a mobile cecum leading to ileocolic intussusception. On the other hand, in case of ileo-ileal intussusception, the neck of the intussusceptum is very tight and therefore obstruction to blood supply and gangrene occur early. This tightness prevents advance of intussuscpiens, so ileo-cecal and ileo-colic intussusception cannot occur. The presentation of ileo-ileal intussusception in early infancy (4-6 months) could be merely reflective of early weaning.

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Hereditary Methemoglobinemia in an Infant

The rare causes of cyanosis in the absence of pulmonary or cardiac disease are methemoglobinemia, sulphhemoglobinemia and carboxyhemoglobinemia(1).

Hereditary or congenital forms of methemoglobinemia are extremely rare(1) and may occur due to NADH-cytochrome b5 reductase deficiency or Hb M disorders.

In view of rare reporting of this condition(2-4), we narrate our experience with an infant with congenital methemoglobinemia.

A 10-month-old baby girl presented with history of fever, cough and respiratory distress for last five days. In the past five months she had similar attacks of respiratory distress twice, that was treated with oral medication. There was no history of any significant drug intake or exposure to well water. Birth, developmental and dietary history was non-contributory. There was history of consanguinity. Mother was married to her own maternal uncle. The baby had a bluish hue since birth, which used to increase during attack of respiratory distress. On examination she had raised temperature, mild respiratory distress and central cyanosis. She had rhonchi and crepts in both the lung fields but other systems were within normal limits. Her pulse-oximetry reading showed oxygen saturation was 85%, Arterial blood gas analysis showed PH 7.38, PaO2 82, PCO2 34, HCO3-24, SaO2 88%. Colour of blood was dark, which was noted during sampling.

She had hemoglobin of 13 gmidL, TLC 16000/cmm, DLC-Neutrophil 30, Lymphocyte 70, ESR-8 mm, PCV 44, and total erythrocyte count 4.6×1012 cells/L. Chest X-ray and electrographic tracing and echocardiographic finding was normal. Hemoglobin
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electrophoresis and G6PD assay was also normal. Methemoglobin assayed by spectroscopic method was 38% (normal ≤1%). Erythrocyte NADH-dependent methemoglobin reductase level was low with 30% of normal. HbM could not be estimated due to financial constraint. She was managed with oxygen inhalation, bronchodilator nebulisation, and oral antibiotics. The respiratory distress settled down after three days but cyanotic hue persisted even at discharge. She was put on oral ascorbic acid and discharged.

Methemoglobin produces detectable cyanosis at concentration. Of 0.5 g/dL(1). In 1948 Horlein and Weber first described a family of hereditary methemoglobinemia in Germany(1). Scott & Griffith in the following year described deficiency of NADH-cytochrome b5 reductase enzyme as the cause of the disease(1). Our case is suffering from hereditary methemoglobinemia (Type I) due to NADH-cytochrome b5 reductase deficiency. Hereditary methemoglobinemia patient generally maintains levels of methemoglobin between 15-30% of normal (1). Treatment is rarely necessary except for cosmetic purpose or with coexisting G6PD deficiency causing superadded acquired methemoglobinemia(5).

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Meconeum Hydrocele Presenting as a Labial Mass

Meconeum peritonitis results from in utero perforation of the bowel and subsequent spillage of meconeum into the peritoneal cavity. Meconeum hydrocele is caused by the free communication of the peritoneal space with the processus vaginalis during gestation. It has been reported often in male scrotums but only one case has been reported in the female neonate earlier ours being the second (1). A one-day-old female baby presented to us at 7 hours after birth with a left labial mass of 7 × 4 × 4 cm, soft, cystic, normal colour of overlying stretched skin, no impulse on crying, noncompressible, positive trans-illumination in the upper 1/5th portion of the swelling and negative below that. The baby passed stool and urine normally on first day of