemorrhoido-Plasty

BLEEDING ANORECTAL VARICES

Anorectal bleeding in patients with a history of long-standing or uncontrolled portal hypertension would give a clinician clues about this condition. However, hemorrhoid is more prevalent in such patients. It is important to differentiate bleeding hemorrhoids from bleeding anorectal varices because the choices of treatment are different. Diagnosis and differentiation between the two conditions is best achieved with anoscopy or flexible sigmoidoscopy.

Management

Intravenous fluid replacement,

Fresh blood transfusion,

Correction of coagulopathy and optimal medication for portal hypertension.

In active variceal bleeding, per anal suture ligation along the course of varices, endoscopic ligation of the varices and injection sclerotherapy are helpful.

FISSURE IN ANO

Diagnosis

Pain, bleeding, skin tag are the typical features.

A Cycle like syndrome i.e., pain, relief, relapse, and difficulty to defecate are the typical features.

Usually starts with hard stool due to spicy food, un-digested red meat mostly due to gulping food rather than taking time to masticate. Obesity, lack of exercise i.e., sedentary activity, pregnancy and improper dietary combination will be the cause.

A vicious cycle of pain, anal spasm and passage of hard stool would exacerbate further traumatic and ischemic injury to anoderm and prevent the fissure from healing. Usually 6 o' clock and 12 o' clock positions are the places where the fissure occurs due to less blood supply to the sphincter.

Management

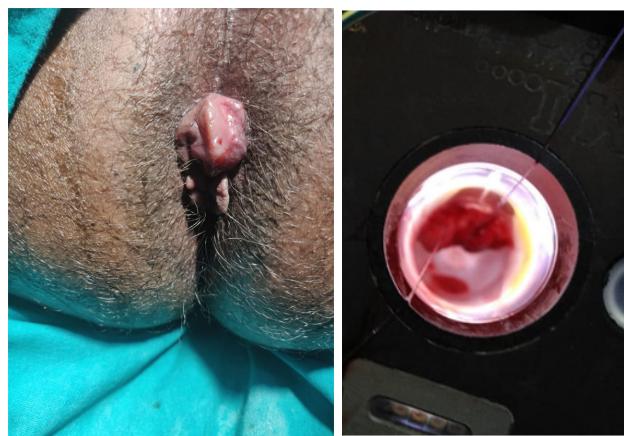
It is necessary to treat this condition as intersphincteric fistula may later be the complication of the simple treatable condition.

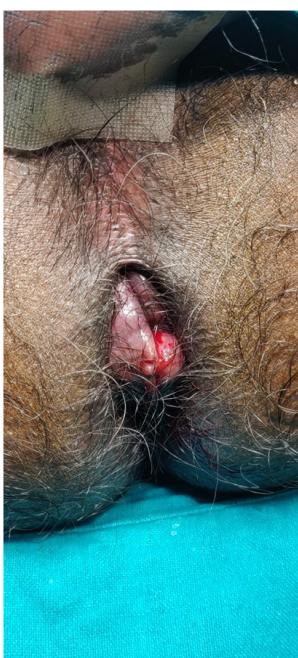
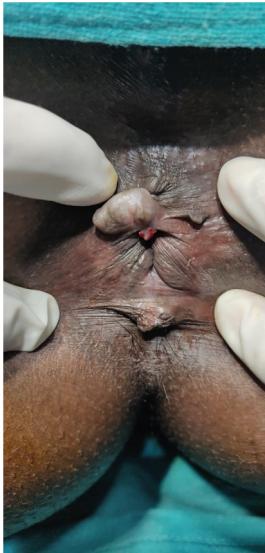
Medical treatment ointments, oral medication and reassurance is the first line of management.

Chemical sphincterotomy with injection Botulism toxin for patients refusing surgery.

Surgical management

Lateral internal sphincterotomy with or without fissurectomy and anal flap.

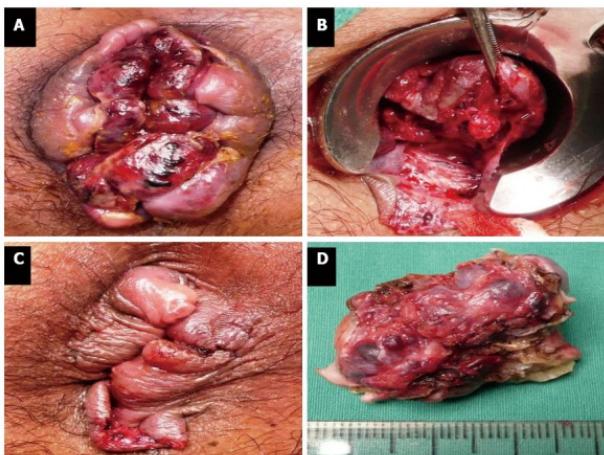




IRREDUCIBLE OR STRANGULATED RECTAL PROLAPSE

Diagnosis

First, clinicians should differentiate prolapsed rectum from circumferentially prolapsed internal hemorrhoid. Classic signs of rectal prolapse are protruding full-thickness rectal wall with concentric rings of mucosa (Figure (Figure3A),3A), while hemorrhoid contains only mucosa and there are radial sulci between hemorrhoid bundles. Irreducible rectal prolapse may occur but acute strangulation of rectal prolapse is quite rare (Figure (Figure3B).3B). Nevertheless, both conditions require prompt intervention.



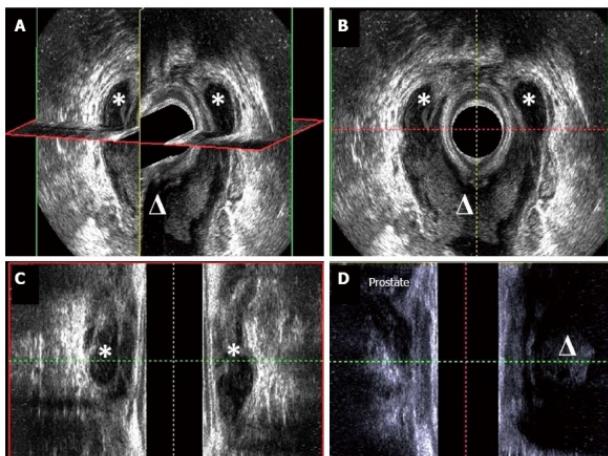
ANAL/PERIANAL ABSCESS

Diagnosis

An abscess forming in the anal area usually originates from an infected anal gland which is located in the anal mucosa and its opening is at the level of dentate line. Once the anal gland is infected, an abscess may form within an intersphincteric area or it could spread to an adjacent area such as perianal region, deep postanal space, ischiorectal fossa or, rarely, a suprarectal space. Acute anorectal abscess may be an initial manifest of anal fistula.

Tender fluctuant, recent onset, in and around the anal area with or without fever is the presenting symptoms increasing pain and difficult defecation with discharging pus is typical. Proper clinical examination and before draining the abscess an

endoanal USG or MRI to rule out fistula is mandatory. The drainage must be done properly and regular dressing by the treating surgeon will prevent formation



Three-dimensional endoanal ultrasonography of horseshoe abscess (A), cross-sectional view (B), coronal view (C) and sagittal view (D). The asterisk means abscess in ischiorectal space and the triangle means abscess in deep postanal space.

PERINEAL NECROTIZING FASCIITIS (FOURNIER GANGRENE)

Diagnosis

A severe life-threatening form of skin and soft tissue infection in the anal and perineal region. Usually a polymicrobial infection that develops secondary to untreated anorectal abscess, genitourinary infection or cutaneous infection. Diabetics, Immunocompromised, Sedentary Un Hygienic are common host.

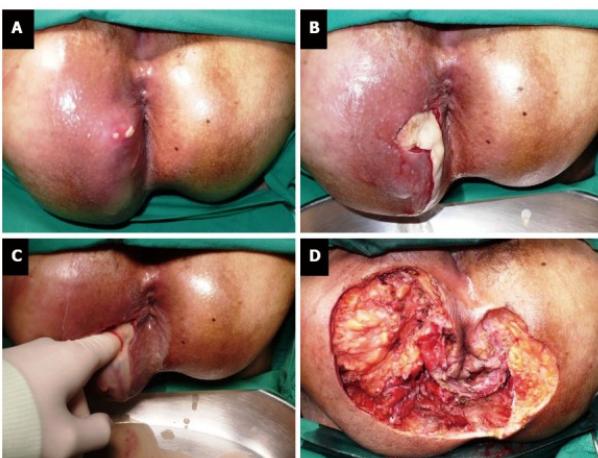
It is characterized by severe perineal pain, high-grade fever, Chills and Rigors, Discharge perianal,

Urinary Retention, Difficulty in Defecation or Stool leakage. Septic Shock develops.

Physical examination Reveals, swelling of buttock and perineum, with or without purple bullae, wetting and Necrosis of skin.

Management

Intravenous fluid, Broad spectrum iv antibiotics. Prompt and adequate surgical debridement of infected tissue is the mainstay of treatment. Colostomy will reduce fecal contamination, Septis Control and wound healing Mitigates. Post Healing Flaps as necessary will help.



ANAL FOREIGN BODIES

Mostly Inserted ones and Rarely Un Digested Hard pieces Like Bones, Twigs, Drugs, Sex Objects ie Toys, Vegetables, Gold Biscuits.

Types are. Either Blunt or Sharp ones.

Surgical advise is sought for Pain, Bleed, Defecation difficulty.

Removal of the Object will be the treatment, Control of Bleeding, Prevention of Abscess and Pain killers always make the pt comfortable.

Psychology counseling will help to prevent recurrence.

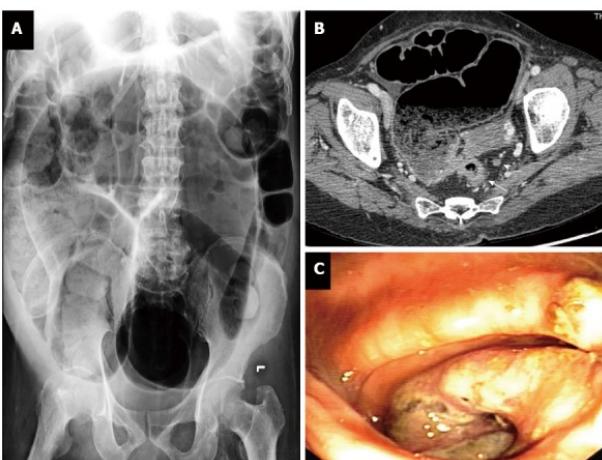
MALIGNANCIES

Fungating or Annular Growths in Sigmoid, Rectum present with acute distal colonic obstruction, Anal Malignancy must always be looked on Physical

examination and proven by Biopsy.

Marked abdominal distension, obstipation and abdominal pain are among cardinal symptoms of this situation. Vomitus with appearance and odor of feces is suggestive of long-standing obstruction. Localized peritonitis and generalized peritonitis may be seen.

Digital rectal examination usually reveals mass causing luminal obstruction. Usg or CT Abdomen will show the Growth. Endoscopic Biopsy to detect the Type.



SEXUALLY TRANSMITTED DISEASE AS ANORECTAL NON-SURGICAL EMERGENCIES

Some sexually transmitted diseases (STDs) may present in an emergency department manifesting as proctitis. Gonorrhea, chlamydia, herpes simplex virus, syphilis and lymphogranuloma venereum (LGV) are common STDs causing proctitis or proctocolitis. It is worth noting that Herpes simplex virus and Treponema pallidum (syphilis) breach both stratified squamous epithelium and columnar epithelium, but Neisseria gonorrhoeae and Chlamydia trachomatis infect only columnar epithelium. *C. trachomatis* serovars D-K is less virulent than *C. trachomatis* serovars L1, L2 and L3, a causative agent for LGV. As a result, LGV may present with severe proctitis, deep rectal ulcer and inguinal lymphadenopathy. Sexually transmitted proctitis in immunocompromised host e.g., HIV-infected individuals could be severe and be coinfected with several pathogens.

ANORECTAL EMERGENCIES IN THE NEONATES

Pediatrician and pediatric surgeon should be familiar with this anorectal disorder which usually presents with delay or failure to pass meconium.

Rule our, meconium plug syndrome, Hirschsprung's disease and anorectal malformations. Vertebral anomalies, Anorectal malformations or Anal atresia,

Clinical examination, including anal inspection for the presence of imperforate anus or perineal fistula,

EARLY POSTOPERATIVE COMPLICATIONS OF ANAL SURGERY

Pain, Bleeding,

Infections,

Urinary Retention,

Constipation,

Post Spinal Headache Vertigo Vomiting

are to be managed as per protocol.

Rarely Intestinal Perforation, Liver Abscess may present.

Dictum is any material removed is to be Sent for Histopathology Examination. Pus for Culture and Sensitivity, Stool to Rlo Covid 19.



Tension Pneumothorax

Dr Surya Kant

Dr Jyoti Bajpai

Abstract

Pneumothorax occurs when air builds up between the parietal and viscera pleurae, causing the lung to collapse. A tension pneumothorax is a serious disorder in which air becomes trapped in the pleural space under positive pressure, shifting all the mediastinal structures and impairing the cardiopulmonary system. A quick diagnosis and treatment of this illness can save a life.

The two types of pneumothorax that occur most frequently are traumatic and tension. Initially presenting as idiopathic spontaneous pneumothorax, tension pneumothorax can occur in 1 to 2% of cases. [1] Since trauma patients have already undergone decompressive needle thoracotomies by the time they are taken to trauma centres, it is challenging to estimate the real incidence of tension pneumothorax. Trauma patients typically experience a pneumothorax or tension pneumothorax 20% of the time. A pneumothorax is 50% more likely to be present in cases of severe chest trauma.[2]

Primary spontaneous pneumothorax is more common in males than females.

The prevalence of asymptomatic Primary spontaneous pneumothorax is unknown. Mild collapse was present in approximately half of individuals, most of them underwent intervention. [3,4]

Pathophysiology

Understanding normal lung physiology is crucial before learning about the pathogenesis of tension and traumatic pneumothorax. Unlike lung and air pressure, intrathoracic pressure is negative. Unlike

the chest wall, which tends to retract outwardly, the lung tends to retract in. As a result, there is a pressure difference between the pleural space and the lung, which prevents the lung from collapsing. Air movement from the lung into the pleural space occurs when connectivity between the pleural space and the lung develops during a pneumothorax. This disrupts the pressure gradient that is typically present, which results in an abrupt increase in intrapleural pressure. This increase in pressure further compresses the lung, lowering its volume and causing hypoxemia.[5]

Due to the disruption of the pleura or tracheobronchial tree, an tension pneumothorax happens when air enters the pleural space but is unable to entirely depart. The lung on the ipsilateral side will collapse as a result of this. [6]The mediastinum shifts toward the opposite side when the pressure rises, which helps to cause hypoxia. Later, this elevated pressure may compress the ipsilateral lung, the heart, and the vasculature, causing hemodynamic instability mostly as a result of compromised cardiac filling and decreased venous return. One of the main causes of pulmonary vasoconstriction, which raises pulmonary vascular resistance, is hypoxemia. If the tension pneumothorax is not treated, hypoxemia, acidosis, and decreased cardiac output can result in cardiac arrest and, eventually, death.

Type of Pneumothorax[7]

- Spontaneous pneumothorax (without antecedent trauma or other obvious cause)

Spontaneous pneumothorax can be divided into-

1. Primary spontaneous pneumothorax.

- 2. Secondary spontaneous pneumothorax.
- Traumatic pneumothorax (occur due to direct or indirect trauma)

A Subcategory of traumatic pneumothorax is *iatrogenic pneumothorax*.

Tension pneumothorax causes include:

- Causes leading to traumatic pneumothorax be it iatrogenic or Non iatrogenic
- **Conversion of spontaneous pneumothorax to tension;**
- **Open pneumothorax**
- **Idiopathic spontaneous pneumothorax**

Etiology: Tuberculosis, chronic obstructive pulmonary diseases, pneumonia, cystic lung diseases are the common underlying lung diseases which can lead to pneumothorax. Accompanying pleural effusion is also seen in this cases. To know the etiology pleural fluid should be examined for tuberculosis by gene expert or culture for tuberculosis.[8,9,10]

Symptoms and Signs : A small pneumothorax may be asymptomatic or can cause very mild symptoms. Sharp chest pain, dyspnoea and cough irritation are the main symptoms. The onset is rapid, and the symptoms are exacerbated by breathing and physical exertion. The pain radiates to the ipsilateral shoulder. The symptoms may be alleviated within 24 h due to adaptation.

Signs :It's common to hear diminished sounds, restricted chest motion, and hollow echoing percussion sounds are often observed. **Asymmetric chest movement is possible. In the case of a minor pneumothorax, the clinical signs may be normal. Cyanosis, hypotension, and tachycardia.** Subcutaneous emphysema may be present (a crepitation on pressing the skin). Signs of injury (haematoma, crepitation from a broken rib, etc.) may be visible on the chest.

Evaluation

Initial assessment is mandatory so as to determine whether the patient is stable or unstable. If the

patient is hemodynamically unstable and in acute respiratory failure, bedside chest x ray or ultrasound should be performed to confirm the diagnosis if it is available for immediate use. Concurrently, patients should be stabilized following the ATLS guidelines that is, complete assessment of airway, breathing, and circulation should be done.

When a patient is hemodynamically stable, radiographic evaluation is can be done with a chest radiograph (CXR) to confirm the diagnosis.(Figure:1)

CXR can demonstrate one or more of the following:

- A thin line representing the edge of the visceral pleura
- Effacement of lung markings distally to this line
- Complete ipsilateral lung collapse
- Mediastinum shift away from the pneumothorax in tension pneumothorax
- Subcutaneous emphysema
- Tracheal deviation to the contralateral side of tension pneumothorax
- Flattening of the hemidiaphragm on the ipsilateral side (tension pneumothorax)

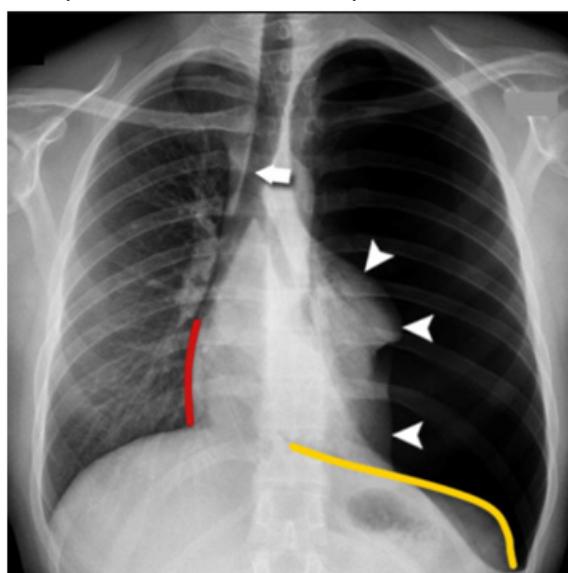


Figure 1:Chest X ray showed left sided pneumothorax and right side mediastinal shift

Sensitivity of Ultrasound thorax is 94% and specificity is 100% accompanied with a skilled operator. So bedside USG may be used generally in unstable patients to assess pneumothorax. Ultrasound findings include the absence of lung sliding and the presence of a lung point. (Figure :2)

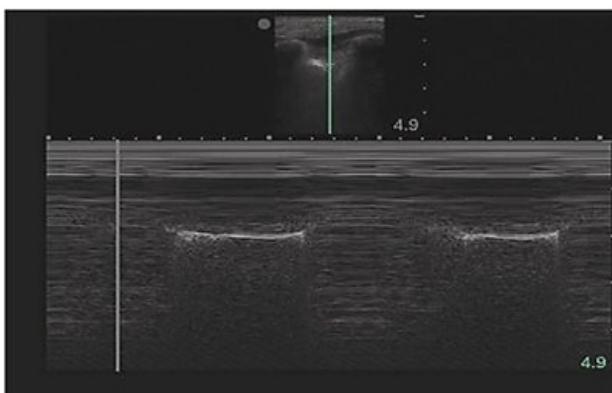


Figure:2 Bar code sign in pneumothorax in M mode

Lung point in M-mode. This M-mode image displays alternating patterns of normal lung and pneumothorax as normal, aerated lung slides over the area of collapsed lung.

Management

Because tension pneumothorax is a medical emergency, it is often treated in the emergency room or intensive care unit. [11] A tension pneumothorax is always cause for prompt medical attention. The tension pneumothorax is changed into an open pneumothorax by inserting a cannula or needle into the pleural cavity.

- Even in cases when a confirming chest radiograph is not possible, thoracocentesis is recommended for patients who are trauma victims or patients who have been revived and are having trouble breathing and showing signs of tension pneumothorax.
- Any injection needle, as large and lengthy as possible, may be utilised for the emergency treatment of tension pneumothorax. If at all feasible, choose a needle that is at least 14G and longer than

5 cm.

- Historically, it has been advised to pierce the midclavicular line at the second intercostal gap. An alternative location is the 4th or 5th intercostal gap in the midaxillary line. To prevent harming the nerves and blood vessels under the upper rib, the puncture is carried out close to the top edge of the lower rib.
- If pleural drainage cannot be started right once, the cannula may be covered during transportation using a specific pneumothorax dressing with a one-way valve to stop outside air from entering the chest cavity (such as the Asherman chest seal).

This results in re-expansion of the collapsed lung. The rapid enlargement of the lung, though, poses a grave threat of pulmonary oedema. If unusual circumstances exist, a chest tube is typically implanted in the fourth intercostal space within the triangle of safety after needle decompression, and an instantaneous CXR is performed to determine whether the pneumothorax has resolved.

The patient's hemodynamic stability often determines the appropriate course of treatment. The "A B C" should be examined in each patient who has tension pneumothorax: airway, breathing, and circulation. As with penetrating chest wounds, traumatic pneumothorax must first be wrapped with an airtight occlusive bandage before being covered in a fresh plastic sheeting. Furthermore, by lowering the alveolar nitrogen partial pressure, 100% additional oxygen supply can aid in reducing the size of the pneumothorax.

If there is a strong clinical suspicion of a pneumothorax in hemodynamically unstable individuals, urgent needle decompression must be carried out. An angio-catheter is used to perform needle decompression in the second intercostal gap in the midclavicular line above the rib.

Patients with bilateral pneumothorax, recurrent ipsilateral pneumothorax, first presentation in those with high-risk occupations like pilots and

drivers, and patients with chronic air leak typically need surgical intervention (for more than seven days). [12,13]

CONCLUSION

The diagnosis and treatment of traumatic and tension pneumothorax necessitate close collaboration amongst various healthcare professionals. Early interventions result from timely and correct assessment, which lowers mortality and morbidity. Care and coordination are essential in these circumstances, and having a diverse team that is prepared to act quickly could save lives. Patients should be advised to avoid flying after a pneumothorax episode until it completely resolves or for at least four weeks after surgery. Pilots and scuba divers should be cautioned against diving or flying until the pneumothorax has completely resolved through pleurodesis or thoracotomy. Additionally, smoking must be discouraged for all patients because it is the main offender.

REFERENCES

1. Sharma A, Jindal P. Principles of diagnosis and management of traumatic pneumothorax. Journal of Emergencies, Trauma and Shock. 2008 Jan;1(1):34.
2. Barton ED, Rhee P, Hutton KC, Rosen P. The pathophysiology of tension pneumothorax in ventilated swine. The Journal of emergency medicine. 1997 Mar 1;15(2):147-53.
3. Gordon R. The deep sulcus sign. Radiology. 1980 Jul;136(1):25-7.
4. Dornhorst AC, Pierce JW. Pulmonary collapse and consolidation: the role of collapse in the production of lung field shadows and the significance of segments in inflammatory lung disease. Journal of the Faculty of Radiologists. 1954 Apr 1;5(4):276-81.
5. Zhang M, Liu ZH, Yang JX, Gan JX, Xu SW, You XD, Jiang GY. Rapid detection of pneumothorax by ultrasonography in patients with multiple trauma. Critical Care. 2006 Aug;10(4):1-7.
6. Soldati G, Iacconi P. The validity of the use of ultrasonography in the diagnosis of spontaneous and traumatic pneumothorax. The Journal of Trauma. 2001 Aug 1;51(2):423-.
7. Shostak E, Brylka D, Krepp J, Pua B, Sanders A. Bedside sonography for detection of postprocedure pneumothorax. Journal of Ultrasound in Medicine. 2013 Jun;32(6):1003-9.
8. **Surya Kant**, "Medical thoracoscopy in pleural infections", Medical thoracoscopy semirigid and rigid "A practical guide"2014; 56-63.
9. A K. Maurya, **S. Kant**, R. A. S. Kushwaha, V L Nag, M. Kumar, TN Dhole Advantage of using IS6110-PCR vs. BACTEC culture for Rapid detection of *M. tuberculosis* from pleural fluid in Northern India. Bioscience Trends 2011; 5(4):159-164
10. AK Mishra, SK Verma, **S Kant**, RAS Kushwaha, R Garg, S Kumar, V Prakash, A Verma, M Sagar A study to compare the diagnostic efficacy of closed pleural biopsy with that of the Thoracoscopic guided pleural biopsy in patients of pleural effusion. South Asian Journal of Cancer 2016; 5(1): 27-28
11. Henry M, Arnold T, Harvey J; Pleural Diseases Group, Standards of Care Committee, British Thoracic Society. BTS guidelines for the management of spontaneous pneumothorax. Thorax 2003;58:ii39-52.
12. MacDuff A, Arnold A, Harvey J; BTS Pleural Disease Guideline Group. Management of spontaneous pneumothorax: British Thoracic Society Pleural Disease Guideline 2010. Thorax 2010;65 Suppl 2():ii18-31.
13. **S Kant***, SK Verma.Rheumatoid Arthritis Presenting As Pleural Effusion. A Case Report, JIMI- 2006; **9 (4)**: 118-120



GI Perforations

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Gastrointestinal perforations include gastric, duodenal, ileal, appendicular, caecal, colonic, and rectal perforation. These are most common cause for acute peritonitis leading to spectrum of systemic inflammatory response syndrome, multiorgan dysfunction syndrome and multiple systemic organ failure and associated mortality (1). They are due to varied reasons from reversible to irreversible causes. (2). Once diagnosed, they need to be dealt swiftly by stabilization of patient and closure of perforation followed by a definitive treatment of the cause of perforation at a later stage (3).

Types

1. Gastric and Duodenal perforation -
2. Small bowel
3. Large bowel

Gastric and duodenal perforation;

Etiopathology

The perforation of peptic ulcer is the second most common complication of acid peptic disease. (5) A known patient of peptic ulcer has a 10% lifetime risk of perforation (6). The main cause of peptic ulcer is increased acid production due to presence of double the number of parietal cells in the stomach in hyper secretors and in some specific blood groups (o+ve) (3,7).

Helicobacter pylori infection was found to be the cause of Type B gastritis due to release of urea splitting enzyme and alkalinisation of mucosa, with production of Ammonium ion NH4+ and rebound release of acid. Long standing infection leads to chronic atrophic gastritis and gastric cancer.

Excessive use of NSAIDS, smoking and alcohol abuse, malnutrition result in gastric mucosal damage and ulceration.(3,4,7)

Table1. causes of gastric and duodenal ulcers and perforations

	Stomach	Duodenum	Perforation
1. Acid peptic disease	H .pylori infection	Hyper secretors H .pylori infection	10%
2. Mucosal damage	NSAIDS, Smoking, Alcohol, Malnutrition, Shock, Curlings ulcer, Cushing's ulcer		
4. Traumatic		a. Iatrogenic trauma b. Blunt injury abdomen	0.013%
5. Malignancy	T4 stage carcinoma	Infiltration from adjacent malignancy eg. Pancreas	

Carcinoma stomach tends to perforate in T4 stage, with infiltration of growth into all the layers of stomach wall. Overall, the duodenal ulcers perforate almost twice as gastric ulcers do. (2)

Traumatic duodenal perforation was a rare complication of upper GI endoscopy. It is the most common perforation after oesophageal perforation, with incidence of 0.13% and with <0.004% mortality rate. the 2nd or 3rd part of duodenum could be perforated during (9,10,11)

Blunt injury abdomen also may lead to perforation of 2nd part of duodenum due to shearing forces. (8)

Small bowel and large bowel perforation

Small bowel perforation (excluding duodenal ulcers) are mainly due to damage to mucosa due to infection, inflammation, ischemia, or malignant infiltration (2,5,6,7,)

Infections like Salmonella typhi, paratyphi, tuberculosis, fulminating enterocolitis lead to mucosal infiltration and perforation. This is by

inflammatory reaction leucocytes and macrophages as in enterocolitis, amoebiasis, proliferation of lymphatic patches as in salmonella infection

Crohn's disease is an immune related inflammation involving all the layers of bowel wall

Ulcerative colitis involves colon and rectum with diffuse inflammation of mucosa and submucosa and bowel distension (toxic megacolon) and perforation.

Appendicitis may lead to appendicular perforation and sometimes caecal perforation.

Rarely a diverticulitis of colon may perforate with a pericolic abscess formation or a general/local peritonitis

Carcinoma of colon is a peculiar clinical condition which may result in a perforation not only at the site of growth but also at the caecum when an obstruction due to annular growth is on the left side of the colon and a competent ileocaecal valve

Table 2. Causes of perforations of small bowel and large bowel and percentage of perforations

Causes	Small bowel	Caecum	Appendix	Colon	Rectum
1.Infection	Salmonella typhi (0.8% to 18%) Tuberculosis (1 to 15%) Fulminating enterocolitis (3 to 6%)	Amoebiasis Tuberculosis	Acute appendicitis (20 to 30%)	Amoebiasis (6%)	
2. Immune related	Crohns(3%)	Ulcerative colitis(6%)		Ulcerative colitis(6%)	
3. Malignancy	Lymphoma(9%)		Carcinoid tumor	Colon cancer (1.2 to 10%)	Rectal cancer (36%)
Radiation	Strictures, Obstruction				Stricture, Fibrosis (0.6%)
Trauma	Penetrating (17%) Blunt(1%)				
Iatrogenic	Ercp(0.03 to 0.3%)			Colonoscopy (0.2 to 5%)	
Ischemia	Vasculitis Superior mesenteric artery and vein thrombosis				

Note — figures in parenthesis indicate perforation rate

act as a closed loop. This effect is seen in only 3% of cases of carcinoma colon (17)

Clinical features

Perforation of gut leads to disruption of the continuity and spillage of bowel contents into the general peritoneal cavity. The higher the perforation, the less the contamination, with least contamination in duodenal ulcer perforation. Hamilton Bailey describes a duodenal ulcer perforation in three clinical stages 1. Stage of chemical peritonitis 2. Stage of illusion 3. Stage of bacterial peritonitis.

The patient experiences sudden severe pain which is masked for few hours followed by development of full-blown peritonitis.

Peritoneum is the organ with the largest surface area after the skin, hence its inflammation causes vasodilatation and seepage of intravascular fluid into the peritoneal cavity, with the resultant hypovolemia, and acute tubular necrosis due to the preferential renal vasoconstriction and shunting of blood to heart and brain. During the compensatory phase of shock there is tachycardia and normal blood pressure. As the 3rd space loss continues with the worsening of peritonitis due to contamination due to ongoing leakage from the site of perforation, a stage of irreversible shock sets in.

The patient looks confused, pale, and dehydrated, with sunken eyes, famously called as Hippocrates facies. Patient will be febrile, pulse - rapid and thready, Blood pressure may not be recordable, cold, and clammy, extremities, abdomen with guarding and rigidity and absent bowel sounds. In a delayed presentation case, with a MODS and MSOF stage signs of breathlessness and irritation, delirium and coma would supervene due to cerebral hypoxia.

Investigations

Investigations and resuscitation should be done simultaneously when the patient presents in a state of shock

Complete blood picture, Random blood sugar,

Serum creatinine, Serum electrolytes, liver function tests, Arterial blood gas analysis should be assessed on a war footing.

Stress induced hyper glycemia, Acute kidney injury parameters like elevated blood urea and serum creatinine may be found if multi organ dysfunctions occurred

Bedside plain x ray abdomen, ultrasound scan abdomen to be done to confirm the cause of peritonitis at the outset.

After initial resuscitation, and recovery of blood pressure CECT Abdomen to confirm the cause of peritonitis and cause of perforation may be attempted.

Pneumoperitoneum is the hallmark of perforation of any hollow viscus (3,4,7,) spillage of contrast (gastrograffin) into peritoneal cavity can be seen when in doubt. Increased inflammatory fluid collections can be seen on ultrasound and CT film. The same can be drawn on paracentesis.

Treatment

Initial resuscitation

When a patient is brought with signs of impending shock or in a state of shock, all the medical and paramedical staff in triage area of casualty should become alert and work in unison. 2 peripheral IV lines should be established and two litres of isotonic fluids such as Ringer lactate, normal saline to be administered speedily, Ryle's tube installed, bladder catheterised, nasal oxygen inhalations started, IV broad spectrum antibiotic (preferably non nephrotoxic) to be given.

IV fluids are to be continued at the rate of 100ml /hour and the vitals and urine output monitored continuously. Once a minimum 400ml of urine output is achieved and pulse becomes palpable and blood pressure recordable, patient may be shifted for radiology department for CECT

Flank drains – if patient is already in a state of irreversible shock, no heroic measures like laparotomy may save him. In such situations, bilateral flank drains under local anaesthesia to drain inflammatory fluid may help in draining the

intraabdominal purulent collections. Laparoscopy / laparotomy can be planned after general condition improves.

Definitive treatment

Laparoscopy or Laparotomy should be planned after adequate correction of fluid and electrolyte balance. Abdomen should be thoroughly explored to identify the site of perforation.

Duodenal ulcer perforation will be on the anterior wall of first part of duodenum. In case of a **gastric perforation**, multiple biopsies from four quadrants of the perforation, to rule out malignancy.

Small perforations(<5mm) can be suture closed with interrupted 3 zero polyglactic acid/ silk suture and reinforced with a Grahm's patch (omentum)(14).

large perforations tend to leak if sutured directly. the perforation must be packed with omentum and then closed over the omentum. A serosal patch with a loop of jejunum on the site of perforation is found to decrease the chance of reperforation to some extent.(15)

Definitive procedures like truncal vagotomy, antrectomy , gastrojejunostomy ,pyloroplasty may be attempted if the patient is stable and peritoneal cavity is not much contaminated.

Ileal perforations tend to be nearer to ileocaecal valve and in areas of Peyer's patches. The entire ileum may be macerated resembling a blotting paper. Small single perforations should be trimmed at edges and closed with 3 zero polyglactic acid sutures. Multiple perforations and unhealthy long segment of ileum needs limited resection of the unhealthy segment and end to end anastomosis. Severe peritonitis and unstable patient warrants a loop ileostomy /end ileostomy (16)

Caecal perforations due to an extending appendicular inflammation usually occur at base of appendix and posterior wall. Initial management should be by a caecostomy followed by a definitive procedure later.

Caecal perforation due to closed loop effect in a colon carcinoma needs resection of entire colon and end ileostomy in lower quadrant.

Colonic perforations due to T4 carcinoma at the site of perforation need special consideration. Radical hemicolectomy and hartman's procedure would save the patient from possibility of a leak in an emergency setting.

Rectal perforations can be due to rectal growth, ulcerative colitis, trauma and penetrating injuries, malignant infiltration from cancer cervix , leading to recto vesical /recto vaginal fistulas simple closure of perforation, or excision of fistulous tract and proximal diversion colostomy.

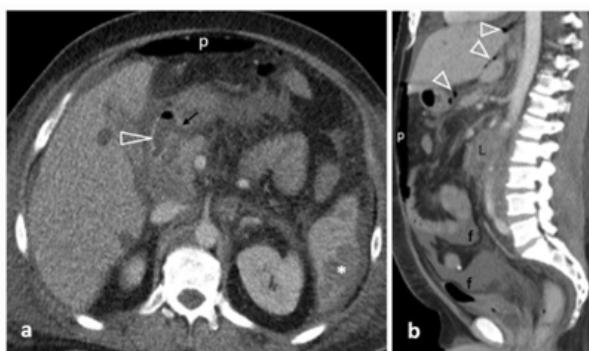
Summary

GI perforations often result due to underlying pathology of the bowel.

They lead to leakage of contents and peritonitis leading to SIRS, MODS, MSOF. Measures to prevent contamination and to reverse the effect of contamination should be executed swiftly to decrease mortality rate which ranges upto 30%.(3,4,6)

Acknowledgements

My sincere thanks to Dr Mahesh assistant professor of surgery GMC Guntur for helping me with figures and tables .



CT

Fig-1 (13) fig-1. 62-year-old patient with perforating gastric carcinoma. A Axial and (b) sagittal contrast-enhanced images show the enhancing antral mucosa (arrowhead) and a focal discontinuity of mucosal enhancement (arrow) associated with fat stranding, pneumoperitoneum (p), free fluid (f), gas bubbles by the posterior wall of the antrum coursing cranially within the lesser sac (arrowheads), co-existing metastases (*), as well as periaortic lymphadenopathy (L)

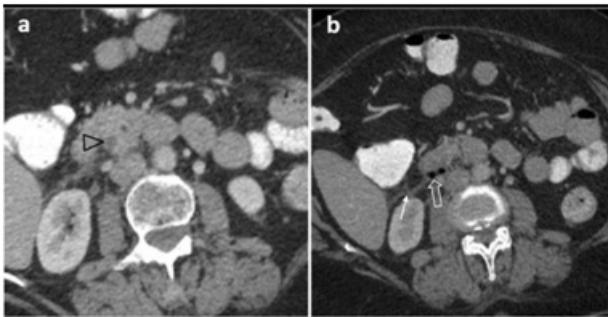


Fig 2. 75-year-old patient with duodenal perforation post-ERCP. **a, b** Axial contrast-enhanced image shows a focal discontinuity of wall enhancement (arrowhead), associated with retroperitoneal gas bubbles (open arrow) posterior to the 3rd duodenal segment and thickening of Gerota's fascia (white arrow)

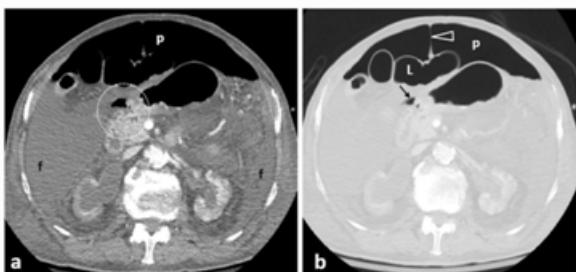


Fig 3. 92-year-old patient on long-term corticosteroids. **a** Soft tissue window and **(b)** lung window contrast-enhanced axial images demonstrate a discontinuity of the hyperenhancing gastric mucosa, postero-medially to an extraluminal gas bubble (area within circle). Free fluid (f) and pneumoperitoneum (p) are outlining air-filled bowel loops (L) and the falciform ligament (arrowhead). The triangular-shaped separate gas bubble (arrow) points towards the perforation site.



Fig 4. 65-year-old patient with perforated

duodenal ulcer. Coronal unenhanced image (modified soft tissue window) following oral contrast administration demonstrates a thickened duodenal wall (*), contrast leaking (black arrow) to the peritoneal spaces and free gas bubbles (arrowheads). Note hyperdensity of perihepatic free fluid (white arrow) compared with the diluted contrast in the rectouterine pouch (open arrow)



Fig 5. 42-year-old patient with appendiceal perforation. Contrast-enhanced parasagittal reformatted image shows a dilated appendix (open arrowhead) containing an obstructive appendicolith (black arrowhead), reactive wall thickening of the ascending colon (*), surrounding fat stranding, as well as an abscess (ab) containing air-fluid levels.

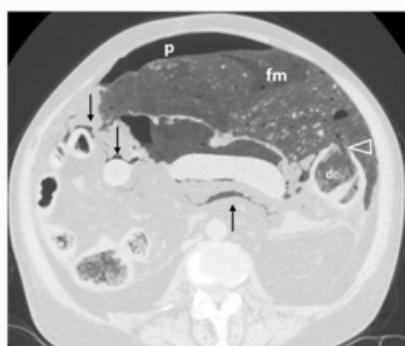


Fig 6(13) 75-year-old patient with perforation secondary to obstructive ileus in the setting of rectal cancer (not shown). Axial image (lung window) shows leaked faecal material (fm), abutting the mesenteric root and surrounding ileal loops (arrows), pneumoperitoneum (p), and the perforation site (arrowhead) as a wall discontinuity of the descending colon (dc).

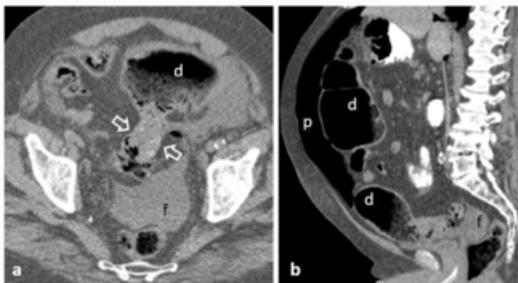


Fig 7. 75-year-old patient with obstructive sigmoid carcinoma. **a** Axial and **(b)** sagittal non-enhanced images show concentric narrowing of the sigmoid lumen (between arrows), prestenotic colonic dilatation (d), massive pneumoperitoneum (p) and free fluid (f).



Fig 8. Xray erect abdomen showing pneumoperitoneum.

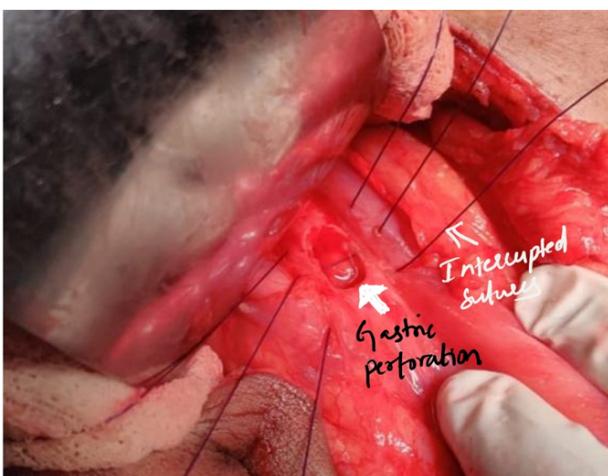


Fig 9. 65 year old patient presenting with perforation peritonitis. Above picture shows 1.5x1 cm perforation in the pyloric antrum(white arrow).

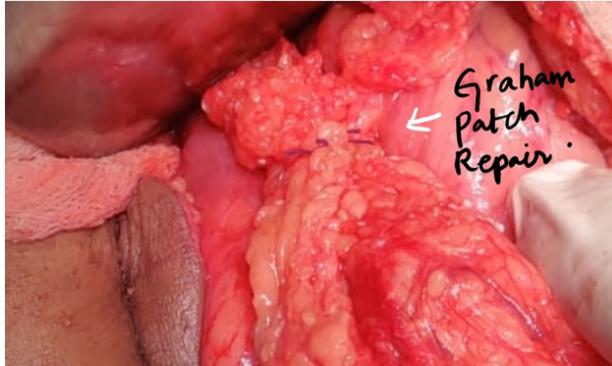


Fig 10. Picture showing graham patch repair.

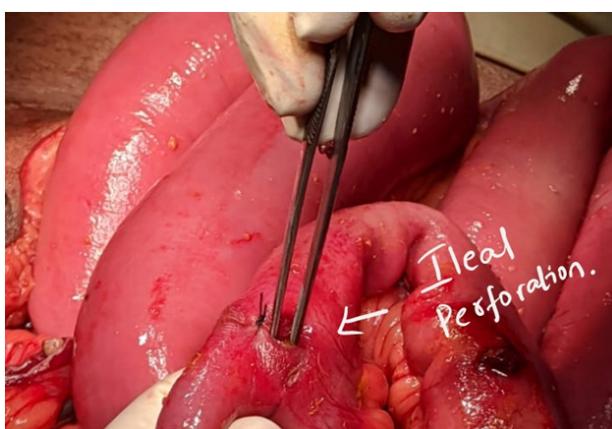


Fig 11. Picture showing ileal perforation

References

1. Causes of Acute Peritonitis and Its Complication Monitoring Editor: Alexander Muacevic and John R Adler Danesh Kumar,¹ Ishan Garg,² Atif Hussain Sarwar,³ Love Kumar,¹ Vikash Kumar,⁴ Sonam Ramrakhia,^{5,6} Sidra Naz,⁷ Amna Jamil,⁸ Zoya Qamar Iqbal,⁴ and Besham Kumar⁹
2. Bowel Perforation Mark W. Jones; Sarang Kashyap; Christopher P. Zabbo. <https://www.ncbi.nlm.nih.gov/books/NBK537224/#:~:text>
3. Bailey and love text book of surgery 25th edition p 1062
4. Acute abdomen Sabiston's text book of surgery 20th edition p1145
5. Schwartz principles of surgery9th edition , page 2200

6. Maingot's Surgery p 471
7. Pathophysiology of duodenal and gastric ulcer and gastric cancer John Calam and J H Baron BMJ v.323(7319); 2001 Oct 27 PMC1121510
8. Duodenal perforation following blunt abdominal trauma Hemanga K Bhattacharjee, Mahesh C Misra, Subodh Kumar, and Virinder K Bansal J Emerg Trauma Shock. 2011 Oct-Dec; 4(4): 514–517
9. Duodenal perforation following esophagogastroduodenoscopy (EGD) with cautery and epinephrine injection for peptic ulcer disease: An interesting case of nonoperative management in the medical intensive care unit (MICU) Jason Chertoff, MD, MPH,^N Vikas Khullar, MD, and Lucas Burke, Int J Surg Case Rep. 2015; 10: 121–125
10. Kavic Stephen M., Basson Mark D. Complications of endoscopy. Am. J. Surg. 2001; 181 (4): 319 – 332. [PubMed] [Google Scholar]
11. Eisen Glenn M. Complications of upper GI endoscopy. Gastrointest. Endoscopy. 2002; 55 (7): 784 – 793. [PubMed] [Google Scholar]
12. Intestinal Perforation Jessica Hafner; Faiz Tuma; Gilles J. Hoilat; Omar Mara https://www.ncbi.nlm.nih.gov/books/NBK538191/#:~
13. Pouli, S., Kozana, A., Papakitsou, I. et al. Gastrointestinal perforation: clinical and MDCT clues for identification of aetiology. Insights Imaging 11, 31 (2020). <https://doi.org/10.1186/s13244-019-0823-6>
14. Hill AG. Management of perforated duodenal ulcer. In: Holzheimer RG, Mannick JA, editors. Surgical Treatment: Evidence-Based and Problem-Oriented. Munich: Zuckschwerdt; 2001. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK6926/>
15. Gupta S, Kaushik R, Sharma R, Attri A. The management of large perforations of duodenal ulcers. BMC Surg. 2005;5:15. Published 2005 Jun 25. doi:10.1186/1471-2482-5-15
16. Wani RA, Parry FQ, Bhat NA, Wani MA, Bhat TH, Farzana F. Nontraumatic terminal ileal perforation. World J Emerg Surg. 2006;1:7. Published 2006 Mar 24. doi:10.1186/1749-7922-1-7
17. Perforation of the cecum resulting from a closed-loop obstruction in a patient with an adenocarcinoma of the sigmoid colon: a case report Jefferson Sing Toledo, Junior, Marley Moreira Correia, Rafael Rodrigues Coutinho, Eduardo Fukamachi Kifer, and Diego de Faria Magalhães Torres^N

Cardiac Tamponade

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INTRODUCTION

The pericardium (Greek word **perikardion**, meaning 'around the heart') is a double-layered sac enclosing the heart and the roots of the great vessels. Between the 2 layers (inner visceral layer and outer serous layer) is the pericardial cavity containing pericardial fluid (15–35 ml of plasma ultrafiltrate). Cardiac tamponade occurs when an excess of pericardial fluid accumulates in its cavity leading to an increase in pericardial pressure and thus leading to progressive compression of the underlying chambers of the heart¹.

CLINICAL PRESENTATION AND DIAGNOSIS

Pericardial fluid accumulation speed decides the clinical presentation. Rapid accumulation of even a small amount of fluid (eg., traumatic and iatrogenic perforations), can lead to a dramatic rise in intrapericardial pressure within minutes leading to cardiac tamponade, whereas slow accumulation of fluid over a period of days to weeks allows a large collection of fluid before a significant increase in pressure develops causing tamponade physiology^{1,3}.

Common symptoms are dyspnoea, orthopnoea, fatigue, palpitations, fever, cough, and dizziness. Hemodynamic clues to clinical diagnosis of cardiac tamponade are evidence of low cardiac output with elevated cardiac filling pressures with high sympathetic tone and exclusion of other causes of shock. The patient will have distended neck veins with elevated jugular venous pressure, tachycardia, peripheral vasoconstriction, low systolic blood pressure, pulsus paradoxus, and diminished heart sounds with low voltage complexes on the electrocardiogram. Equalization

of diastolic pressures across all chambers is observed when a pulmonary artery catheter is placed³.

Two-dimensional echocardiography remains the primary diagnostic tool for pericardial diseases. It can confirm the size, location, and other characteristics of the pericardial collection. End-diastolic echo-free space between the epicardium and parietal pericardium can be measured and effusion can be classified as small (<10 mm), moderate (10–20 mm), large (>20 mm)³.

Use of CT scan and CMR help in the detection of loculated effusion and pericardial thickening and masses, and associated chest abnormalities¹.

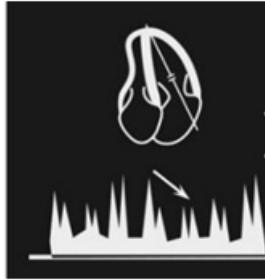
Echocardiographic features of tamponade physiology³



1. Collapse of the right ventricle in early diastole



2. Collapse of the right atrium in systole



3. Respiratory variation in mitral valve inflow (akin to pulsus paradoxus)



4. Plethoraic inferior vena cava (< 50% collapsible with sniffing)

5. Decreased early filling (E wave) of mitral valve inflow (related to loss of the y descent).

MANAGEMENT

The role of pericardiocentesis depends on the presence of tamponade physiology and the size of effusion. Pericardiocentesis must be either fluoroscopy or echocardiography-guided under local anesthesia. Blind procedures must be restricted to very rare situations that are immediately life-threatening to avoid the risk of laceration of the heart or other organs.

PROCEDURE

The patient is propped up to a level of about 45 degrees. After sterile prep and draping, skin and subcutaneous tissues are infiltrated with lidocaine along the proposed path of entry. (fig1&2)

FLUOROSCOPIC-GUIDED PERICARDIO-CENTESIS

The lateral angiographic view provides the best visualization of the puncturing needle and its relation to the adjacent structures. Echocardiography can be used to confirm the direction of the needle and approximate depth of

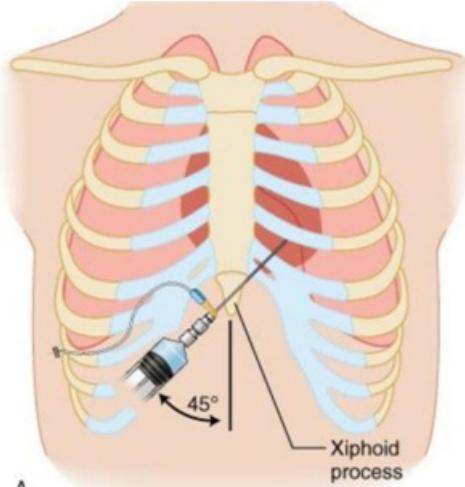


Fig 1: For the standard subxiphoid approach needle is inserted at 30-45° towards the posterior aspect of the left shoulder.

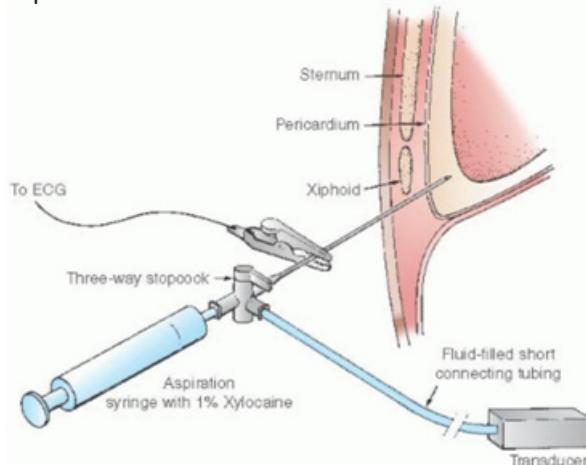


Fig 2: Subxiphoid approach for pericardiocentesis with ST-segment and pressure monitoring. A 16-gauge needle is connected via a 3-way stopcock to an aspiration syringe filled with saline and 1% xylocaine and a fluid-filled tube connected to a pressure transducer for measurement of intrapericardial pressure. Sterile ECG lead may be attached to the metal needle hub via an alligator clip to detect ventricular ectopic when the needle touches the epicardium.

insertion. The needle is slowly advanced under moderate suction until pericardial fluid is aspirated. If hemorrhagic fluid is freely aspirated, a few millilitres of contrast medium may be injected under fluoroscopic control to verify the position of the needle. A soft J-tip guidewire is then introduced and after dilatation is exchanged for a

multihole pigtail catheter, through which the fluid is evacuated with monitoring of intrapericardial pressure². Fluid aspiration should be done slowly as rapid removal of fluid can lead to acute ventricular dysfunction, pulmonary edema, and cardiogenic shock⁵.

ECHOCARDIOGRAPHIC-GUIDED PERICARDIOCENTESIS

The ideal site for needle entry would be the point where the effusion is closest to the transducer or body surface and where the fluid collection is maximal, without any intervening vital organ. Possible sites are sub-xiphoid, para-apical, left and right parasternal, left axillary, and posterolateral regions, avoiding vital structures such as the liver, lungs, myocardium, internal mammary artery (3–5 cm away from the parasternal border), and the neurovascular bundle at the inferior border of each rib (superior margin of intercostal space). (Fig 3)

Echocardiography-guided pericardiocentesis can be performed at the bedside during emergency situations. Pericardial space is entered using 16- or 18-gauge polytef sheathed needle and agitated saline can be injected through the sheath to confirm its position inside the pericardial space^{2,6}.

Resolution of tamponade physiology usually occurs after aspiration of 50 to 200 ml of fluid. With fall in intrapericardial pressure to a level of

d” 0 mm Hg, return of normal diastolic y descent in RA pressure waveform, resolution of pulsus paradoxus, and rise in systemic arterial blood pressure indicates relief of tamponade physiology².

A sample of fluid should be sent for analysis that can aid in the diagnosis of infectious, malignant, or cholesterol effusions. The drainage catheter is then sewn in place for periodic additional aspiration. Sterility must be strictly maintained during the whole procedure. The pericardial catheter is removed when the drainage has reduced to < 25 to 50 ml/24 hr with no signs of reaccumulating fluid on echocardiography. For recurrent large effusions and cardiac tamponade, a pericardial window can be created surgically or by video-assisted thoracoscopy or by percutaneous balloon pericardiotomy to make communication between pericardial space and pleural space for drainage of fluid ^{1,4}.

COMPLICATIONS

Pericardiocentesis is most likely to be uncomplicated if both anterior and posterior eco-free spaces are at least 10 mm. The most common complications include cardiac chamber puncture, arrhythmias, haemothorax, pneumothorax, pneumopericardium, hepatic injury, and injury to vessels⁶.

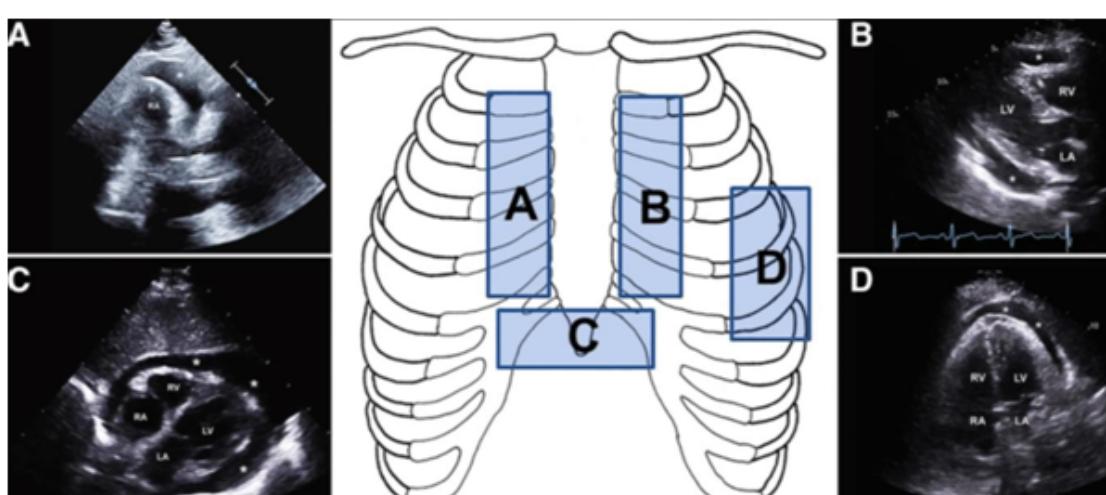


Fig 3: Sites for echocardiographic-guided pericardiocentesis. A. Right parasternal B. Left parasternal C. Subxiphoid D. Apical.

REFERENCES

1. Adler Y, Charron P, Imazio M, et al. Guidelines for the diagnosis and management of pericardial diseases: The Task Force for the Diagnosis and Management of Pericardial Diseases of the European Society of Cardiology (ESC). *Eur Heart J.* 2015;36:2921-2964.
2. Imazio M, Adler Y. Management of pericardial effusion. *Eur Heart J.* 2013;34:1186-1197.
3. Kearns MJ, Walley KR. Tamponade: Hemodynamic and Echocardiographic Diagnosis. *Chest.* 2018 May;153(5):1266-1275.
4. Maisch B, Seferovic PM, Ristic AD, et al. Guidelines on the diagnosis and management of pericardial diseases: executive summary; the task force on the diagnosis and management of pericardial diseases of the European society of cardiology. *Eur Heart J.* 2004;25:587-610.
5. Bernal JM, Pradhan J, Li T, et al. Acute pulmonary edema following pericardiocentesis for cardiac tamponade. *Can J Cardiol.* 2007;23:1155-1156.
6. Seward JB, Callahan JA, Sinak LJ, Daley JR, Schnidt L, Tajik AJ. 500 consecutive echo-directed pericardiocentesis. *J Am Coll Cardiol.* 1992;19(suppl A):356A.



Antepartum Hemorrhage

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Antepartum haemorrhage (APH) is defined as bleeding from or in to the genital tract, occurring from 24 weeks of pregnancy and prior to the birth of the baby. The most important causes of APH are placenta praevia and placental abruption, although these are not the most common. APH complicates 3–5% of pregnancies and is a leading cause of perinatal and maternal mortality worldwide.[1]

Causes of APH

1. Cervical and vaginal causes

- Cervical ectropion or 'erosion'

- Cervicitis
- Cervical polyps
- Cervical carcinoma(rare)

2. Placental causes

- Placenta praevia, placenta accreta, increta and placenta percreta
- Placental Abruption
- Vasa previa
- Uterine rupture (rare)

3. Trauma including domestic violence

Risk Factors for APH

PLACENTA PREVIA (2-8)	ABRUPTION	VASA PREVIA	UTERINE RUPTURE
Multiple pregnancy	Previous abruption	Velamentous insertion of cord	Congenital anomalies
Age>40	Hypertension	Succenturiate lobe	Previous scar (LSCS, myomectomy)
High parity	Pre eclampsia	Bilobed placenta	multiparity
Previous LSCS	Smoking	Placenta previa in the 2 nd trimester	Advanced age
Endometritis	Cocaine use, smoking	Multiple gestation	Abnormal placentation
Curettage	First trimester bleeding	IVF pregnancy	Gestation>40 weeks
Fibroid uterus	Coagulopathies		Obstructed labour
Previous placenta previa	Premature rupture of membranes		
Smoking	Advanced age		
Assisted reproduction	Multiple pregnancy		
Manual removal of placenta	Abdominal trauma		

Diagnosis and management of APH

Women who present with major or massive haemorrhage and signs of shock should be seen in a maternity unit with involvement of a multidisciplinary team.

The initial assessment of a woman with APH should include the ABC approach to stabilising the patient. Patients with major or massive obstetric haemorrhage should be managed in left lateral tilt to avoid exacerbatory hypotension secondary to uterine compression of the inferior vena cava.[9] If an adequate history cannot be provided due to maternal compromise, then resuscitation and stabilization of the mother is the key priority.

If there is no maternal compromise a full history should be taken. Examination of the woman should be performed to assess the amount and cause of APH. All women presenting with APH should have their pulse and blood pressure recorded.

As per RCOG guidelines ,investigations should be performed to assess the extent and physiological consequences of the APH.[10] Ultrasound can be used to diagnose placenta praevia but does not exclude abruption. Placental abruption is a clinical diagnosis and there are no sensitive or reliable diagnostic tests available. Ultrasound has limited sensitivity in the identification of retroplacental haemorrhage.In women who have experienced a massive blood loss or a major abruption, the development of a disseminated intravascular coagulation (DIC) should be considered. Clotting studies and a platelet count should be urgently requested.In cases of major or massive haemorrhage, blood should be analysed for full blood count and coagulation screen and 4 units of blood cross-matched. Urea, electrolytes and liver function tests should be assayed

An assessment of the fetal heart rate should be performed, usually with a cardiotocograph (CTG) in women presenting with APH once the mother is stable or resuscitation has commenced, to aid decision making on the mode of delivery. Whenever possible, CTG monitoring should be

performed where knowledge of fetal condition will influence the timing and mode of delivery. Ultrasound should be carried out to establish fetal heart pulsation if fetal viability cannot be detected using external auscultation.

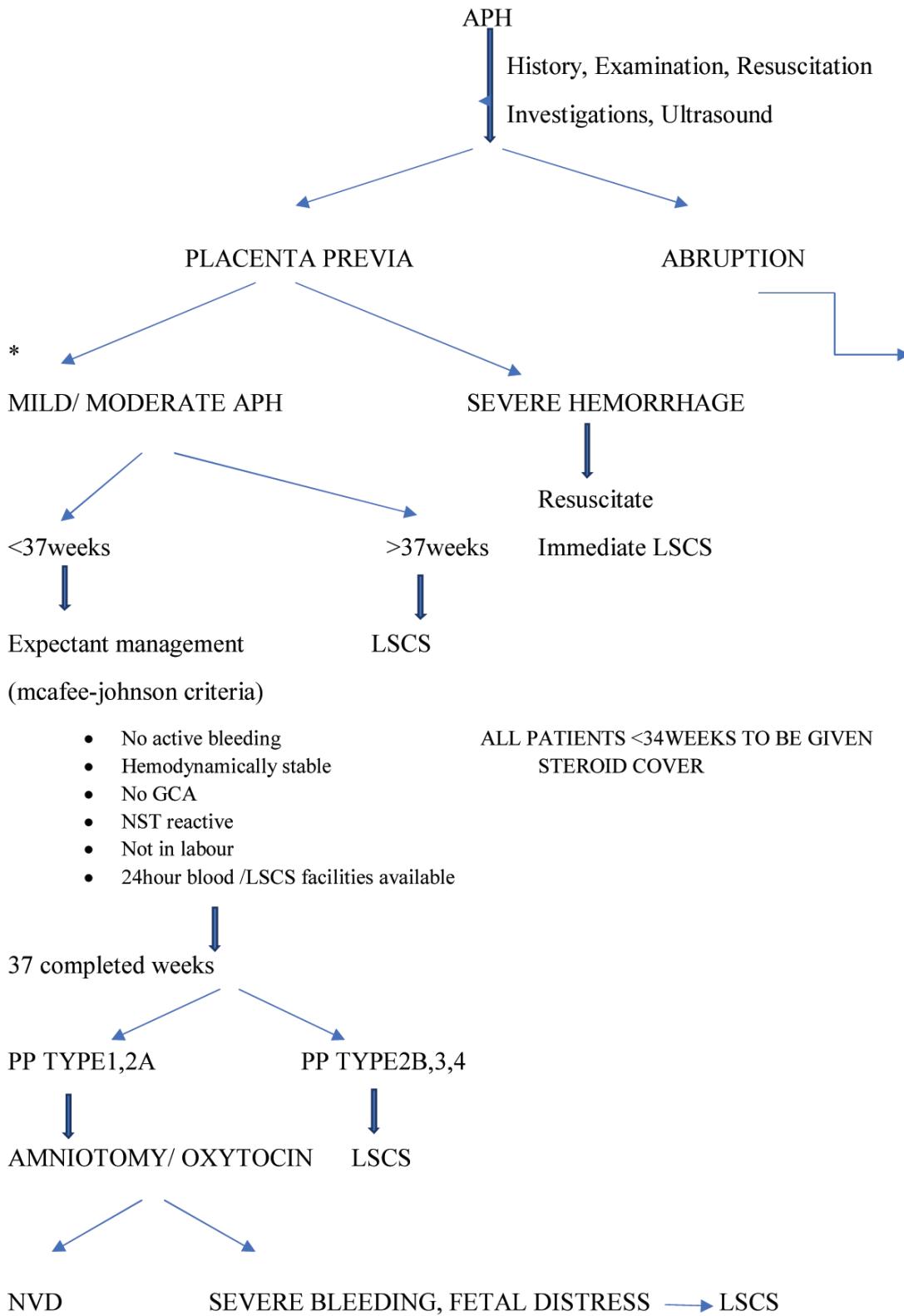
Postpartum haemorrhage (PPH) should be anticipated in women who have experienced APH. Women with APH resulting from placental abruption or placenta praevia should be strongly recommended to receive active management of the third stage of labour. Consideration should be given to the use of ergometrine-oxytocin, to manage the third stage of labour in women with APH resulting from placental abruption or placenta praevia in the absence of hypertension

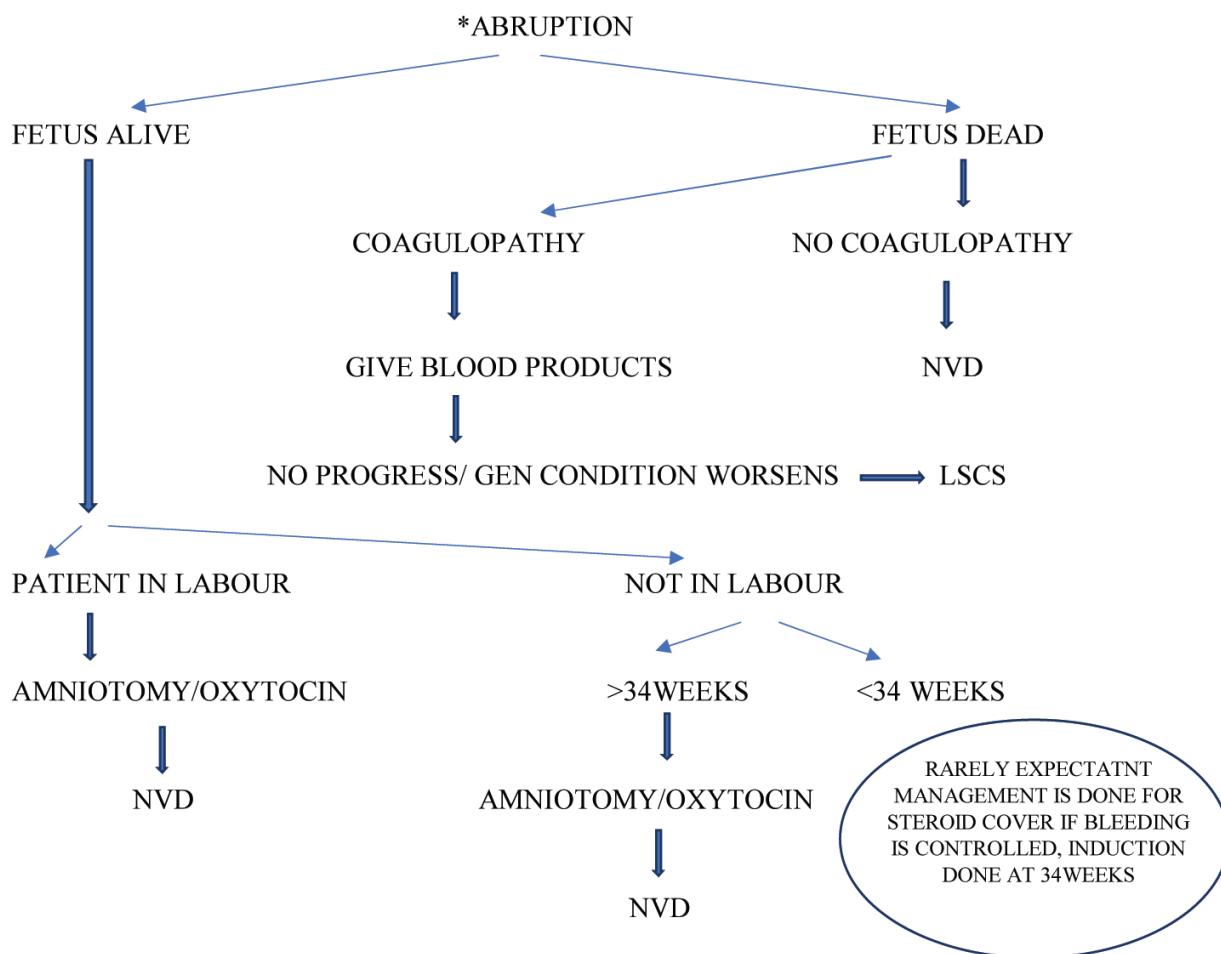
Anti-D Ig should be given to all non-sensitised RhD-negative women after any presentation with APH, independent of whether routine antenatal prophylactic anti-D has been administered.[11]

Bibliography

- 1) Calleja-Agius J, Custo R, Brincat MP, Calleja N. Placental abruption and placenta praevia. EurClinObstetGynaecol2006;2:121–7.
- 2) Parazzini F, Dindelli M, Luchini L, La Rosa M, Potenza MT,Frigerio L, et al. Risk factors for placenta praevia. Placenta1994;15:321–6.
- 3) Sheiner E, Shoham-Vardi I, Hallak M, Hershkowitz R, Katz M,Mazor M. Placenta previa: obstetric risk factors and pregnancy outcome.JMaternFetal Med 2001;10:414–9.
- 4) Faiz AS, Ananth CV. Etiology and risk factors for placenta previa: an overview and meta-analysis of observational studies.J MaternFetal Neonatal Med 2003;13:175–90.
- 5) Healy DL, Breheny S, Halliday J, Jaques A, Rushford D, Garrett C,et al. Prevalence and risk factors for obstetric haemorrhage in 6730 singleton births after assisted reproductive technology in Victoria Australia.HumReprod2010;25:265–74.
- 6) Rasmussen S, Albrechtzen S, Dalaker K.

MANAGEMENT OF APH





Obstetric history and the risk of placenta previa. Acta Obstet Gynecol Scand 2000;79:502–7.

- 7) Ananth CV, Smulian JC, Vintzileos AM. The association of placenta previa with history of cesarean delivery and abortion: a meta-analysis. Am J Obstet Gynecol 1997;177:1071–8.
- 8) Hendricks MS, Chow YH, Bhagavath B, Singh K. Previous cesarean section and abortion as risk factors for developing placenta previa. J Obstet Gynaecol Res 1999;25:137–42.
- 9) Royal College of Obstetricians and Gynaecologists. Prevention and Management of Postpartum Haemorrhage. Green-top Guideline No. 52. London: RCOG; 2009.

- 10) Antepartum haemorrhage RCOG green top guideline 63. 2011, https://www.rcog.org.uk/globalassets/documents/guidelines/gtg_63.pdf.
- 11) Royal College of Obstetricians and Gynaecologists. The Use of Anti-D Immunoglobulin for Rhesus D Prophylaxis. Green-top Guideline No. 22. London: RCOG; 2011



Acute Intestinal Ischemia

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1. Definition and inclusions

Let's start with the basics and elucidate the term "acute intestinal ischemia" and the spectrum of disorders it encompasses. The term ischemia refers to restriction of blood flow to an organ. The term "intestinal" could include any part of the gastrointestinal tract from mouth to anus. Lastly, with respect to intestinal ischemia, the term "acute" refers to a state where without intervention the bowel may proceed towards gangrene. This is in contrast to chronic ischemia where state of blood supply is chronically reduced and any event like eating which increases requirement of blood flow, induces pain[1]

Technically, acute intestinal ischemia should include all the ischemic conditions of esophagus, stomach and upto the colon but that is rarely the case. Esophagus, stomach duodenum and rectum rarely suffer from ischemia in view of their rich collateral supply. Also conditions like colonic ischemia[2], gastric ischemia and even small bowel ischemia due to localised infections, hernias and obstructions have a different pathology and management all together, and thus are rarely included in the group of acute intestinal ischemia. In this way 'acute mesenteric ischemia' seems like a better term as vascular events in mesentery lead to ischemia and rapidly progressive acute abdomen. Also Mesentery has been accepted as a separate organ in[3]. Thus 'acute mesenteric ischemia' is used interchangeably. The discussion hereon would limit to the vascular events in small and large bowel mesentery leading to acute abdomen.

2. Etiopathogenesis and presentation

Mesenteric vascular compromise would include

either venous outflow, arterial inflow or both. The presentation and management principles vary between the various types.

2.a. Mesenteric venous thrombosis

Thrombosis of the mesenteric venous system most commonly occurs in the superior mesenteric vein to portal vein axis. Less than 10% include the inferior mesenteric vein and even lesser are clinically significant[4]. Indian data suggests higher number of venous occlusions of upto 40% or more compared to western data where they comprise of 10-15% of all the ischemic events[5]. Venous occlusions have primary or secondary causes. Primary defects in coagulation and other idiopathic causes can lead to spontaneous thrombosis in mesenteric veins, whereas malignancies, trauma including surgery and localised infections are some examples of secondary causes.

This outflow obstruction lead to significant bowel and mesenteric edema with acute abdominal pain out of proportion to clinical signs. This further leads to an increased retrograde pressure and resultant arteriolar obstruction with vascular compromise. The progression of this ischemia is slower and leads to gangrene and development of peritoneal signs in 1-2 days. Intra-operatively, the gangrene is widespread with ill defined margins with normal bowel. The location of thrombus determines the rapidity of deterioration and the spread of gangrene. The distal venular thrombi lead to quicker ischemia albeit to a localised segment. On the other hand proximal blocks with extensive thrombi including portal, splenic and mesenteric veins are the ones which lead to gangrene of a devastatingly large segment of bowel sometimes including segments of

proximal most jejunum and duodenum with very difficult intraoperative management and poor postoperative outcomes[6].

2.b. Mesenteric arterial obstructions

Arterial causes included obstruction due to embolus , thrombus or vasospasm. Embolic obstructions are due to clots given out by heart or proximal thrombi. Due to low takeoff angle of superior mesenteric artery it becomes the most common site for embolic entry. The embolus usually obstructs the artery after takeoff of few proximal branches like middle colic artery and proximal jejunal branches. This becomes the reason for sparing of mid colon and proximal jejunal segments and thus helping in surgical management[7]. Multiple emboluses are also common and upto 20% patients might have multiple affected segments.

Thrombotic plaques are present at the proximal ostial openings of superior and inferior mesenteric arteries. These patients do have a associated history suggestive of chronic mesenteric ischemia. Rupture of plaques or an associated thrombotic or embolic event leads to an added acute or chronic event. These blockages are proximal and extensive leading to involvement of proximal jejunal segments upto duodenum and massive gangrene of the whole SMA territory from duodenojejunal flexure to mid transverse colon. Non obstructive vaso-occlusive arterial obstruction is a separate entity which occurs in terminally ill patients with very poor prognosis and will be discussed in the end.

Inferior mesenteric arterial obstructions are rarely a source of concern. The marginal artery of Drummond along the colon is very well capable of keeping the colon safe in such conditions. On the other hand in superior mesenteric artery obstructions the arc of Riolan and meandering mesenteric artery can let an unobstructed IMA to help out in proximal SMA obstruction[8].

2.c. Arterial versus venous ischemia

Arterial gangrenes are associated with quick onset and rapid progression of signs and symptoms.

The gangrenous areas are well demarcated and associated ascites and mesenteric edema is less. This makes resections technically easier and primary anastomosis are attempted with fewer re-laparotomies than venous causes. Venous thrombosis on other hand progress slowly and the ongoing gangrenous process might be surgically intervened at any stage. This progressive phenomenon leads to ill defined margins, unsurety about the results of primary anastomosis and higher rate re-laparotomies. The Mesentery is bulky and thrombi exudes out of terminal mesenteric vessels during resection. Ascites develops early and hemorrhagic component suggests transmural involvement of bowel ischemia. It is advised to resect the bowel to the point where these terminal capillary thrombi stop to appear.

2.d. Microscopic changes, clinical presentation and investigations

Splanchnic circulation is upto 25% of the cardiac output which can increase upto 200% in fed states. The bowel has low oxygen extraction ratios to leave the rest for liver. Most of the oxygen requirement is by mucosa and submucosa (~>80%) and thus these are the layers which get affected the first in total vascular occlusion. Within 15 mins villi shows loss of structural integrity and mucosal sloughing starts in 3 hours. A total occlusion can lead to transmural involvement in 6 hours , whereas bowel can tolerate a 75% reduction in blood flow upto 12 hours or so.[9]

Patients present with a spectrum of signs and symptoms depending upon the progression of intestinal ischemia. Pathologically they will progress from mucosal ischemia, to transmural necrosis, to perforation and fecal contamination. Clinically these patients might come walking with stable vitals and only central abdominal pain to presentation with fever, toxemia and shock! Their blood tests might not show any abnormality to having acidosis, elevated lactates, leuckocytosis and raised creatinine. There is no single test or an early marker that can rule in or rule out acute intestinal ischemia. Various tests at different stages can detect the condition.

PET CT and some newly discovered biochemical markers can be one of the earliest tests that can detect intestinal ischemia , but in emergency settings their role remains best experimental.[8,10] Xray can detect thumbprinting of intestines and portal gas but these are rare, are frequently missed and are exposure dependent. Pneumoperitoneum in xrays is a late finding and warrant exploration. Raised leucocytes, elevated lactates, CRPs, acidosis , altered renal and liver function tests, raised serum amylases, elevated D-dimers, all these tests alongwith relevant clinical findings can point towards ischemic intra-abdominal pathology but doesn't confirm it. Contrast enhanced CT abdomen is the investigation of choice as it can confirm the diagnosis with sensitivity and specificity over 95%[8]. It can show thickened non enhancing bowel loops , hyperenhancing loops with reperfusion, pneumatisis intestinalis, portal gas and associated ascitis. It also helps to look for thrombus or embolus in mesenteric vasculature thus helping in elucidating the cause and guiding in intraoperative management strategies regarding perfusion. The simplest protocol involves just doing a CECT abdomen whenever clinical findings are suggestive. A negative enteral contrast to distend the bowel loops would be helpful but is not advisable in emergency settings. A compromised renal function becomes the main hinderance in this protocol where other measures like non-contrast CT , diagnostic aspiration of ascites, color doppler of mesenteric vessels, and even diagnostic laparoscopy has to be considered.

3. Management principles and surgical interventions

Keeping intestinal ischemia in differentials at the time of presentation and getting a timely Contrast enhanced CT abdomen goes a long way in management of these patients. Also the clinical stage at which the patients presents decides the treatment approaches. Two scenarios should be discussed at this stage. First a pre-morbid patient with advanced age, MODS, and poor performance status might be better served with palliative care than invasive procedure where chances of short term and long term survival is scarce. Second

during surgery if massive bowel gangrene is encountered and prolonged par-enteral nutrition and intestinal transplant is not a viable option in view of comorbidities, short life span, financial constraints or unavailability of these therapies in that local or national setup; an "open and close" or abandoning the surgery might be considered[7].

3.a Venous obstructions

Discussing the management of venous obstructions leading to intestinal ischemia, lot of factors come into play. Firstly acute venous thrombosis , especially smaller venules and SMV leads to ischemia and gangrene. On the other hand PV thrombosis , or acute or chronic thrombosis with preformed collaterals might save the intestine from going into critical ischemia. Anticoagulation is the cornerstone of treatment in these cases and should be started as soon as the diagnosis is confirmed(ref). If possible a thrombotic profile should be sent before starting anticoagulation to look for primary genetic causes. Even with increased bleeding chances, anticoagulation should be given in perioperative periods and continued post operatively. A patient who presents early with reversible ischemic changes might improve completely on anticoagulation and the patient who requires surgical intervention should be given perioperative anticoagulation to prevent further progression of thrombus[11]. Intraoperative vessel clearance by thrombectomy and lytic therapies is not accepted as success rate is low and re-thrombus occurs soon after intervention with no improvement in overall survival[7]. That being said catheter directed thrombolytic therapies are done in rare cases with extensive thrombosis in PV-SMV system , in cases with thrombus progression despite being on heparin, and patients experiencing continuous pain on anticoagulation. These lytic therapies can be administered through transjugular- tranhepatic route, through end branches of SMA, or intraoperatively direct into SMV- PV axis[12].

CT shows bowel wall thickness greater than 3mm , indistinct bowel margins, new or unexplained ascites and thickened mesentery associated with

non enhancement of the draining veins. Surgically we explore the patient with laparotomy , although diagnostic laparoscopy can be considered in doubtful cases. Gangrenous or perforated bowels are resected and vessels are ligated. Resections are done to the point where the mesentery starts to appear normal and thrombi stop exuding out of small venules. SMA pulsations can be felt on palpation and massive ascites is usually encountered.

3.b Anticoagulation in venous thrombosis

For therapeutic anticoagulation heparin should be started at 80 IU/kg bolus with 18IU/kg maintenance done and APTT should be repeated every 4th hourly with target of 50-70 secs. It can be stopped or reversed if emergency surgery is contemplated . Post surgery LMWH can be started once patient is stabilised , bowel functions have started and renal compromise is not an issue. In cases of known genetic thrombotic syndromes anticoagulation is continued for life. In cases of known trauma or cause it can be stopped after 3 months. In idiopathic cases a call for 3 months vs lifelong anticoagulation has to be taken[11]. Here the role of pre anticoagulation thrombotic profile comes into play which can help in this dilemma. In cases where it was not done preoperatively , postoperatively after 3months it can be done after stopping the anticoagulation therapy for 1 week.

3.c Mesenteric arterial occlusions

In arterial obstructions revascularisation of bowel is a major goal. The success and strategy of revascularisation depends upon the causative pathology of arterial obstruction. The timing and approach of revascularisation depends upon the clinical state the patient is in and the pathological stage the ischemia is in. In patients who present early where full thickness ischemia has not set in are candidates for percutaneous interventions. Patients who present with acute abdomen require surgery and decision for revascularisation is taken during surgery. For sick and unstable patients we need a damage control surgery with salvage of the patient first and consideration for revascularisation later.

Embolectic blockages require abdominal exploration and revascularisation is attempted with open embolectomy of SMA. After this revascularisation of bowel is assessed clinically after 20-30 minutes with overt colour changes , doppler signals at anti-mesenteric border or obvious pulsations in distal mesentery. In cases of perforation the bowel resection is dealt with first. Open resection is the preferred approach in embolic arterial blocks , but rarely in early presentations percutaneous thrombectomy or thrombolysis can be attempted[13]. Prolonged endovascular procedures can risk further bowel injury and are not the first line management in these cases

On the other hand thrombotic blocks are best treated with endovascular approaches like thrombolysis and stenting. Disadvantage is that it should be undertaken when full thickness ischemia is not present, also it is not possible to assess the bowel condition to surety , thus the threshold for open procedure should be low. Most of the cases present with late signs like peritonitis and laparotomy is undertaken to remove the dead bowel. Surgical revascularisation techniques are extensive and involve autologous and prosthetic shunt reconstructions with aorta. Not only they are technically challenging and time taking , using prosthetic grafts in infected field is not uniformly accepted. Retrograde arterial stenting with embolectomy is a quicker option in these patients where a single source of thrombosis is present proximally[14]. All this said; in unstable and septic patients that usually present to us , surgical vascular reconstruction is not a viable option and most of these patients go for endovascular revascularisation approaches after surgery and stabilisation. For those centers where vascular surgical team expertise is not there it is recommended to resect the dead bowel , stabilise the patient and transfer to higher center.

3.d Surgical principles in intestinal ischemia and peri-operative management

Most of the patients present with acute abdomen warranting laparotomy. We resuscitate the patient with IV fluids , antibiotics, and supplementary oxygen while preparing the OR for laparotomy

and ensuring a bed in ICU. We explore the patient with a midline incision and routinely send ascitic fluid for culture. In cases where significant proximal bowel is healthy we do not usually attempt anastomosis and create 2 ostomies on both sides of the laparotomy scar of proximal and distal bowel, or a double barrel stoma if bowel edema permits. In very proximal gangrenes where an ostomy would make thriving difficult, a hand-sewn anastomosis is done with high expectant leak rates. In cases where line of demarcation with normal bowel is well defined and the patient's physiology is well preserved we do go ahead with primary anastomosis explaining them the risk re-laparotomy and stoma formation. Also re-vascularisations are attempted in clinically well preserved patients only. Postoperatively we involve medical gastroenterologists, dieticians , stoma therapists , psychiatrists and nurse staff to manage the patients. The need for prolonged treatment and multiple surgeries is explained well in advance. If stoma output is high despite medical therapies we start re-feeding of the bowel contents into the distal bowel ostomy. Our medical management mimics the management for a short bowel syndrome patient including anti-motility agents , double dose PPIs, high salt content of diet and staged feeding programmes from elemental to complex diets. In addition we re-feed chyme in distal bowel in very proximal ostomy cases and we also feed elemental diet and rehydrations solutions into the distal bowel if needed. Keeping fluid, electrolyte and caloric balance in check is paramount. These patients with ostomies require re-laparotomy and restoration of continuity which we undertake in minimum 4-6 weeks after the procedure. These intervening weeks and months are critical where macro and micronutrients, hydration and electrolytes, and associated disorders of a short gut have to be managed. Along-with psychological issues and financial constraints become a source of concern. This first surgical procedure is not the only source of mortality in these patients, rather this interviewing period, further surgeries and financial and social issues is a sizeable component.

4. Damage control laparotomy

A very sick and unstable patient in coagulopathy might require a damage control laparotomy. Here the abdomen is entered and only source control is done. Source control involves resecting the dead and perforated bowel with ligating the ends of healthy bowel with temporary closure of abdomen. Quick Revascularisation procedure like embolectomy can be attempted. Patient is stabilised in intensive care units and relook laparotomy is attempted after 48-72 hours[15].

5. Non obstructive mesenteric ischemia

Non obstructive mesenteric ischemia should be dealt separately as it is a rare entity with a different cause , diagnostic tests and treatment approaches. It comprises less than 2% of all intestinal ischemias and occur in patients who are critically ill in intensive care units. These patients often are above 50years of age , have comorbidities and are generally having cardiac , renal and hepatic issues. There is insidious but progressive vasospasm in splanchnic arteries and arterioles leading to progressive intestinal ischemia. Diagnosis is delayed in view of various factors. Critically ill patients in ICU are sometimes unable to properly voice their concerns, and the signs and symptoms get clouded with other disease processes. Also the clinical suspicion of a rare diagnosis remains low. Contrast enhanced imaging modalities of abdomen can suggest towards the diagnosis which is confirmed by conventional angiogram. Conventional angiography is the gold standard in diagnosis and also therapeutic as intravascular administration of papaverine or prostaglandin is the treatment. The catheter is kept in place and papaverine administration is repeated as and when required . Clinical improvement of the patient as a whole is essential for resolution of the condition[16].

Conclusions:

Acute intestinal ischemia requires Resuscitation and Rapid diagnosis. Once that is done next step is Revascularisation and Resection and if required Re-look laparotomy to salvage the patient. Postoperative Rescue and support is equally

important and lastly Restoration of bowel continuity which completes the surgical cycle.

Abbreviations

MODS- multiorgan dysfunction syndrome

CRP- C reactive protein

SMA- superior mesenteric artery

SMV- superior mesenteric vein

PV- portal vein

IMA- inferior mesenteric artery

PET CT - Positron emission tomography, computed tomography

ICU- intensive care unit

CECT- contrast enhanced CT

PPI- proton pump inhibitor

APTT- activated prothrombin time

LMWH- low molecular weight heparin

References

1. Char D, Hines G. Chronic mesenteric ischemia: diagnosis and treatment. Heart Dis. 2001 Jul-Aug;3(4):231-5. doi: 10.1097/00132580-200107000-00005
2. Update on the Diagnosis and Management of Colon Ischemia Update on the Diagnosis and Management of Colon Ischemia Curr Treat Options Gastroenterol. 2016 Mar;14(1):128-39. doi: 10.1007/s11938-016-0074-2. Authors Ann D Flynn 1, John F Valentine 2
3. The mesentery: structure, function, and role in disease. Coffey, J Calvin et al.The Lancet Gastroenterology & Hepatology, Volume 1, Issue 3, 238 - 247
4. Medina-Perez R, Campbell DJ, Acosta Rullan JM, Gonzalez S. Partial Thrombosis of Inferior Mesenteric Vein With Thrombophlebitis. Cureus. 2021 Aug 5;13(8):e16900. doi: 10.7759/cureus.16900
5. Nagaraja R, Rao P, Kumaran V, Yadav A, Kapoor S, Varma V, Mehta N, Nundy S. Acute Mesenteric Ischaemia-An Indian Perspective. Indian J Surg. 2015 Dec;77(Suppl 3):843-9. doi: 10.1007/s12262-014-1034-5. Epub 2014 Jan 22..
6. Hmoud B, Singal AK, Kamath PS. Mesenteric venous thrombosis. J Clin Exp Hepatol. 2014 Sep;4(3):257-63. doi: 10.1016/j.jceh.2014.03.052. Epub 2014 Apr 13.
7. Bala, M., Kashuk, J., Moore, E.E. et al. Acute mesenteric ischemia: guidelines of the World Society of Emergency Surgery. *World J Emerg Surg* **12**, 38 (2017). <https://doi.org/10.1186/s13017-017-0150-5>
8. Olson MC, Fletcher JG, Nagpal P, Froemming AT, Khandelwal A. Mesenteric ischemia: what the radiologist needs to know. Cardiovasc Diagn Ther. 2019 Aug;9(Suppl 1):S74-S87. doi: 10.21037/cdt.2018.09.06.
9. Matheson PJ, Wilson MA, Garrison RN. Regulation of intestinal blood flow. J Surg Res. 2000 Sep;93(1):182-96. doi: 10.1006/jsre.2000.5862.
10. Smith MV, Yang M, Roarke MC. Identification of Acute Mesenteric Ischemia on 18F-FDG PET/CT. Clin Nucl Med. 2022 Jan 1;47(1):e103-e104. doi: 10.1097/RLU.0000000000003819.
11. Kim HK, Chun JM, Huh S. Anticoagulation and delayed bowel resection in the management of mesenteric venous thrombosis. World J Gastroenterol. 2013 Aug 14;19(30):5025-8. doi: 10.3748/wjg.v19.i30.5025.
12. M. Marshad, M. Maresch, T. Al Abbasi, Intraoperative catheter directed thrombolytic therapy for the treatment of superior mesenteric and portal Vein thrombosis, International Journal of Surgery Case Reports, Volume 53, 2018, Pages 242-245, ISSN 2210-2612, <https://doi.org/10.1016/j.ijscr.2018.09.030>.

13. Klar E, Rahamanian PB, Bücker A, Hauenstein K, Jauch KW, Luther B. Acute mesenteric ischemia: a vascular emergency. *Dtsch Arztebl Int.* 2012 Apr;109(14):249-56. doi: 10.3238/arztebl.2012.0249. Epub 2012 Apr 6.
14. Do N, Wisniewski P, Sarmiento J, Vo T, Aka PK, Hsu JH, Tayyarah M. Retrograde superior mesenteric artery stenting for acute mesenteric arterial thrombosis. *Vasc Endovascular Surg.* 2010 Aug;44(6):468-71. doi: 10.1177/1538574410366168. Epub 2010 May 18.
15. Brillantino A, Lanza M, Antropoli M, Amendola A, Squillante S, Bottino V, Renzi A, Castriconi M. Usefulness of damage control approach in patients with limited acute mesenteric ischemia: a prospective study of 85 patients. *Updates Surg.* 2022 Feb;74(1):337-342. doi: 10.1007/s13304-021-01192-3. Epub 2021 Oct 22.
16. Trompeter M, Brazda T, Remy CT, Vestring T, Reimer P. Non-occlusive mesenteric ischemia: etiology, diagnosis, and interventional therapy. *Eur Radiol.* 2002 May;12(5):1179-87. doi: 10.1007/s00330-001-1220-2. Epub 2001 Dec 21.

Post Partum Hemorrhage

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DEFINITION

Postpartum hemorrhage (PPH) is an obstetric emergency complicating 1%–10% of all deliveries.¹ It continues to be the leading obstetric cause of maternal death.

Classically, it was defined as quantified bleeding of more than 500 ml for vaginal deliveries and more than 1000 ml for cesarean deliveries, occurring within the first 24 h of delivery.

In 2017, the American College of Obstetricians and Gynecologists (ACOG) changed the definition to blood loss of more than or equal to 1000 ml, or blood loss that was accompanied by signs or symptoms of hypovolemia occurring within 24 h after birth, regardless of the mode of delivery.(2)

ETIOLOGY

The following are the most common causes of post partum hemorrhage :

1. TONE- uterine atony accounts for nearly 70 percent cases of PPH (3). It can be anticipated after prolonged labour, multiple fetal gestation, polyhydramnios, macrosomia, high parity, general anesthesia.
2. TRAUMA- accounts for 15-20 percent of all cases. It can be attributed to perineal or cervical lacerations, perineal hematomas or uterine rupture.
3. ISSUE – retained products of conception can increase the risk of PPH by 3.5 times. Risk factors include succenturiate placenta or any previous instrumentation. (4)

4. THROMBUS- Coagulation defects can also lead to PPH. They can be inherited or acquired disorders.

Inherited disorders include von willibrand disease, hemophilia and idiopathic thrombocytopenic purpura. Acquired disorders include use of anticoagulant and DIC after abruption.

MANAGEMENT – PREVENTION

Active management of third stage of labour plays the most important role for the prevention of PPH. The use of uterotronics for prevention of PPH during the third stage of labor is recommended for all births. Oxytocin (10 IU intravenously/intramuscularly [IV/IM]) is recommended for the prevention of PPH for vaginal delivery and cesarean section.(5)

1. In settings where oxytocin is unavailable or its quality cannot be guaranteed, the use of other injectable uterotronics (if appropriate ergometrine/methylergometrine 200 µg IM/IV; hypertensive disorders can be safely excluded prior to its use) or oral misoprostol (400–600 µg orally) or carbetocin 100 µg IM/IV is recommended for the prevention of PPH(5)
2. The combinations of ergometrine plus oxytocin or misoprostol plus oxytocin may be more effective uterotonic drug strategies for the prevention of PPH e "500 ml compared with the current standard, oxytocin. This comes at the expense of a higher risk of adverse effects (vomiting and hypertension with ergometrine and fever with misoprostol).

3. In settings where skilled birth attendants are not present to administer injectable uterotronics and oxytocin is unavailable, the administration of misoprostol (400–600 µg orally) by community healthcare workers and lay health workers is recommended for the prevention of PPH.
4. In settings where skilled birth attendants are unavailable, controlled cord traction (CCT) is not recommended.
5. Sustained uterine massage is not recommended as an intervention to prevent PPH in women who have received prophylactic oxytocin
6. Postpartum abdominal uterine tonus assessment for early identification of uterine atony is recommended for all women
7. Oxytocin (IV or IM) and CCT is the recommended method for removal of the placenta for the prevention of PPH in cesarean delivery.

TREATMENT OF POSTPARTUM HEMORRHAGE – PPH BUNDLE

1. First line emergency interventions for PPH (ATONIC) includes uterine massage, initiation of large intravenous access, emptying the uterus and the bladder and administration of oxytocin and tranexamic acid (first response bundle). If the uterus fails to contract, ergometrine or misoprostol should be administered, bimanual uterine or aortic compression should be initiated, a UBT placed And NASG applied (response to refractory PPH bundle). These two sets of approaches have recently been defined as **PPH Bundle.** (7)
2. Intravenous oxytocin alone is the recommended first-line uterotonic drug for the treatment of PPH.(4) . If intravenous oxytocin is unavailable, or if the bleeding does not respond to oxytocin, the use of intramuscular ergometrine, oxytocin-ergometrine fixed

dose, or a prostaglandin drug (including sublingual misoprostol, 800µg) is recommended.

3. The use of isotonic crystalloids is recommended in preference to the use of colloids for the initial intravenous fluid resuscitation of women with PPH. (4)
4. Early use of intravenous tranexamic acid as soon as PPH is diagnosed but within 3 h of birth in addition to standard care is recommended for women with clinically diagnosed PPH following vaginal birth or cesarean delivery.. Administration of 1 g (100 mg/ml) tranexamic acid intravenously at 1 ml/min (i.e. administered over 10 min), with a second dose of 1 g in-travenously if bleeding continues after 30 min.(8,9)

NON SURGICAL MEASURES FOR PPH TREATMENT

1. NASG- Nonpneumatic antishock garment- The NASG is a low-technology, affordable, first-aid compression device for the management and stabilization of women with hypovolemic shock due to PPH and management of refractory PPH.The NASG is a lightweight, lower body compression device comprising six articulated neoprene and hook-and-loop fastener segments that provide lower body circumferential counterpressure to improve cardiac output and blood pressure.The estimated circumferential pressure applied by the NASG is around 20–40 mm Hg. Direct abdominal and pelvic compression reduces the total vascular space in the lower body and decreases pelvic perfusion to pelvic compartment organs and smaller pelvic blood vessels, promoting hemorrhage arrest. Additionally, the pressure applied increases cardiac output and the central circulation, allowing an increased distribution of blood flow to vital upper body organs (heart, lung, brains) and contributing to a rapid recovery from

shock. Likewise, the direct compression of the descending aorta reduces bleeding from the uterine arteries and the mesenteric bed's vasculature, perhaps decreasing up to 90% of blood flow at the level of the superior mesenteric artery. Additionally, it is extremely cost-effective even for very low-resource settings, and is reusable as it can be disinfected and washed over 100 times without losing its compressive effects.

2. UTERINE BALLOON TAMPONADE

Despite active management of the third stage of labor, 2%–7% of women experience postpartum blood loss of more than 500 ml.¹ Uterine balloon tamponade (UBT) is both a diagnostic and therapeutic tool. If bleeding does not stop after its insertion, then it is better to review the etiology of PPH. Placement of a UBT device should be considered early when emergency measures for management of PPH are initiated.

3. UTERINE ARTERY EMBOLIZATION

Uterine artery embolization has been shown to be effective in the treatment of various causes of PPH including placenta accreta: Both UAE and uterine artery ligation have reported success rates of greater than 90% with low complication rates. If both techniques are available, embolization is the preferred first choice as it obviates laparotomy. Ligation can be attempted subsequently if embolization is unsuccessful. In contrast, after an unsuccessful uterine artery ligation, embolization may be extremely difficult or even impossible, leaving hysterectomy as the only remaining option.

SURGICAL TREATMENT

In all cases of post partum the uterus should be fully exteriorized, if possible. Blood loss should be minimized while fully extending the uterus, causing vasoconstriction and a tourniquet placed

around the low segment until surgical compression sutures are successfully applied. Placement of endouterine square hemostatic sutures may also be used to control bleeding from the placental bed.

If compression sutures fail, next step involves step wise devascularization. First step involves uterine artery ligation (first unilateral and then bilateral).

Internal iliac artery ligation is the next step in devascularization if hysterectomy is to be avoided. It reduces pelvic pressure by half and pulse pressure by 85 %, thereby simulating a venous rather than arterial circulation and achieving hemostasis.

Peripartum hysterectomy is performed in the treatment of pph when other conventional methods to control bleeding have failed. The most common indications for peripartum hysterectomy are abnormal placentation, placenta previa and placental abruption (38 %), uterine atony (27 %) and uterine rupture (26 %)

CARBETOCIN FOR PREVENTION OF PPH

Carbetocin, is a long-acting synthetic analog of oxytocin, having similar pharmacologic properties to those of natural oxytocin, but having a longer half life (40 minutes). It binds to smooth muscle receptors of the uterus and has been reported to produce a tetanic uterine contraction within two minutes, lasting approximately six minutes, followed by rhythmic contractions for one hour. It has been recommended for the prevention of PPH by WHO (5)

Potential advantages of carbetocin are its long duration of action and availability of a heat-stable formulation. Heat-stable carbetocin is advantageous in resource-limited areas where medications requiring refrigeration are not practical; however, it requires parenteral injection

Carbetocin 100 mcg is administered IV over 30 to 60 seconds [10,11] It can also be given by IM injection. The dose is the same after vaginal birth, scheduled cesarean birth, and intrapartum cesarean birth . Lower doses may be sufficient,

and repeated if required, up to a total maximum cumulative dose of 100 mcg. The long duration of action eliminates the need for an infusion after the initial dose.

References

1. Borovac-Pinheiro A, Pacagnella RC, Cecatti JG, et al Postpartum hemorrhage: new insights for definition and diagnosis. *Am J Obstet Gynecol.* 2018;219:162– 16
2. Committee on Practice Bulletins-Obstetrics. Practice bulletin no. 183: postpartum hemorrhage. *Obstet Gynecol.* 2017;130:e168– e186
3. Oyelese Y, Anant CV. Postpartum hemorrhage: epidemiology, risk factors, and causes. *Clin Obstet Gynecol.* 2010;53:147– 15
4. Sheiner E, Sarid L, Levy A, Seidman DS, Hallak M. Obstetric risk factors and outcome of pregnancies complicated with early postpartum hemorrhage: a population-based study. *J Matern Fetal Neonatal Med.* 2005;18:149– 154.
5. World health organizations, WHO recommendations: uterotonic for the prevention of post partum hemorrhage. Accessed AUGUST 2021.
6. Escobar MF, Fuchner CE, Carvajal JA, Et al- experience in the use of Non pneumatic anti shock garment in the management of post partum hemorrhage with hypovolemic shock in the Fundacion Valle Del Lili, Cali, Columbia. *Reprod Health.*2017; 14:58.
7. Althabe F, Therrien MNS, Pingray V, Hermida J, Gülmezoglu AM, Armbruster D, Singh N, Guha M, Garg LF, Souza JP, Smith JM, Winikoff B, Thapa K, Hébert E, Liljestrand J, Downe S, Garcia Elorrio E, Arulkumaran S, Byaruhanga EK, Lissauer DM, Oguttu M, Dumont A, Escobar MF, Fuchner C, Lumbiganon P, Burke TF, Miller S. Postpartum hemorrhage care bundles to improve adherence to guidelines: A WHO technical consultation. *Int J Gynaecol Obstet.* 2020 Mar;148(3):290-299. doi: 10.1002/ijgo.13028. Epub 2019 Dec 23. PMID: 31709527; PMCID: PMC7064978.
8. WOMAN Trial Collaborators (2017) Effect of Early Tranexamic Acid Administration on Mortality, Hysterectomy and Other Morbidities in Women with Post-Partum Haemorrhage (WOMAN): An International, Randomised, Double-Blind Placebo-Controlled Trial. *The Lancet.* 389, 2105-2116.
9. World Health Organization. WHO recommendation on tranexamic acid for the treatment of postpartum hemorrhage. Accessed August 11, 2021. <https://www.who.int/reproductivehealth/publications/tranexamic-acid-pph-treatment/en/>
10. Rath W. Prevention of postpartum haemorrhage with the oxytocin analogue carbetocin. *Eur J Obstet Gynecol Reprod Biol* 2009; 147:15.
11. Hunter DJ, Schulz P, Wassenaar W. Effect of carbetocin, a long-acting oxytocin analog on the postpartum uterus. *Clin Pharmacol Ther* 1992; 52:60.

Ectopic Pregnancy

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An ectopic pregnancy is an extrauterine pregnancy. Almost all ectopic pregnancies occur in the fallopian tube, but other possible sites include cervical, interstitial, hysterotomy (cesarean) scar, ovarian, or abdominal(1). In rare cases, a multiple gestation may be heterotopic (include both an intrauterine pregnancy [IUP] and an extrauterine pregnancy)(2). The overall rate of EP is 1–2 % in the general population, and 2–5 % among patients who have utilized assisted reproductive technology (ART). Although the overall mortality has decreased over time, ruptured EPs still account for up to 6 % of all maternal deaths(3).

Clinical presentation – Abdominal pain and/or vaginal bleeding are the most common symptoms of ectopic pregnancy. Ectopic pregnancy should be suspected in any patient of reproductive age with these symptoms, especially those who have risk factors for ectopic pregnancy. However, over 50 percent of patients are asymptomatic before tubal rupture and do not have an identifiable risk factor for ectopic pregnancy(4).

Diagnosis – In a pregnant patient with no IUP on transvaginal ultrasound (TVUS), the diagnosis of ectopic pregnancy is:

Suspected when any of the following are present(5):

- An extraovarian adnexal mass or intraperitoneal bleeding on TVUS.
- An abnormally rising serum human chorionic gonadotropin (hCG) level.
- Abdominal pain and/or vaginal bleeding in a patient with risk factors for ectopic

pregnancy.

Confirmed when any of the following are present(6):

- An extrauterine gestational sac with a yolk sac or embryo on TVUS.
- No products of conception are identified on uterine aspiration (if performed).
- Ectopic pregnancy tissue is visualized at surgery with histologic confirmation following resection.

Hemodynamically unstable patients – Patients with ectopic pregnancy may become hemodynamically unstable if there is a rupture of, and hemorrhage from, the structure in which the pregnancy is implanted, usually the fallopian tube

- Rupture should be suspected in patients with a sudden onset of severe and persistent abdominal pain, symptoms of faintness, or vital signs suggestive of hemodynamic compromise (eg, hypotension, tachycardia). These patients should be evaluated immediately in an emergency department.
- A ruptured ectopic pregnancy is a clinical diagnosis made primarily based on a finding of echogenic fluid (consistent with blood) in the pelvic cul-de-sac (and typically also surrounding the uterus and adnexa on the side of the patient's pain) and/or abdomen on focused abdominal ultrasound combined with the presence of abdominal pain and/or tenderness(7).
- If rupture is suspected, the patient should undergo emergency surgical management.

SURGICAL OPTIONS:

The standard surgical intervention was laparotomy until the laparoscopic approach was introduced in 1973 by Shapiro and Adler; it has since gained wide acceptance(8).The surgical approach is also determined by the status of the patient's contralateral fallopian tube, the patient's plans for future fertility, and surgeon comfort or preference.

- **Salpingectomy** - If there is a lot of damage or bleeding, the affected tube would have to be removed. Salpingectomy is recommended in cases of extensive tubal damage and/or rupture, uncontrolled bleeding, prior tubal sterilization, or a large tubal EP (5 cm or more in diameter)(9).
- **Salpingotomy** - If the damage is minimal, and there is concern about the other Fallopian tube, the ectopic can sometimes be removed from the tube by making a small cut, leaving the tube intact but with a small scar.After a 1–2 cm linear incision is made with electrocautery, laser or scissors over the bulging ectopic gestation, the contents are removed using forceps or high pressure irrigation, also called hydrodissection(9).After salpingostomy, weekly α -hCG measurements are necessary to rule out persistent trophoblastic tissue, which can occur in up to 20 % of cases.

Hemodynamically stable patients – In hemodynamically stable pregnant patients with a nondiagnostic initial TVUS,evaluate for an ectopic pregnancy versus an IUP

- The discriminatory zone is the serum hCG level above which a gestational sac should almost always be visualized on ultrasound when an IUP is present. This level varies by laboratory and institution. However, a diagnosis of ectopic pregnancy cannot be made based on a single hCG result(10).
- Additional laboratory testing that may be performed includes a complete blood count to evaluate for anemia, RhD blood typing in case anti-D immune globulin is indicated, and renal and liver function tests.

Differential diagnosis –If an ectopic pregnancy has been ruled out, the patient should be evaluated for other causes of abdominal pain and/or vaginal bleeding in pregnancy (eg, spontaneous abortion, subchorionic hemorrhage, or nonobstetric causes, such as cervical polyp or other cervical pathology)(11).

Consequences- If left untreated, an ectopic pregnancy in the fallopian tube can progress to a tubal abortion or tubal rupture, or it may regress spontaneously.

Tubal rupture –Tubal rupture is usually associated with profound hemorrhage, which can be fatal if surgery is not performed expeditiously to remove the ectopic gestation. Ruptured ectopic pregnancy is the major cause of pregnancy-related maternal mortality in the first trimester(12). Most of these deaths occur prior to hospitalization or proximate to the patient's arrival in the emergency department.

Tubal abortion – Tubal abortion refers to expulsion of the products of conception through the fimbria. This can be followed by resorption of the tissue or by reimplantation of the trophoblasts in the abdominal cavity (ie, abdominal pregnancy) or on the ovary (ie, ovarian pregnancy). Tubal abortion may be accompanied by severe intraabdominal bleeding, necessitating surgical intervention, or by minimal bleeding, which would not require further treatment.

Spontaneous resolution – Spontaneous resolution of an ectopic pregnancy can occur, although it is difficult to predict which patients will experience uncomplicated spontaneous resolution. This is discussed in more detail separately.

- In rare instances, gestational products left in the fallopian tube can cause tubal obstruction(13).

CONCLUSION

An ectopic pregnancy can represent in any form within a wide clinical spectrum, ranging from an asymptomatic patient to one in shock and in any

age group and parity. The high index of suspicion for history and clinical features and associated biochemical markers, ultrasonography is sensitive still in non-availability of resources.

REFERENCES

1. Bouyer J, Coste J, Fernandez H, et al. Sites of ectopic pregnancy: a 10 year population-based study of 1800 cases. *Hum Reprod* 2002; 17:3224.
2. Alkatout I, Honemeyer U, Strauss A, et al. Clinical diagnosis and treatment of ectopic pregnancy. *ObstetGynecolSurv* 2013; 68:571.
3. Barnhart K. Ectopic pregnancy. *N Engl J Med*. 2009;361:379–87.
4. Rausch ME, Hampilos N, McNeill W, et al. Ectopic Pregnancy. In: *Handbook of Obstetric and Gynecologic Emergencies*, 5th ed, Benrubi GI (Ed), Lippincott Williams & Wilkins, 2018.
5. Seeber BE, Barnhart KT. Suspected ectopic pregnancy. *ObstetGynecol* 2006; 107:399.
6. Barnhart KT, Katz I, Hummel A, Gracia CR. Presumed diagnosis of ectopic pregnancy. *ObstetGynecol* 2002; 100:505.
7. Stovall TG, Kellerman AL, Ling FW, Buster JE. Emergency department diagnosis of ectopic pregnancy. *Ann Emerg Med* 1990; 19:1098.
8. Shapiro HI, Adler DH. Excision of an ectopic pregnancy through the laparoscope. *Am J Obstet Gynecol*. 1973;117:290–1.
9. Alkatout I, Honemeyer U, Strauss A, Tinelli A, Malvasi A, Jonat W, et al. Clinical diagnosis and treatment of ectopic pregnancy. *ObstetGynecolSurv*. 2013;68:571–81.
10. Condous G, Kirk E, Lu C, et al. Diagnostic accuracy of varying discriminatory zones for the prediction of ectopic pregnancy in women with a pregnancy of unknown location. *Ultrasound ObstetGynecol* 2005; 26:770.
11. <http://www.acr.org/~media/ACR/Documents/AppCriteria/Diagnostic/FirstTrimesterBleeding.pdf> (Accessed on March 20, 2013).
12. Anderson FW, Hogan JG, Ansbacher R. Sudden death: ectopic pregnancy mortality. *ObstetGynecol* 2004; 103:1218.
13. Tulandi T, Ferenczy A, Berger E. Tubal occlusion as a result of retained ectopic pregnancy: a case report. *Am J ObstetGynecol* 1988; 158:1116.



Acute Burn Compartmental syndrome

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Compartment syndrome usually occurs due to high voltage electrical burns, and even in deep circular burns with delayed escharotomy. With no care, ischemic tissue damage leads to irreversible damage and necrosis. It is mandatory to do emergency surgical decompression as early as possible with in six hours to get better results. Any such patients, irrespective of Doppler positive or negative finding, should undergo fasciotomy decompression if on pulse oximeter the reading is below 90 percent oxygen saturation. Because of bulky dressings in burn patients the diagnosis of compartment syndrome is always hard to make. The pressure transducer used in central arterial catheters can be used to measure pressure in muscular compartments. The normal pressure within a compartment is less than 10 mmHg. If the intracompartmental pressure reaches 30 mmHg or greater, acute compartment syndrome is present. Threshold intracompartmental pressure is 35 mmHg. If the intracompartmental pressure becomes higher than arterial pressure, a decrease in arterial inflow will also occur. The reduction of venous outflow and arterial inflow result in decreased oxygenation of tissues causing ischemia. If the deficit of oxygenation becomes high enough, irreversible necrosis may occur. When ever there is sign of unnecessary pain or ischaemic changes with pulse oximeter reading below 90 percent in circumferential burns of extremities or there is restriction of respiration and abdominal movement in circumferential deep burn then escharotomies should be done immediately and if it seems not effective then on the same time go for fasciotomy decompression of compartments to release the constrictive pressure on the muscle, nerve and vascular tissues. Escharotomies are usually

performed in patients with circumferential third degree burns of the extremities or anterior trunk. Fasciotomies are recommended for patients who sustained high voltage (or associated crush) injuries, with entrance or exit wounds in one or more extremities. Carpal tunnel release is practiced routinely for cases of electrical injury.

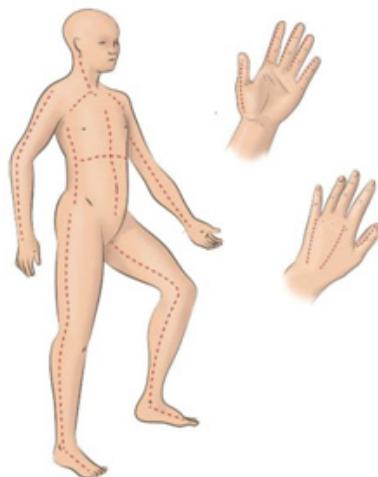


Fig 1 Escharotomies



Fig 2A High Electrical Voltage Burn



Fig 2B Fasciotomy



Fig 2C Later wound covered with skin graft

Acute compartment syndrome must be treated in hospital. It is a surgical emergency. There is no effective nonsurgical treatment. A compartment syndrome is a disease process where the pressure within a compartment, such as the fascial compartment surrounding the muscle tissue, exceeds the vascular perfusion pressure of that compartment resulting in decreased blood flow of the muscle contents of the compartment and hence tissue ischemia. In the case of a burn injury, this can be caused by the accumulation of tissue fluid or by the extrinsic compression by the burned tissue acting as a tourniquet and resulting in high pressures in a closed fascial space. This results in impaired perfusion of the tissues within the compartment compromising their viability if left untreated. Specifically, compartment syndromes are most commonly observed in patients with high-voltage electrical burns or patients with circumferential third-degree burns.

Surgery (fasciotomy) is the only treatment for acute compartment syndrome. The muscle compartment is cut open to allow muscle tissue to swell, decrease pressure and restore blood flow. Sometimes, the swelling is severe enough that the skin incision cannot be closed immediately. The incision is repaired later when swelling subsides. Sometimes a skin graft is used.

Compartment syndrome describes increased pressure within a muscle compartment of the arm

or leg. This pressure increase causes nerve damage due to decreased blood supply. Muscles are contained in compartments covered by thick fibrous bands of tissue or fascia. Because of injury, pressure can increase within the compartment to swelling (fluid accumulation) or bleeding. In non-contracting muscle, the compartment pressure is normally about 0-15 mmHg of pressure. If the pressure within the compartment increases (usually greater than about 30 -45mmHg; or are within 30 mm of the diastolic blood pressure) most individuals develop compartment syndrome. When these high compartment pressures are present, blood cannot circulate to the muscles and nerves to supply them with oxygen and nutrients. Symptoms such as pain and swelling will result. As the muscle cells lose their blood and oxygen supply, they begin to die. If the condition is not recognized and treated, the whole muscle can die, scar down, and contract. Similarly, nerve cells that are damaged may fail causing numbness and weakness in the structures beyond the injury site. If infection or necrosis develops, the individual may need the limb amputated to prevent death. Often there is elevation of creatine phosphokinase (CPK) which suggest muscle breakdown from ischemia, damage, or rhabdomyolysis. If rhabdomyolysis is being considered, renal function tests, urine myoglobin, and urinalysis should be done as it leads to renal failure and so taken care by fluid and diuresis therapy. Proper complete blood count and coagulation studies should be done to take care of any hematological complications.

Complications may include muscle loss, amputation, infection, nerve damage, and kidney failure. Acute compartment syndrome is a potentially devastating condition. Return of normal function and minimizing injury depends upon quick recognition of the situation and prompt surgical fasciotomy to resolve the increased pressure. The longer the delay to surgery, the more potential for permanent loss of muscle and nerve function.

The prognosis after treatment of compartment syndrome depends mainly on how quickly the

condition is diagnosed and treated. When fasciotomy is done within 6 hours, there is almost 100% recovery of limb function. After 6 hours, there may be residual nerve damage. Data show that when the fasciotomy is done within 12 hours, only two-thirds of patients have normal limb function. In very delayed cases, the limb may require an amputation.

Fasciotomy revision was associated with a fourfold increase in mortality. The most common revision procedures were extension of fascial incisions and opening new compartments. The most commonly unopened compartment was the anterior compartment of the lower leg. Patients who underwent delayed fasciotomies had twice the rate of major amputation and a threefold higher mortality. Therefore Fasciotomy decompression not escharotomy in acute third degree thermal burns or high voltage electrical burns should be the first choice when pulse oximeter reading is below 90 percent irrespective of Doppler findings if done.



Adult Intestinal Volvulus

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Introduction

Cases of volvulus were described in ancient Egypt as early as 1550 BC. It occurs most frequently in Africa, the Middle East, and India. Rates of volvulus in the United States are about 2–3 per 100,000 people per year. Sigmoid and cecal volvulus typically occurs between the ages of 30 and 70. The term volvulus is from the Latin “volvere”; which means “to roll”.

A volvulus is a surgical condition where the intestines twist around themselves. This condition can occur at any age. However, it is more frequent in children and infants.⁽¹⁾ One of the more well-known and most common forms of volvulus is sigmoid colon volvulus, a condition common in Asia and India due to their high-fiber diet, and also common in Peru and Bolivia because of high altitude. In this chapter we will focus on adult intestinal volvulus, etiology, symptomatology, clinical and radiological findings along with the different anatomical presentations, management protocols with the prognostic outcomes.

Definition and etiology

A volvulus refers to abnormal twisting of a part of the large or small intestine that may lead to a bowel obstruction and may require prompt diagnosis and treatment without which it can cause severe complications. Although the etiology of sigmoid volvulus, cecal volvulus, ileosigmoid knotting, or midgut volvulus is not well described, three common potentiating factors emerge. First, there is usually an associated long mesentery in the volvulated segment, resulting in a freely mobile bowel segment. Second, there is usually a dietary

practice of consuming a coarse, high-fiber vegetable diet in the presence of an empty bowel. Third In the absence of congenital malrotation, congenital bands, or postoperative adhesions may be the primary etiology for midgut volvulus.⁽²⁾ In adults, the sigmoid colon is the most commonly affected part of the gut followed by the cecum. In children, the small intestine is more commonly involved.⁽³⁾ In most cases, sigmoid volvulus is an acquired disorder. Cecal volvulus, on the other hand, may occur due to incomplete dorsal mesenteric fixation of the right colon or cecum or an elongated mesentery. Sigmoid volvulus is more common in individuals with neuropsychiatric disorders, multiple sclerosis, and Parkinson's disease. Neuroleptic drugs can also interfere with colonic motility and may trigger volvulus. Nursing home patients who are bedridden and have chronic constipation have a greater risk of developing sigmoid volvulus. A higher incidence of volvulus is also noticed among patients with associated myopathy like Duchene muscular dystrophy, visceral myopathy, etc. In developing countries, consumption of high fiber diet leads to overloading of the sigmoid colon, causing it to twist around the mesentery. Similarly, Chagas disease or megacolon can also predispose to sigmoid volvulus. However, in addition to the factors listed above, there is some evidence to suggest that parasitic infections and diabetic autonomous neuropathy play a role.^{(4) (5)} Rarely, appendicitis or surgery may lead to excessive adhesions leading to volvulus.

Men and women have an equal predilection for sigmoid volvulus in Europe and the United States, and most are over 60 years of age with a history of institutional care.⁽⁶⁾ The reasons for the male

preponderance of volvulus in parts of Africa, Asia, and Latin America are yet to be delineated. For sigmoid volvulus, some proposed explanations of this sex difference are based on the broader, more relaxed female pelvis in women, allowing for spontaneous reduction of the volvulus.⁽⁷⁾ Other studies have found that men have longer and narrower sigmoid mesenteric pedicles than females, resulting in a male predisposition to axial rotation of the sigmoid colon.⁽⁸⁾

Colonic volvulus constituted nearly 2% of all the cases of bowel obstructions admitted in the United States between 2002-2010.⁽⁹⁾ Sigmoid volvulus, accounting for 8% of all intestinal obstructions, occurs between the third and the seventh decades of life. It is more frequent in elderly males, African Americans, adults with chronic constipation, and associated neuropsychiatric disorders. On the other hand, cecal volvulus is more common in younger females. The age group of midgut volvulus is strikingly different from colonic volvulus. It is typically seen in babies with rotation anomalies of the intestine. Segmental volvulus of other portions of the gut can occur in people of any age, usually because of abnormal intestinal contents or adhesions.

Risk factors

Risk factors for volvulus include intestinal malrotation, Hirschsprung disease, an enlarged colon, pregnancy, and abdominal adhesions. A higher incidence of volvulus is also noticed among hospitalized patients with neuropsychiatric disorders such as Parkinson's disease, multiple sclerosis, etc. High fiber diet, chronic constipation with chronic use of laxatives and/or enema, and associated myopathy like Duchene muscular dystrophy, etc. are also associated with an increased risk of sigmoid volvulus. In adults, the sigmoid colon and cecum are the most commonly affected. On the contrary, splenic flexure is least prone to volvulus. In children, the small intestine and stomach are more commonly involved. Diagnosis is mainly clinical, however, characteristic radiological findings on plain radiograph, ultrasound, and upper GI series help in differentiating from other differentials.⁽¹⁰⁾ The

present article will cover volvulus in adults with specific differences from midgut volvulus in children. However, a detailed discussion of malrotation and midgut volvulus is beyond the scope of this article.

Pathophysiology

In western society, chronic constipation can lead to an overloaded sigmoid colon.

In developing nations, a high fiber diet leads to sigmoidal overload. When the bowel loop is overloaded with material, it becomes susceptible to torsion along the axis of an elongated mesentery. A large pelvic mass or a large gravid uterus can alter the position of the intra-abdominal organs, predisposing to the formation of volvulus. With recurrent attacks of torsion, the base of the mesentery can become chronically inflamed and eventually shortens.

This leads to recurrent volvulus. The twisting of a mobile loop of bowel can happen spontaneously and may be congenital or acquired. Acquired causes of volvulus include^(11,12,13)

1. Adhesions
2. Iatrogenic e.g. lower GI endoscopy
3. Bowel atony
4. Hirschsprung's disease
5. Pregnancy

Gastric Volvulus

Normally, there are ligaments such as the gastrocolic, gastrohepatic, gastrosplenic and gastrophrenic ligaments that keeps the stomach in place by attaching it to other abdominal organs and the diaphragm.^(14,15) However, the stomach can twist around its horizontal or vertical axis. Gastric outlet obstruction may occur as a result of abnormal rotation more than 180 degrees. Chronic rotation can cause bleeding by decreasing venous return and increasing capillary pressure.

Cecal Volvulus

The cecum is especially liable to being mobile congenitally^(16,17,18) The cecum becomes mobile when failure of the ascending colon mesentery to

fuse with the posterior parietal peritoneum occurs. Autopsy studies have shown that about 10-25% of the population have a mobile cecum and ascending colon sufficient to develop a volvulus. A congenital mobile cecum can also cause mobile cecum syndrome. There are three types of cecal volvulus, type I and II are the most common, type III accounts for the remaining 20% of cases.

Type I - organoaxial:

The cecum twists in a clockwise manner along its axis.

The cecum fills with air and remains in right lower quadrant.

Type II - organoaxial:

The cecum and a proximal part of the ileum twist in a counterclockwise direction.

The cecum becomes inverted and is relocated to the left lower quadrant.

Type III - mesentericoaxial:

The cecum folds upwards and back on itself rather than rotating along its axis.

Cecal volvulus can be either organoaxial (ceccolic or true cecal volvulus) or mesentericoaxial (cecal bascule). In the organoaxial variety, the ascending colon and distal ileum twist around each other in a clockwise manner. However, in the mesentericoaxial sub-type, the caecum is not completely fixed and is located anteriorly over the ascending colon at a right angle to the mesentery. Since there is no twisting of the vascular pedicle, vascular compromise is rarely associated with cecal volvulus. ⁽¹⁹⁾

Ileal and Sigmoid Volvulus

The mesentery anchors the ileum and sigmoid colon to the posterior abdominal wall ^(20,21)

An air filled loop of the sigmoid colon or the terminal ileum, sometimes, twists itself about the axis of the mesentery. The incidence of volvulus occurring increases with a redundant or longer than normal mesentery. If the degree of twisting is beyond 180 - 360 degrees, then the bowel loop

will become obstructed and ischemia will develop. Ileosigmoid knotting is a variant of sigmoid volvulus where the ileum wraps around the sigmoid in a clockwise direction. Sigmoid volvulus is typically caused by two mechanisms i.e. chronic constipation and a high-fiber diet. In both instances, the sigmoid colon becomes dilated and loaded with stools, making it susceptible to torsion. The direction of the volvulus is counterclockwise. With repeated attacks of torsion, there is a shortening of mesentery due to chronic inflammation. Subsequently, there is the development of adhesions which then entrap the sigmoid colon into a fixed twisted position

General principles of Management of Volvulus

Diagnosis of volvulus will typically require traditional x-rays and imaging, during which time doctors will look for a telltale "coffee bean" shape or "bent inner tube" shape in the bowel. A barium enema may also be performed in order to enhance the imaging of the potential condition. CT scans and ultrasound may also be considered for proper diagnostic imaging.



Fig – 01. Abdominal and chest radiography demonstrating enormously distended bowel and elevation of both hemidiaphragms

Pre operative optimization of the patient is very important and also the post operative management especially about the nutrition issues. Treatment for volvulus will typically involve attempts to unblock the obstruction and untwist the intestine. If this can be accomplished with surgery, that is always ideal. However, if the obstruction remains and the bowel remains twisted/rotated, surgical treatment may be required.

For most surgical treatments of volvulus, laparotomy is a common option. This involves an incision made in the abdominal region. Another option is laparoscopy, which uses small incisions and small instruments to perform abdominal procedures. In cases of gangrenous volvulus, a procto-sigmoidectomy (Hartmann's operation) will be performed.

Gastric volvulus

Gastric volvulus can manifest either as an acute abdominal emergency or as a chronic intermittent problem. The presenting symptoms depend on the degree of twisting and the rapidity of onset.

Acute gastric volvulus

The Borchardt triad (ie, pain, retching, and inability to pass a nasogastric tube) is diagnostic of acute volvulus and reportedly occurs in 70% of cases.⁽²²⁾ Carter et al described three additional findings that are suggestive of gastric volvulus, as follows⁽²³⁾

Intra-abdominal gastric volvulus most commonly manifests as the sudden onset of severe epigastric or left-upper-quadrant pain. Intrathoracic gastric volvulus manifests as sharp chest pain radiating to the left side of the neck, shoulder, arms, and back. This condition is often associated with cardiopulmonary compromise from gastric distention and may mimic an acute myocardial infarction.

Diagnosis

Biochemical investigations usually are not diagnostic for gastric volvulus; however, hyperamylasemia and elevated serum alkaline phosphatase (ALP) have been reported.⁽²⁴⁾ There has also been a report of hyperamylasemia in gastric volvulus leading to a missed diagnosis of pancreatitis.⁽²⁵⁾ Imaging studies, such as radiography (plain film and barium contrast) and computed tomography (CT), confirm the diagnosis. Findings on upper gastrointestinal (GI) endoscopy may be suggestive of gastric volvulus.

Treatment

In general, the treatment of an acute gastric volvulus remains emergency surgical repair.⁽²⁶⁾ In

patients who are not surgical candidates (secondary to comorbidities or an inability to tolerate anesthesia), endoscopic reduction may be attempted. Chronic gastric volvulus may be treated on a nonemergency basis, and surgical treatment is increasingly being performed via a laparoscopic approach. A review of patients with chronic gastric volvulus who were managed conservatively reported a high recurrence rate but very few serious complications.⁽²⁷⁾

Some authors have advocated consideration of emergency endoscopic reduction in the setting of acute gastric volvulus in patients who are at high risk for surgery.⁽²⁸⁾ This strategy may allow the patient to be adequately resuscitated and medically optimized before undergoing definitive surgical repair.

Small bowel volvulus

Small bowel volvulus is classified into two types, primary and secondary. Primary small bowel volvulus occurs in an otherwise normal abdominal cavity. The cause is poorly understood, and its rarity in North America and Europe remains unexplained.⁽²⁹⁾ The primary type is more frequently observed among the population of certain parts of Africa,⁽³⁰⁾ in Middle Eastern countries⁽³¹⁾, in Afghanistan,⁽³²⁾ and in Iran,⁽³³⁾ as well as in the Indian subcontinent,⁽³⁴⁾ where it may make up to 31% to 100% of cases. Several authors⁽³⁵⁾ conclude that in their series the high incidence of primary small bowel volvulus was caused by a combination of factors. There is speculation that in some populations a longer mesenteric length and a shortness of the mesenteric root would allow abnormal mobility of the entire small bowel or of a segment of it. But also an important cause could be abrupt changes in dietary habits with ingestion of a single large amount of bulky food after long intervals of fasting or on an empty bowel. This happens during special events such as religious periods like Ramadan or during summer months, when great numbers of marriages and feasts in underdeveloped rural areas are celebrated.

Management

Both retrospective analyses⁽³⁶⁾, as well as a

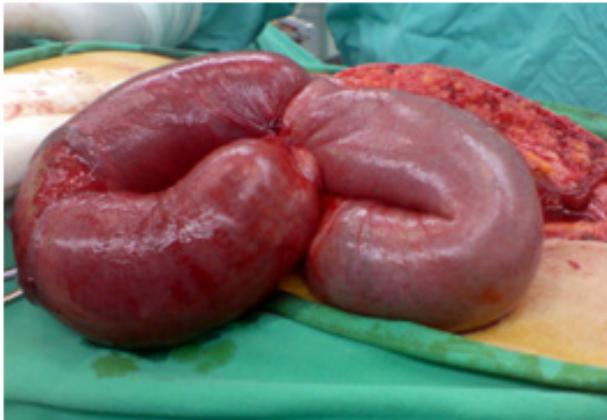


Fig - 02 Volvulus Small intestine

prospective study⁽³⁷⁾ for preoperative detection or exclusion of strangulated bowel consistently demonstrated, that neither one of the individual clinical parameters (fever, tachycardia, peritoneal signs, bowel sounds) or clinical judgment by experienced senior attending surgeons, nor blood chemical studies (hemoglobin concentration, hematocrit level, WBC count, or electrolytes) would appear to be sensitive or specific enough to discriminate between strangulated and simple bowel obstruction. They also do not help to clarify the need for operative intervention. ⁽³⁸⁾ Surgical options for small bowel volvulus include bowel resection and bowel conservative surgery.

CECAL VOLVULUS

Cecal volvulus is the second most frequent type of colonic volvulus, following sigmoid volvulus. ⁽³⁹⁾ There are two forms of cecal volvulus. The more common form involves an axial twist of the ileum, cecum, and proximal ascending colon around the mesentery.⁽⁴⁰⁾ The other involves folding of the cecum upward toward the hepatic flexure, termed cecal bascule, and accounts for 10% of cecal volvulus cases. Patients with cecal volvulus tend to be younger than patients with sigmoid volvulus and it is more common in women than in men.⁽⁴¹⁾ As many as 10% of patients with cecal volvulus are pregnant at presentation. To develop cecal volvulus, the cecum must be mobile with little fixation of the ascending colon to the retroperitoneum; however, this alone is likely inadequate. Other contributing risk factors include high-fiber intake, chronic constipation, acute medical illnesses, mental disorders, and previous

abdominal surgery. ⁽⁴²⁾

In patients with cecal volvulus, plain radiographs are diagnostic less than 20% of the time.⁽⁴³⁾ Classically, there is a "coffee bean" sign in the left upper quadrant. However, in less clear cases, there may be evidence of cecal and small bowel dilatation, and absence of colonic gas, which should prompt further imaging. Computed tomography imaging is highly diagnostic and usually demonstrates the "coffee bean" and mesenteric whirl. The location of the mesenteric whirl can help distinguish between sigmoid and cecal volvulus, as well as the type of cecal volvulus.⁽⁴⁴⁾

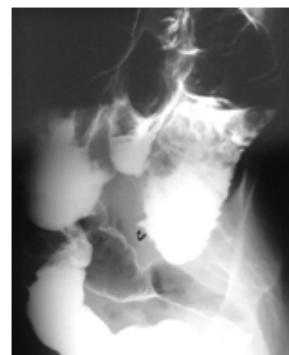


Fig – 03 .Right oblique image from a barium enema examination and the image shows a bird-beak appearance (arrow).

Unlike sigmoid volvulus, endoscopic detorsion is generally not recommended as initial management for cecal volvulus because of lower success rates and higher rates of ischemia in the volvulized segment.⁽⁴⁵⁾ Therefore, once cecal volvulus is diagnosed, surgical exploration is the appropriate course of action.

The first step in surgical management is assessing the volvulized segment. If it is gangrenous, resection is mandated. A primary anastomosis even in the setting of gangrene is acceptable; however, depending on the patient's nutritional status, comorbid conditions, or other factors negatively influencing healing, ileostomy creation may be preferred.⁽⁴⁶⁾ Several options have been described for treatment including detorsion alone, cecopexy, cecostomy placement, and resection usually in the form of a right hemicolectomy. Cecopexy involves suturing the right colon to the right paracolic gutter using sutures. Cecostomy

placement involves placing a tube through the abdominal wall and into the cecum, which affixes the colon and prevents volvulus. Rabinovici et al reviewed 561 cases in the literature with cecal volvulus and found that detorsion, cecopexy, and cecostomy had recurrence rates between 12 and 14% compared with resection which had no recurrences.⁽⁴⁷⁾ Additionally, the morbidity and mortality for cecostomy creation is 52 and 32%, respectively, compared with resection which is 29 and 22%, respectively. More recent studies have demonstrated lower morbidity and mortality for resection, and because of the lower recurrence rates, this is the preferred treatment of choice for cecal volvulus.⁽⁴⁸⁾

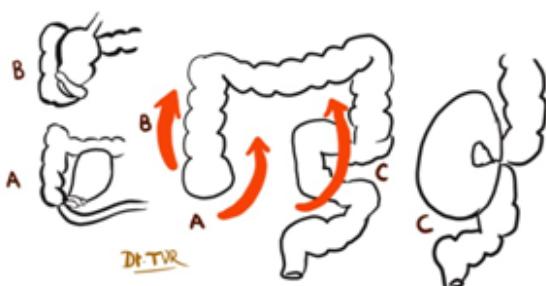


Fig - 4 .Cecal and sigmoid volvulus: (A) axial cecal volvulus; (B) cecal bascule; (C) sigmoid volvulus

Sigmoid volvulus

Depending on the duration of symptoms, the proximal colon can decompress into the distal small bowel, as long as the ileocaecal valve is incompetent. CT scan shows the characteristic "whirl" appearance of the twisted mesentery, as well as the distended loop of sigmoid colon with an air-fluid level. Free air on either the abdominal radiograph or the CT scan indicates a and requires emergency surgery.⁽⁴⁹⁾



Fig – 05.Abdominal radiograph , a large sigmoid loop in the shape of a "bent inner tube" or "omega loop"



Fig – 06.Laparotomy for sigmoid volvulus. A large dilated colon with long mesentery is identified

Surgical options for sigmoid volvulus include bowel resection and bowel conservative surgery. Bowel resection is recommended over conservative surgery (sigmoidopexy or mesenteric plication) as recurrence rates are higher with the later. If there is no faecal peritonitis, a primary resection can be done. While if there is bowel perforation, then a Hartmann procedure can be performed. Minimally invasive approach for sigmoid volvulus can be considered depending on the surgeon's preference and experience. Elderly patients may benefit from minimally invasive procedures. (Fig 7)⁽⁵⁰⁾

Prognosis

Any delay in the diagnosis of cecal or sigmoid volvulus can be associated with high morbidity and mortality. Mortality rates appear to be much higher for cecal volvulus compared to sigmoid volvulus. When volvulus is treated non-surgically rates of recurrence are very high approaching 40-60%. When surgery is done in unstable patients, mortality rates of 12-25% have been reported.

Complications

If untreated, volvulus can cause bowel strangulation, gangrene, perforation, and peritonitis. Complications of surgery include the following:Recurrence (if conservative surgery is performed)

Anastomotic leak,,Wound infection,Pelvic abscess,Sepsis,Fecal fistulaComplications of colostomy and/or ileostomy.

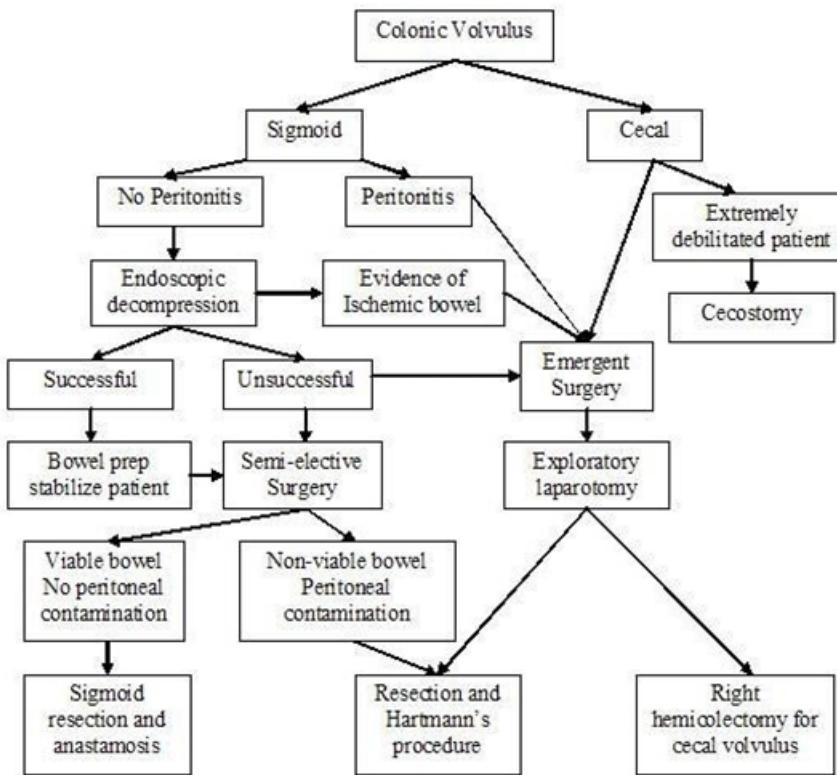


Fig- 07 Algorhythm of Large bowel volvulus management

References

1. Millar AJ, Rode H, Cywes S. Malrotation and volvulus in infancy and childhood. *Semin Pediatr Surg.* 2003 Nov;12(4):229-36
2. White RR, Jacobs DO. Volvulus of the stomach and small bowel In Yeo Charles et al., eds. *Shackelford's Surgery of the alimentary tract.* vol 1 6th ed. Philadelphia: Elsevier Saunders, 2007, pp 1035–1037. [Google Scholar]
3. DUTRA RM. [Upon a case of megacolon (syndrome of Hirshprung)]. *Hospital (Rio J).* 1948 Oct;34(4):545-8.
4. Papadimitriou G, Marinis A, Papakonstantino A. Primary midgut volvulus in adults: report of two cases and review of the literature. *J Gastrointest Surg* 2011;15:1889–92
5. Vaez-Zadeh K, Dutz W. Ileosigmoid knotting. *Ann Surg.* 1970;172:1027–1033.
6. Mangiante EC, Croce MA, Fabian TC, et al. Sigmoid volvulus. A four-decade experience. *Am Surg.* 1989;55:41–44.
7. Raveenthiran V *Observations on the pattern of vomiting and morbidity in acute sigmoid volvulus* *J Postgrad Medicine* 2004; 50:2729
8. Cirocchi R, Farinella E, Francesco L. The sigmoid volvulus: surgical timing and mortality for different clinical types. *World J Surg.* 2010;5(1):10–1186
9. Halabi WJ, Jafari MD, Kang CY, Nguyen VQ, Carmichael JC, Mills S, Pigazzi A, Stamos MJ. Colonic volvulus in the United States: trends, outcomes, and predictors of mortality. *Ann Surg.* 2014 Feb;259(2):293-301.
10. Yasui Y, Shironomae T, Kuwahara T. Target and Whirl Signs: Volvulus of Intussuscepted Colon in an Infant. *Clin Gastroenterol Hepatol.* 2020 May;18(5):A34.
11. John H, Gyr T, Giudici G, Martinoli S, Marx A (1996). "Cecal volvulus in pregnancy. Case report and review of literature". *Arch.*

- Gynecol. Obstet. 258 (3): 161–4. PMID 8781706.
12. Radin DR, Halls JM (1986). "Cecal volvulus: a complication of colonoscopy". Gastrointest Radiol. 11 (1): 110–1. doi:10.1007/BF02035046. PMID 3943670.
13. Sarioðlu A, Tanyel FC, Büyükpamukçu N, Hiçsonmez A (1997). "Colonic volvulus: a rare presentation of Hirschsprung's disease". J. Pediatr. Surg. 32 (1): 117–8. PMID 9111111.
14. Rashid F, Thangarajah T, Mulvey D, Larvin M, Iftikhar SY (2010). "A review article on gastric volvulus: a challenge to diagnosis and management". Int J Surg. 8 (1): 18–24. doi:10.1016/j.ijsu.2009.11.002
15. Shivanand G, Seema S, Srivastava DN, Pande GK, Sahni P, Prasad R, Ramachandra N (2003). "Gastric volvulus: acute and chronic presentation". Clin Imaging. 27 (4): 265–8.
16. Husain K, Fitzgerald P, Lau G (1994). "Cecal volvulus in the Cornelia de Lange syndrome". J. Pediatr. Surg. 29 (9): 1245–7. PMID 7807358.
17. DONHAUSER JL, ATWELL S (1949). "Volvulus of the cecum with a review of 100 cases in the literature and a report of six new cases". Arch Surg. 58 (2): 129–48. PMID 18111729.
18. Rogers RL, Harford FJ (1984). "Mobile cecum syndrome". Dis. Colon Rectum. 27 (6): 399–402. PMID 6734364.
19. Ramsingh J, Hodnett R, Coyle T, Al-Ani A. Bascule caecal volvulus: a rare cause of intestinal obstruction. J Surg Case Rep. 2014 Apr 11;2014(4).
20. Shepherd JJ (1969). "The epidemiology and clinical presentation of sigmoid volvulus". Br J Surg. 56 (5): 353–9. PMID 5781046.
21. VerSteeg KR, Whitehead WA (1980). "Ileosigmoid knot". Arch Surg. 115 (6): 761–3. PMID 7387365.
22. Miller DL, Pasquale MD, Seneca RP, Hodin E. Gastric volvulus in the pediatric population. Arch Surg. 1991 Sep. 126 (9):1146-9
23. Carter R, Brewer LA 3rd, Hinshaw DB. Acute gastric volvulus. A study of 25 cases. Am J Surg. 1980 Jul. 140 (1):99-106.
24. Godshall D, Mossallam U, Rosenbaum R. Gastric volvulus: case report and review of the literature. J Emerg Med. 1999 Sep-Oct. 17 (5):837-40.
25. Williams L, Lansdown MR, Larvin M, Ward DC. Gastric volvulus: a rare cause of hyperamylasaemia. Br J Clin Pract. 1990 Dec. 44 (12):708-9.
26. Light D, Links D, Griffin M. The threatened stomach: management of the acute gastric volvulus. Surg Endosc. 2016 May. 30 (5):1847-52.
27. Hsu YC, Perng CL, Chen CK, Tsai JJ, Lin HJ. Conservative management of chronic gastric volvulus: 44 cases over 5 years. World J Gastroenterol. 2010 Sep 7. 16 (33):4200-5.
28. Kulkarni K, Nagler J. Emergency endoscopic reduction of a gastric volvulus. Endoscopy. 2007 Feb. 39 Suppl 1:E173.
29. Ellis H. Special forms of intestinal obstructions. In Schwartz SI, Ellis H, ed. Maingot's Abdominal Operations. 9th Edition. Volume 1, Chapter 40. Norwalk, CT: Appleton & Lange, 1989, pp 905- 932.
30. De Souza U. Volvulus of the small bowel. Br Med J 1976; 1: 1055- 1056.
31. Wapnick S. Treatment of intestinal volvulus. Ann R Coll Surg Engl 1973; 53:57-61
32. Duke JH, Yar MS. Primary small bowel volvulus: cause and management. Arch Surg 1977; 112:685-688.
33. Saidi F. The high incidence of intestinal volvulus in Iran. Gut 1969; 10:838-841.
34. RL, Misra MK. Volvulus of the small intestine

- in northern India. Am J Surg 1970
35. Shatila AH, Chamberlain BE, Webb WR. Current status of diagnosis and management of strangulation obstruction of the small bowel. Am J Surg 1976; 132:299-303.
36. Shatila AH, Chamberlain BE, Webb WR. Current status of diagnosis and management of strangulation obstruction of the small bowel. Am J Surg 1976; 132:299-303
37. Holder WD. Intestinal obstruction. Gastroenterol Clin North Am 1988; 17(2):317-340.
38. Deutsch AA, Eviatar E, Gutman H, Reiss R. Small bowel obstruction: a review of 264 cases and suggestions for management. Postgrad Med J 1989; 65:463-467.
39. Jones I T, Fazio V W. Colonic volvulus. Etiology and management. Dig Dis. 1989;7(4):203–209.
40. Ballantyne G H, Brandner M D, Beart R W Jr, Ilstrup D M. Volvulus of the colon. Incidence and mortality. Ann Surg. 1985;202(1):83–267.
41. Swenson B R, Kwaan M R, Burkart N E. et al. Colonic volvulus: presentation and management in metropolitan Minnesota, United States. Dis Colon Rectum. 2012;55(4):444–449
42. Ballantyne G H, Brandner M D, Beart R W Jr, Ilstrup D M. Volvulus of the colon. Incidence and mortality. Ann Surg. 1985;202(1):83–92.
43. Rabinovici R, Simansky D A, Kaplan O, Mavor E, Manny J. Cecal volvulus. Dis Colon Rectum. 1990;33(9):765–769
44. Delabrousse E, Sarlièvre P, Sailley N, Aubry S, Kastler B A. Cecal volvulus: CT findings and correlation with pathophysiology. Emerg Radiol. 2007;14(6):411–415.
45. Friedman J D, Odland M D, Bubrick M P. Experience with colonic volvulus. Dis Colon Rectum. 1989;32(5):409–416.
46. Tuech J J, Pessaux P, Regenet N, Derouet N, Bergamaschi R, Arnaud J P. Results of resection for volvulus of the right colon. Tech Coloproctol. 2002;6(2):97–99.
47. Rabinovici R, Simansky D A, Kaplan O, Mavor E, Manny J. Cecal volvulus. Dis Colon Rectum. 1990;33(9):765–769.
48. Swenson B R, Kwaan M R, Burkart N E. et al. Colonic volvulus: presentation and management in metropolitan Minnesota, United States. Dis Colon Rectum. 2012;55(4):444–449.
49. Mba EL, Obiano SK, Mshelia NM. Compound volvulus: a case report and literature review. J Surg Case Rep. 2018 Nov;2018(11):rjy311
50. Carmo L, Amaral M, Trindade E, Henriques-Coelho T, Pinho-Sousa J. Sigmoid Volvulus in Children: Diagnosis and Therapeutic Challenge. GE Port J Gastroenterol. 2018 Sep;25(5):264-267.



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Started in 2015



With the Aim to provide financial assistance to its member and his/her spouse, children and parents in the event of hospitalization for treatment, diagnosis and management of diseases

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This provision makes IMA National Health Scheme unique from other schemes.

Fee Structure at the time of joining (First Year) Payment Chart of NHS according to age group

Payment chart at the Time of Joining

Age	AF	AMS	AFAC	Total
Less than 25yrs	1000	500	2500	4000
Above25 below35yrs	1000	500	3000	4500
35 to below 45yrs	1250	500	3000	4750
45yrs to 55yrs	1750	500	3000	5250
55 to below 60yrs	5000	500	5000	10500
60 to below 65yrs	7000	500	7000	14500
65yrs to below70yrs	8000	500	8000	16,500
70 to 80 yrs	10,000	500	10,000	20,500

Admission Fee is onetime Payment AMS & AFAC have to be paid every Year

Payment chart for renewal from 2nd Year onwards

Age	AF	AMS	AFAC	Total
Less than 25yrs	nil	500	2500	3000
Above25 below 35yrs	nil	500	3000	3500
35 to below 45yrs	nil	500	3000	3500
45yrs to below 55yrs	nil	500	3000	3500
55 to below 60yrs	nil	500	5000	5500
60 to below 65yrs	nil	500	7000	7500
65yrs to below 70yrs	nil	500	8000	8,500
70 to 80 yrs	nil	500	10,000	10,500

AMS &AFAC have to be paid every Year

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1. In IMA NHS there is no escalation of annual premium amount even if the age progresses to the next slab for that particular insured amount. For example a person joining the scheme at the age of 25 years will be paying the same amount of Rs.3500/- till 55yrs as renewal fee per annum for an upper limit of 2 lakhs benefit.
2. Scrutinizing committee will examine the genuineness of the claim. 75% of the Total bill will be reimbursed to a maximum of 2 lakhs. Will be increased to 2.5-3lakhs
3. Allowed to join other insurance schemes and State Health Schemes. Total 3+2= 5 Lakhs benefits State HS+ National H.S
4. All pre-existing diseases are covered including Cancer and Organ Transplant
5. No medical screening test required for joining IMA National Health Scheme.
6. Member can join till the age of 80 years. It is the only Scheme which allows the IMA member and his family to join above the age of 65yrs .For all other schemes age limit is restricted to 65 yrs as upper limit
7. Immediate relatives of life members of IMA can also join.
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9. More than insurance companies, IMA NHS exist for the medical Fraternity and its family to provide financial help at the time of hospitalization.
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Annual Subscription: Every year member should pay Rs.500 as annual subscription for 25 years. After that member will become Honorary Member and become eligible for all rights.

One Time Payment : By paying Rs.4,00,000 as Non refundable deposit the member will become life Member and become eligible for all rights and no need to pay other payments.

Member Benefits or Rights: • On the event of demise of a member : Fraternity Contribution RS.300/- (200/- to Fraternity Contribution & Rs.100/- to corpus fund) on the event of demise of any member (To a maximum of 50 deaths per year over which the Fraternity Contribution will be taken from the corpus fund) • Disabled or incapacitated Practitioner member will get a benefit from the Corpus Fund which will be judicially decided by the Management Committee.

LOCK IN PERIOD : • If the Age joining the scheme is below 50 years, the lock in period is 2 years. • If the Age of joining the scheme is above 50 years, the lock period is 3 years.

FOR MORE DETAILS: Log on to www.ima-india.org IMA National Family Welfare Scheme.



IMA National Social Security Scheme (IMANSSS)

BENEFITS : On event of death (by any cause), rest of the members contribute a token share of Rs. 100-00, from which Rs. 70-00 is passed on to the nominee.

After 25 years of continuous membership, the member has not to contribute the same and the Fraternity Contribution (F.C.) will be paid to the nominee by the scheme.

BENEFIT FOR MEMBERS ENROLLED AFTER 19/07/2002 : For members enrolled after 19/07/2002, benefit of Fraternity Contribution of the scheme liable after completion of one year of membership of I.M.A. N.S.S.S. However nominee of member be entitled for such benefits if death of member occurs in accident event within one year of joining the scheme.

ELIGIBILITY : Any life member of I.M.A., up to age of 60 years residing in India is eligible to become a member of this scheme but members above the age of 40 years and below the age of 60 years, must be life member of IMA atleast for 3 years on the day of joining the scheme.

At present we have 19,070 members enrolled and paid Rs. 11,50,450.00 to the last deceased member's nominee.

Contact for Membership

Kindly contact on below email / call to get the membership information

Call us +91-79-26585430

E-mail: imansss1@gmail.com | Mail: contact@imansss.org | Website : www.imansss.org