Discussion

Congenital granular cell myoblastoma is one of the very rare tumor seen in pediatric age group. The female preponderance and failure to grow postnatally suggests the hypothesis of some intrauterine stimulus, Estrogen derived from fetal ovaries under the stimulus of chorionic gonadotropins may be this intrauterine stimulus. Various theories of the histogenesis of these neoplasms have been suggested ranging from lontogenic, fibroblastic, histiocytic, myogenic and neurogenic as reviewed by Blair and Edwards(4). The electron microscopic and immunocytochemical studies have indicated it to be of Schwann cell origin.

The present case was seen in an eight hour old neonate and was treated by complete surgical excision without recurrence after one year of excision which is in agreement with earlier experienced). The case presented characteristic histology of a benign granular cell myoblastoma.

REFERENCES


Spontaneous Gastric Perforation in a Neonate

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Spontaneous gastric perforation of the newborn is a rare entity with just over 200 cases reported in literature(1). Although the condition entails a very high mortality, its pathogenesis is greatly debated. Congenital absence of the gastric wall musculature(2), stress ulceration secondary to neureogenic difficulties(3) and ischemia of the gastric wall secondary to vascular shunting(4) have been proposed as etiologic factors. We report a case of spontaneous gastric perforation seen by us in a one-day-old baby.

Case Report

A one-day-old male presented with fast respiration and abdominal distension since birth. There was no history of vomiting and the baby had passed meconium. The baby
had not accepted feeds since birth. He was a full term normal vaginal delivery at home conducted by an Auxiliary Nurse Midwife. There was history of birth asphyxia with the baby having cried 15 minutes after birth. On examination, the baby was full term appropriate for gestational age and weighed 3.2 kg. The general condition was not satisfactory and the cry, activity and neonatal reflexes were depressed. He was markedly dyspneic with a respiratory rate of 120/min. The heart rate was 148/min. The abdomen was markedly distended with absent bowel sounds and the liver dullness was masked. Rest of the examination was within normal limits.

On investigation, hemoglobin was 18 g/dl and TLC 16,000 cu/mm (P75 L20 M3 E2). X-ray abdomen erect film showed free gas under the diaphragm. The viscera were displaced medially, producing the typical 'saddlebag' appearance (Fig. 1). The X-ray chest, blood urea and serum electrolytes were within normal limits.

A diagnosis of gastric perforation was made and an urgent laparotomy undertaken. This revealed a linear perforation 1 cm long with ragged edges, on the anterior wall, near the gastroesophageal junction. The surrounding stomach wall was ischemic. A wide excision of the ischemic edges was done and the perforation was closed in two layers with silk. Postoperatively intravenous fluids were administered and nasogastric aspiration done every 2 hours. Intragastric feeds were started on 5th postoperative day. The baby made uneventful recovery and was discharged on the 14th postoperative day.

Discussion

Spontaneous gastric perforation as reported earlier is more common in the pre-term baby with perforations most commonly occurring between the 2nd and 7th days of age(5,6). The highest reported incidence of rupture is on the 3rd day of life(6). Our baby was a full term baby and the perforation had occurred on the first day of life. Sudden abdominal distension and respiratory distress have been the reported predominant symptoms(1,5). The incidence of prenatal and perinatal risk factors is reportedly high(5,7). Those commonly observed include prematurity, respiratory distress at birth, asphyxia and resuscitation, premature rupture of membranes and breech, Cesarean or twin delivery, in decreasing order of frequency. Severe birth asphyxia was the high risk factor seen in the present case.
Most commonly the perforations have been linear tears, seen on the greater curvature, usually high and measured between 0.5-8 cm (1,5). In the present case, the perforation was also a linear tear and measured 1 cm. It was located on the upper anterior wall, close to the gastroesophageal junction.

Prompt surgical intervention with repair of the gastric tear is the recommended management. Any delay in surgery will result in a higher mortality (8,9).

The pathogenesis of spontaneous gastric perforation is much debated (2-4,10-12). Gastric tissue ischemia secondary to hypoxia is one plausible explanation (4, 11). During severe hypoxic stress there is selective shunting of blood away from the splanchnic vascular bed leading to vascular damage and perforation. This was the probable sequence of events that operated in our patient.

REFERENCES