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(l). Scott & Griffith in the following year described deficiency of NADH-cytochrome b5 reductase enzyme as the cause of the disease(l). Our case is suffering from hereditary methemoglobinemia (Type l) 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

life. On the second day of life, the swelling increased in size to 10 × 5 × 5 cm, turned yellowish in colour and there was impending perforation (*Fig.1*). The swelling ruptured spontaneously and the sloughed out skin was excised. The open cavity was dressed with povidone iodine and swelling never refilled. The baby passed stool normally. She was given intravenous antibiotics and discharged after 7 days. The wound healed up with granulation tissue. The baby is doing well at 3 months follow up.

Meconeum peritonitis results from extrusion of sterile meconeum into the peritoneal cavity after fetal perforation. The rich content of digestive enzymes of the meconeum leads to peritoneal irritation and inflammatory response characterized by foreign body granuloma formation and calcification. The majority of patients with meconeum peritonitis require surgical intervention but many have spontaneous resolution by sealing of perforation.

The processus vaginalis forms by 6 months of gestation as an evagination of parietal peritoneum and descends ventral to the gubernaculum into scrotum or labium majus(2). In males, patency is maintained until birth or shortly thereafter with subsequent proximal obliteration leaving the residual tunica vaginalis.

In females, the processus vaginalis is small and obliterated by 8 months of gestation. Autopsy studies have proved that the female gubernaculum or round ligament is different from the male in ending just outside the external ring(3). If patency persists, hydrocele of the canal of Nuck has been described and ultrasound has been suggested as the best mode of diagnosis(4). Delayed presentation may lead to formation of infection. Associated inguinal hernia has been reported in one-third

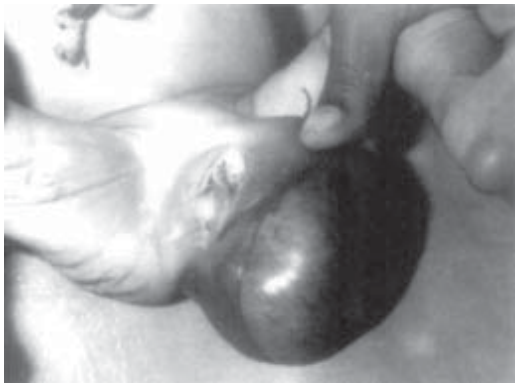

Fig. 1. Meconeum hydrocele of the canal of nuck.

of the cases(5). The differential diagnosis of meconeum hydrocele includes inguinal hernia, ectopic ovary, labial lipoma, fibroma, leiomyoma or hemangioma, round ligament sarcoma, inguinal lymphadenopathy and epidermal cyst(5).

If there is a labioscrotal swelling, this remote possibility should always be kept in mind otherwise it may lead to severe inflammation and spontaneous rupture.

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What is Appropriate Post-Exposure Rabies Prophylaxis?

The report on rabies developing in a child even after immunoprophylaxis has some lessons to teach us(1). Post-exposure prophylaxis consists of wound treatment and immunoprophylaxis. In this child the wound should have been flushed with soap and water immediately after the bite. By taking the child to the dispensary first aid was delayed, but even then soap and water flushing should have preceded providone iodine application. Soap disrupts rabies virus particles instantaneously by dissolving the lipid coat, rendering them non- infectious. Water disperses soap into the wound and also mechanically removes unadsorbed virus particles. Since soap and water are available in every household, there should be no delay in washing after animal bite. There is actually no need of chemical treatment (which require longer contact time to kill virus) after flushing with water and soap.

Viruses deposited in the wound get adsorbed on to local fibroblastic cells within a short period. Soap prevents virus adsorption or kills even adsorbed virus before cell entry. If first aid was delayed or unsuccessful, then virus enters and multiplies in the cells, and huge numbers are released into the surrounding tissue. Eventually viruses meet with nerve endings and enter nerve cells.

Each cell cycle of multiplication takes several hours. Passive immunization provides immediate antibody-block to virus entry in fibroblasts, either in the first cycle itself or in subsequent cycles. Thus, virus entry into nerve endings is prevented, especially by the locally injected immunoglobulin. Antibodies in body fluids (from intramuscular injection) keep up this block. Active immunization ensures sustained high antibody levels.

When the bite is at a site with sparse nerve endings (*e.g*., the leg), there is a window period (of up to a few days) between the bite and virus entry into nerve endings. Only in such cases may active immunization alone suffice. In the recommended vaccination schedule, antibody production would commence after the second dose, and will be detectable by laboratory testing by day 7 (always before day 10). However, if the biting animal was rabid, passive immunization must be given irrespective of the site of bite.

The window period is too short in case of bite on sites with high density of nerve endings (*e.g*., face, hand, genitalia), as in the reported child. Here, viruses may come directly into contact with nerve endings. Any delay, even of minutes, could make the difference between life and death. In general, immune protection will not help once rabies virus has entered the nerve cell. In the reported case there was deficiency in first aid and delay in passive immunization (of 6 hours). The fact that rabies developed after a short incubation period (of 17 days) shows not only that virus travel to the brain was quick, but also that virus entered