COMPLICATIONS OF PREMATURITY

Early: RDS, Jaundice, PDA, IVH, Early anemia of prematurity. These occur while the patient in hospital.

Late: ROP, BPD (CLD), Late anemia of prematurity, Rickets, CNS damage. These occur while patient in hospital or after discharge.

EARLY COMPLICATIONS OF PREMATURITY:

1. Respiratory distress syndrome (RDS) or (HMD)

2. Patent Ductus Arteriosus (PDA)

Definition: It is a failure of the ductus arteriosus to close in the first few days of life or reopening after functional closure. Typically it results in Lt – Rt shunt of blood once pulmonary vascular resistance (PVR) has decreased. If PVR remain high – blood may be shunted Rt—Lt resulting in hypoxemia (PPHN).

Incidence is up to 60% in preterm <1500 grams, higher in < 1000 grams up to 70%. female: male ratio is 2:1. Obligatory PDA is found in 10% of infants with other CHD.

Risk factors: PDA most often related to hypoxia and immaturity. Term infants with PDA usually have structural defects in the wall of ductal vessels.

Clinical features and diagnosis: Examination show 1-4/6 continuous machinery murmur, loudest in the left upper sternal border (LUSB), or left infraclavicular area. They may have apical diastolic rumbling murmur because of increased flow across mitral valve. Bounding peripheral pulses with wide pulse pressure >60 mmHg differences between systolic and diastolic BP. Hyperactive precordium and palmar pulses may also be present in large shunt.
ECG is normal or LVH in small-moderate PDA. Bilateral VH in larger PDA. Chest x ray may have cardiomegaly, increased pulmonary vascular markings. Echocardiography will confirm the diagnosis of PDA.

Management: fluid restriction and diuretics, if no response in 24-48 hours, give indomethacin iv infusion in a dose of 0.2 mg / kg iv every 12 hours for 3 doses. Oral ipobrufen is an alternative. 80% will close by these measures in preterm neonates. If failed-- surgical closure by ligation of the duct.

3. Intracranial hemorrhages (ICH): periventricular Hg, intraventricular Hg, subarachnoid Hg, subdural Hg, and intracerebral Hg. The most common Hg in Preterm NB is Intraventricular Hg (IVH).

3. Intraventricular Hg (IVH):

IVH usually arising in the germinal matrix and periventricular region of the brain.

Patho-physiology: in the periventricular germinal matrix, poor structural support, failure of auto-regulation of BP in these sick preterm NB, and venous stasis due to many reasons.

Incidence: 30-40% of preterm NB <1500 grams, 50-60% of those <1000 grams. IVH is highest during first 72 hours of life.

Clinical features: IVH can present with seizure, apnea, bradycardia, lethargy, coma, hypotension, metabolic acidosis, anemia not corrected by blood transfusion, increasing OFC, bulging fontanel, cutaneous mottling. Those with grade 1, 11 may be asymptomatic, those with 111, V1 grades may have catastrophic event that eventually lead to shock, coma and even death.

Diagnosis: clinical course with follow up of OFC and Hb, ultrasound of brain through anterior fontanel in first and second week, and may need CT scan.

Ultrasound of brain is used in diagnosis and classification of IVH. Screening is indicated in premature <32 weeks in the first week and should be repeated in the
second week. Grading is based on maximal Hg seen in the second week of age. Grade 1 – Hg in germinal matrix only, Grade 11 – IVH without ventricular dilatation, Grade 111 – IVH with ventricular dilatation, Grade 1V – IVH with periventricular hemorrhagic infarcts.

Prevention: by maintain acid base balance; avoid fluctuation in BP, and pharmacologic prophylaxis by indomethacin for prevention of severe Hg.

Outcome: death or surviving. Surviving with grade 111 may have 30-40% incidence of motor and cognitive impairment, while those with grade V1 may have 60-80% incidence of motor and cognitive impairment.

Treatment: supportive by ventilation for apnea, packed blood transfusion for anemia and shock, and plasma, vitamin k and platelets to correct coagulation disorder. Hydrocephalus when developed may require serial daily lumbar puncture, external ventriculostomy or permanent ventriculo-peritoneal shunt.

4. Early anemia of prematurity: usually occur in the first 2 weeks of life, could be caused by blood loss or haemolysis or decreased RBCs production. It is treated by blood transfusion and vitamin E.

5. Jaundice of Prematurity: due to poor enteral intake, delayed stooling and increased enterohepatic circulation.

Late complications of prematurity:

1. Retinopathy of prematurity (ROP), (Retrolental fibroplasia):

ROP is the interruption of the normal progression of retinal vascularization. it is responsible for many cases of blindness.

Etiology: ROP is due to exposure of the immature retina to high 02 concentration, which will result in vasoconstriction and obliteration of the retinal capillary network, which is followed by vaso- proliferation. The risk is greatest in the most immature infants.

2. Broncho-pulmonary dysplasia (BPD):
**Classic BPD** is a neonatal form of chronic lung disease that follows a primary course of respiratory failure (RDS, Meconium aspiration syndrome) in the first days of life. BPD is also defined as persistent 02 dependency up to 28 days of life.

Incidence is influenced by many factors, the most important of which is lung maturity. It increase with decreasing body weight, affect 30% of <1000 grams.

The major factors contributing to BPD are inflammation, mechanical ventilation (volutrauma/barotraumas, which is now decreased by early use of nasal CPAP), and oxygen exposure (aim now for pao2 of 85-93%).

3. **Late anemia of prematurity:**

It occurs in preterm infants after 6 weeks. It is purely iron deficiency anemia, so put patients on prophylactic iron 3mg/kg/day of elemental iron that started from 6th week of life or once full enteral feeding is achieved. Check Hb, if > 10 g/dl --- no treatment. If < 10 g/dl --- do reticulocyte count --- if high, give iron. If low retic. --- give blood.

4. **Rickets:** preterm babies got poor vitamin D stores and rapid growth, so it is better to put them on vit. D supplements from 6th week of life of 400 iu/ day.

5. **CNS damage:** Preterm baby is more prone to develop the following:

1. Major developmental delay, MR, CP, epilepsy, blindness, deafness.

2. Other less severe sequels: cranial nerve palsies, cerebral palsies, progressive hydrocephalus, learning problem, behavioral problem, mental retardation and epilepsy.

**Small for gestational age (SGA)**

It is defined as 2 SD below the mean weight for GA or below the 10th percentile.

**Etiology:** SGA is commonly seen in infants of mothers with hypertension, preeclampsia, or who smoke.

Other maternal diseases like chronic disease, malnutrition, conditions affect the blood flow and oxygenation.
Placental factors like infarction, previa, abruption, anatomic malformations.

Fetal factors like congenital infections (TORCH), Chromosomal abnormalities, dysmorphic syndromes, other congenital anomalies.

**Incidence:** 3-10% of all pregnancies are associated with IUGR, and 20% of still-born infants are growth retarded. 1/3 of LBWN are growth retarded and not premature. SGA neonates have higher morbidity and 10 times higher mortality than AGA.

**Clinical presentation:** The history will raise the index of suspicion regarding suboptimal growth. The infant will have reduced birth weight for GA. Using growth charts and the Ballard scoring can help assess GA and intrauterine and postnatal growth. IUGR infants have characteristic physical appearance. They are thin with loose peeling skin, scaphoid abdomen and disproportionately large head.

**Complications of SGA:**

1. Hypoxia due perinatal asphyxia, persistent pulmonary hypertension, RDS, meconium aspiration, PDA.

2. Hypothermia

3. Metabolic like hypoglycemia, hyperglycemia, hypocalcaemia, liver disease (cholestasis, non-fatty liver disease).

4. Hematologic like hyper viscosity and polycythemia.

5. Altered immunity.

6. Others like NEC.

**Management:** Antenatal diagnosis is the key to proper management of IUGR.

1. History of risk factors.

2. Delivery and skilled resuscitation should be available because of perinatal depression.

4. Hypoglycemia by close monitoring of blood sugar and treat it by iv dextrose and early feeding.

5. Hematologic disorders by central pcv and partial exchange transfusion if needed for polycythemia.

6. Congenital infection.

7. Genetic anomalies by screening.

**Large for gestational age neonate (LGA):**

NB with BWT OF 2 SD above mean or above 90th percentile

Etiology: 1. constitutionally large infants (large parents)

2. Infant of diabetic mothers class A, B, C

3. Some post term infants

4. Beckwith – Weidmann and other syndromes

Management: look for possible etiology

- Look for birth trauma evidence like brachial plexus injuries
- Look for perinatal depression
- Early feeding, blood sugar monitoring, some may have hypoglycemia
- Consider polycythemia

**Post term infants:**

Infant that is delivered after gestation of > 42 completed weeks. They accounts for 3-12% of pregnancies.

Etiology: unknown in the majority.
Known Associations: anencephaly due to absence of pituitary – adrenal axis, Triosomies, seckles` syndrome (bird headed dwarfism), erroneous estimation of GA.