

## TEACHING FILE

### GRAND ROUNDS

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#### DENGUE FLUIDS

**Case Report:** A 2 years old boy presented with fever for 7 days, vomiting for 2 days, pain in abdomen for 2 days and oliguria for a day. There was no jaundice, loose motions or altered sensorium. On examination, the child has heart rate of 150/min, respiratory rate of 46/min with minimal distress, some dehydration, BP of 80/60 mm of Hg and an erythematous maculopapular rash. He had a right sided minimal pleural effusion with tender hepatomegaly. Other systems were normal. He was suspected to have dengue hemorrhagic fever (DHF) and was given 2 normal saline boluses of 20cc/kg. Investigations showed leucopenia, hemoconcentration with thrombocytopenia, normal creatinine with elevated BUN, hyperkalemia, elevated liver transaminases, prolonged prothrombin time and partial thromboplastin time with Chest X-Ray showing right sided pleural effusion. Dengue IgM was positive. He was continued on IV fluids of 6cc/kg/hour to which his hypotension responded and hematocrit decreased. However, he had passed urine of only 0.5cc/kg/hour in next 12 hours. At the end of 12 hours, he was noticed to have respiratory distress (respiratory rate = 46/min) with lower intercostal retractions, deep and not to rapid breathing with puffiness of eyes.

**What is the problem? How should this child be managed now?**

#### Expert's opinion :-

This child has DHF. With fluid resuscitation, his intravascular compartment seems to have restored but the oliguria continues. Thus the child seems to have an additional renal component of the cause of oliguria. Even though serum creatinine is normal, the child does have hyperkalemia. Also the respiration seems to be like an acidotic breathing making one suspect metabolic acidosis. Metabolic acidosis without dehydration also suggests a renal involvement. This child also has puffiness of eyes which may be due to third spacing. The distress of breathing especially with activity of lower intercostals suggests congestion of the bases of lung. Congestion can be the lung parenchyma i.e., pulmonary edema or in the pleural spaces due to third space losses. Pulmonary edema in a DHF may occur either due to volume overload {which should lead to increased urine output} or due to myocardial dysfunction. Thus, there seems to be a heart involvement in this child too. Thus, in this child, there seems to be a multisystem organ problem. The pulmonary edema should be treated with a diuretic which may also help the kidneys to open up. Prior to that the extra fluids should be stopped and child would require inotropic support to maintain the intravascular volume. This child was given Dopamine, extra fluids were stopped and when blood pressure was around 75th centile, a diuretic was given following which the child passed 400 ml of urine in next 6 hours. His blood gases did show metabolic acidosis Echocardiography showed left ventricular dysfunction. With this treatment, child had a marked improvement.

Hence in a patient with DHF, optimum fluid management is very essential to prevent complications.

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#### INDIRECT HYPERBILIRUBINEMIA

**Case Report:** A 2 month old boy presented with jaundice without high coloured urine since 1 month of age. He was a full term, caesarean section delivery in view of fetal distress with birth weight of 3.5 kg and cried immediately after birth. He was on exclusive breast feeds and was immunized till date. On examination, he was well nourished (weight = 4.4 kg), had jaundice without hepatosplenomegaly. Other systems were normal. Investigations showed:

- Hemoglobin = 10.9 gm/dl
- WBC count = 6,500/cumm (polymorphs = 25%, lymphocytes = 74%, monocytes = 1%)
- Platelet count = 3,39,000/cumm
- Reticulocyte count = 1.2%
- Bilirubin = 9.8 mg/dl (indirect bilirubin = 8.8 mg/dl)
- Liver transaminases = Normal
- G6PD = Normal
- Thyroid function tests = Normal
- Peripheral smear = RBC morphology is normal.

Child's breast milk was discontinued for 2 days but jaundice did not resolve. There was no Rh or ABO incompatibility and no improvement with phototherapy. He was then treated with phenobarbitone for 5 days following which his bilirubin levels decreased.

**What is the diagnosis?**

#### Expert's opinion :-

This healthy neonate has presented with indirect hyperbilirubinemia. The common causes of neonatal indirect hyperbilirubinemia such as Rh or ABO incompatibility, Polycythemia, hereditary spherocytosis, G6PD deficiency, breast milk jaundice and even hypothyroidism have been ruled out. Also it is unlikely to be exaggerated physiological jaundice as it would not persist beyond 1st month of life. (Physiological jaundice usually resolves by Day 14 of life). Thus other possibilities such as conjugation defect in bilirubin should be kept in mind such as Crigler Najjar syndrome. There are 2 types of Crigler Najjar syndrome -Type 1 in which case the jaundice increases inspite of phototherapy and patients are at risk for kernicterus. This is due to complete absence of enzyme UDP-glucosyl transferase. Type 2 Crigler Najjar syndrome is characterized by partial activity of UDPG transferase enzyme and conjugation activity is potentiated by a short course of phenobarbitone which leads to decrease in bilirubin. In this child also there was decrease in bilirubin following phenobarbitone suggesting that Crigler Najjar Type 2 may be a possibility. The confirmation can be done by estimation and enzyme on liver biopsy.

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### **PNEUMONIA IN A 4 MONTH OLD**

**Case Report:** A 4-month-old boy presented with fever, cough, cold since 5 days and breathlessness since 1 day with decreased appetite. He was a full term normal delivery without antenatal or postnatal complications on bottle feeds with cow's milk. He was immunized till date with OPV, DPT and BCG. On examination, he had respiratory rate of 58, min with intercostals, substernal retractions and was febrile. There were bronchial breath sounds on right infrascapular region. Other systems were normal. Investigations showed:

- Hemoglobin = 10.2 gm/dl
- WBC count = 30,800/cumm (58% polymorphs, 42% lymphocytes)
- Platelet count = 6,75,000/cumm
- Arterial blood gas = pH = 7.39, PCO<sub>2</sub> = 35 mm, PO<sub>2</sub> = 61 mm of Hg, bicarbonate = 21 mEq/L and oxygen saturation of 91 percent.
- ESR = 115 mm at end of 1 hour
- Chest X-Ray = Bilateral lower zone pneumonia with minimal pleural effusion and inter fissural effusion on right side.

#### **Which is the organism causing the pneumonia?**

##### **Expert's opinion :-**

The common organisms causing pneumonia in this age group are:

- Streptococcus pneumoniae
- Hemophilus influenza b (Hib)
- Staphylococcus aureus

- Atypical organisms such as Chlamydia
- Viral pneumonia.

This child has high counts and has bronchopneumonia with effusion. Hence viral pneumonia seems unlikely. The atypical organisms usually cause cough and breathlessness and have crepitations. Also in infants, chlamydia pneumonia is usually seen following neonatal conjunctivitis due to Chlamydia infection. Mycoplasma and ureaplasma are usually seen in school going children. Thus, atypical pneumonias cause more signs of interstitial lung disease rather than lobar pneumonia and also seem unlikely in this child. Among the bacterial infections, Hib is the commonest organisms in this age group and can lead to effusions. Also this child has not received Hib vaccine. Streptococcus pneumoniae leads commonly to lobar pneumonia whereas staphylococcus aureus leads to a rapidly progressive necrotizing pneumonia with deterioration in a matter of hours. Thus most likely cause of pneumonia in this child is a Hib infection. This child's blood culture grew H influenza b and he responded to ceftriaxone.

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