Neonatal Convulsions

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Introduction

- Seizure: clinical expression of abnormal, excessive, synchronous, discharge of neurons primarily in the cerebral cortex.
- Neonatal seizure occurs in 1st 4 weeks in term baby and within 44 weeks post conception in the preterm.
- Neonatal seizures are relatively common with variable clinical presentation. Incidence in US 80-120/100000 neonates/yr. (this is higher than in any other age group worldwide =3.5/1000). Occur most frequently in the 1st 10 days of life.
- Neonatal seizures are risk factors that predicts increased long term morbidity/mortality.

Seizures pathogenesis

- Increased cerebral excitatory neurotransmitter activity (amino acids- Glu, Asp)
- Reduced inhibitory neurotransmitter activity (GABA)
- Disruption of ATP-dependent membrane potential with increased flow of Na^+ and extrusion of K^+ from the neuron. E.g. hypoxic ischemic encephalopathy (HIE)

Classification of neonatal seizures

- Most are focal but generalised fits described in a few instances. Focal nature is due to incomplete maturity of CNS

1. **Subtle fits**: full term> preterms e.g. chewing, peddling, ocular movements. Usually not demonstrable electrographically (EEG)
2. **Clonic fits**: often involves 1 extremity or one side of the body. Slow rhythm (1-3/sec). Usual seen on EEG.
3. **Myoclonic**: focal or multi-focal. Focal &multifocal usually not EEG associated. Generalised myoclonic fits do occur and are clinical equivalent of infantile spasms.

Classification cont

4. **Tonic fits**: one extremity usually but may be generalised tonic clonic (GTC)

   Focal fits associated with EEG changes. GTC usually not associated with EEG changes and manifest with tonic extension of all limbs and may involve axial muscles in opisthotonic fashion.

5. **Jitteriness**: must be differentited from seizures. Usually no ocular movements and are stimulus sensitive (i.e easily stopped with passive movement of the limbs). No associated autonomic changes (altered HR, RR, BP, flushing, salivation, pupillary dilatation)

Causes

1. **HIE**: important cause of fits. Presents usually within 72hrs of life. Fits subtle/clonic/ generalised
2. **Intracranial haemorrhage:** generally preterm>term. May be difficult to differentiate from HIE clinically.

   - **Sub-arachnoid bleed**: term>preterm occurs frequently and is usually not clinically significant. Typically infants remarkably well.

   - **Germinal matrix IVH** : preterm>term especially < 34/40. subtle fits common in this type

   - **Subdural bleeds:** term>preterms usually severe and associated with cerebral contusion.

Causes cont

3. **Metabolic disturbances**: hypoglycemia, hypoCa+, hypoMg+, less frequently inborn errors of metabolism which arise more than 72 hrs of life and typically after onset of feeding.
4. **Intracranial infections**: meningitis, encephalitis, toxoplasmosis,CMV. Common bacteria E. coli, GBS, listeria)
5. **Major malformation syndromes**: lissencephaly, polymicrogyria, linear sebaceous nevus syndrome.

Causes cont

6. **Benign neonatal seizures**:

   - benign familial neonatal seizures: occur in 48-72hrs of life. Disappear by 2/12-6/12. usual +ve family Hx. Development normal.

   - benign idiopathic (fifth day fits): occur btn 4-6 days of life. Multifocal. CSF normal. Usually lasts 24 hrs.

   -benign sleep myoclonus: rhythmic movements during NREM (non rapid eye movement)sleep mimic seizures. EEG (no fits)

7. **Others**:

   Anoxia, benign epilepsy syndromes, mitochondrial cytopathies, myoclonic epilepsy, myoclonus, organic acidosis, pyridoxine dependent epilepsy.

History

- Family Hx: +ve family Hx of fits points to genetic syndromes. Many syndromes are considered benign & frequently disappear in the post-neonatal period.
- Ante-natal Hx: TORCHES, fetal distress, PET, maternal infection.
- Birth Hx: type of delivery,antecedent events during labor, Apgar, resuscitation.
- Post-natal Hx: tremors(hypoCa+/drug withdrawal), Temp, BP instability,

Physical exam

- Usually lethargy between fits. Ill looking
- CNS exam: abnormal findings that may correlate with focal or neurological syndromes. In some cases CNS exam may be normal in between fits.

Work-up(Lab)

- RBS, UEs including Ca,
- CSF studies: pleocytosis, xanthochromia, lactic acid, pyruvate levels, PCR for HSV, glucose tests, M/C/S
- TORCH studies, urine organic acids, serum amino acids, serum long chain FA assay.
- Renal and LFTs: to r/o post hypoxic renal dysfxn. Hypoxic multiple organ damage may be suggested by raised liver transaminases.
- Pulse oximetry, ABGs, FBC, blood cultures

Work-up(imaging)

- Cranial ultrasound: may detect intracranial bleeds e.g. IVH. It is limited as may not detect cortical lesions/SAH.
- Cranial CT: more sensitive than U/S for parenchyma abn e.g. congenital malformations but may miss subtle malformations.
- MRI: most sensitive imaging modality

Work-up (other tests)

- EEG: may help differentiate seizures from non-epileptic events. Video EEG may monitor esp infrequent fits.
- ECG: cardiac hypomotility associated resulting from diffuse hypoxia.
- Karyotyping

Treatment

- Etiologic therapy is critical and may prevent further brain damage; and some fits may be poorly controlled unless cause is treated.
- Ensure adequate ventilation and perfusion,
- Treat aggressively. Optimal treatment controversial.
- Determine aetiology by rigorous work-up
- Correct electrolyte imbalances preferrably via CVP line. Caution when correcting Calcium to avoid adverse effects. Correct glucose.
- Treat infection initially empirically until lab results gotten then change to the appropriate anti-microbial.
- In case of inborn errors of metabolism institute IVF and stop feeding.

Treatment

- Phenobarbitone: initial AED of choice. Give minimal amount required and wait for anticonvulsant effect to develop.
- Phenytoin: add to phenobarb in persistent fits.
- Benzodiazepines: lorazepam & midazolam may be used in persistent seizures.
- Vitamins: pyridoxine usually tried in patients not responding to the above. Pts with pyridoxine dependent fits respond immediately

Treatment

- Monitor head circumference esp in ICH to detect hydrocephalus.
- Drug levels may be monitored. Duration of AED depends on cause,severity and EEG findings. Usually stop drugs 2/52 after last fit but in some cases treatment may be upto 6/12.
- Neurological evaluation & follow-up.
- Development evaluation for detection of physical and cognitive deficits with prompt institution of physiotherapy.

Complications

- Cerebral palsy, cerebral atrophy, spasticity, hydrocephalous, epilepsy, feeding difficulties
- Prognosis: depends on aetiology. Normal EEG denotes good prognosis, severe EEG changes denote poorer prognosis (spikes on EEG associated with 30% risk of future epilepsy)
- Isolated SAH= 90% excellent prognosis with no further deficits. half of all cases have good prognosis.