

the course of illness. This is also known as transient synovitis, observation hip or irritable hip. The diagnosis is one of exclusion and can be made only if other potential causes for painful hip have been eliminated. It usually occurs between 2 and 12 years of age. Spock(6) laid down the following criteria for diagnosing toxic synovitis: (i) pain occurring at rest or with active motion in the involved hip; (ii) restriction in the range of passive motion in the involved hip; (iii) roentgenograms of hip disclose no bony abnormality, and (iv) complete clinical recovery within 2 months. Our patient fulfilled all the criteria. Although, the cause of toxic synovitis is unknown, hypotheses include trauma, infection, and allergy, but none of these has been substantiated(7). In this condition, the fever is absent or low grade, and leucocyte count and sedimentation rate are normal. High fever, leucocytosis and elevated sedimentation rate observed in this patient could be ascribed to the septicemic process. We used ciprofloxacin to treat septicemia. Although, ciprofloxacin is not recommended in children due to fear of arthropathy, it has been used in neonates for treating infections with multiresistant organisms(8). The management of toxic synovitis is expectant. It is a self-limited condition but 1-3% of children with

toxic synovitis go on to develop Perthes' disease(6).

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### Late Sepsis in a G-6-PD Deficient Newborn

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Erythrocyte glucose-6-phosphate dehydrogenase (G6PD) deficiency is a well

known cause of hemolytic jaundice in the newborn period. Less well known is its as-

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sociation with G6PD deficiency in the polymorphonuclear (PMN) leucocytes of these patients. PMN G6PD deficiency increases the susceptibility of these patients to infections with catalase positive organisms(1).

Severe PMN G6PD deficiency has been associated with a mild chronic granulomatous disease. Mallouh *et al.*(2) reported an increased risk of bacterial infections in children between the age of one month and 14 years. One case report of fatal septicemia(3) at the age of two years has been described. To our knowledge there has been a single study by Abu-Osba *et al.*(4) correlating G6PD deficiency with increased susceptibility to late sepsis in the newborn period caused by catalase positive organisms. We report here a case of *Klebsiella* septicemia in a neonate with erythrocyte G6PD deficiency.

#### Case Report

A male baby was born to a 27-year-old primipara at 39 weeks of gestation by a low midcavity forceps delivery. There was thick meconium stained liquor and the one minute apgar score was two. The neonate was resuscitated by intubation and endotracheal suction followed by intermittent positive pressure ventilation.

The Apgar score improved to eight at five minutes and was 10 at ten minutes of age. The birth weight was 2300 g, length 46 cm and head circumference 42 cm.

At 22 hours of life there was asymptomatic hypoglycemia which improved with intravenous dextrose. At 30 hours, the baby developed clinical jaundice. The peak indirect bilirubin was 20 mg/dl at 72 hours of age. Blood group of both mother and baby was B positive and the direct Coombs test was negative. Screening for sepsis was negative. Peripheral smear

showed presence of nucleated red blood cells (4/100 WBC) and reticulocytosis. The packed cell volume dropped from 65% at 22 hours of age to 50% at 30 hours. Red blood cell (RBC) G6PD level at 80 hours of age was 9.9 ( $\mu\text{g/g}$  of hemoglobin (normal 8-18  $\mu\text{g/g}$  of hemoglobin). This was estimated by the rate of increase in NADPH concentration<sup>^</sup>). A repeat RBC G6PD estimation at five weeks of age was 5.4 ( $\mu\text{g/g}$  of hemoglobin. Due to lack of facilities we were unable to quantitate PMN G6PD levels or evaluate PMN function by the nitroblue tetrazolium reduction test(6).

On the seventh day, the baby developed remittent fever and was noticed to have an abscess at the site of an intravenous access. Over the next three days, four more abscesses appeared, unrelated to intravenous sites. A repeat screening for sepsis revealed a significant band neutrophil ratio of 0.3. Pus from the abscess, blood and cerebrospinal fluid were cultured. *Klebsiella* species with identical antibiotic sensitivity patterns were isolated from all three specimens. The abscesses were surgically drained and intravenous ceftazidime and gentamicin were administered for 21 days. The neonate showed good response to antibiotic and supportive therapy and was discharged well on the 25th day of life.

#### Discussion

The fundamental abnormality in the G6PD deficient phagocyte is its inability to mount a respiratory burst with motility, phagocytosis and degranulation being normal. This respiratory burst failure results from lack of NADPH, the reducing agent required for  $\text{O}_2$ -production(1). NADPH production, which is G6PD dependent, is usually affected only when enzyme deficiency is severe.

However, the clinical implications of PMN G6PD deficiency still appear unclear. Rodey *et al*(6) in their study of PMN G6PD levels and bactericidal activity concluded that the susceptibility to infection was probably not a function of the total quantity of the enzyme. Further the increased susceptibility to sepsis may be due to increased iron concentration caused by erythrocyte hemolysis(4). The clinical profile of infections in G6PD deficient neonates include: (i) A male predominance; (ii) Late sepsis (sepsis >72 hours of age): the explanation for this lies in the fact that there is normally a decline in G6PD activity corresponding to the fall in the leucocyte count around the first week of life. This is accentuated in babies with deficiency(4); and (iii) Infection with catalase positive organisms, viz., klebsiella, enterobacter, *E.coli* and *S. epidermidis*(1).

The above features were classically seen in our patient who was a male and developed catalase positive (*Klebsiella*) septicaemia on the seventh day of life. A study of this case and review of relevant literature suggests that G6PD deficiency is a risk factor for severe infections with catalase positive organisms in the newborn period. It

thus seems imperative that infants with G6PD deficiency be aggressively treated for these infections.

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### Etiology of Neonatal Jaundice at Shimla

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The etiological factors responsible for

neonatal jaundice are likely to be affected by the population studies(1), gestational

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