Acute gastric dilatation secondary to septicemia in newborn: A rare entity

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ABSTRACT

Acute gastric dilatation in a newborn is a rarely encountered clinical entity. Such dilatation without any obstruction is furthermore rare. The authors hereby present a rare case of acute gastric dilatation that developed in a two-days old child who was born premature and was suffering from septicemia and respiratory distress. The causes and the differential diagnosis of the rare condition are discussed.

Key words: Acute, dilatation, gastric, newborn

INTRODUCTION

Acute gastric dilatation (AGD) in newborn is rarely encountered in clinical practice. It may be obstructive or non-obstructive.[1] Non-obstructive acute gastric dilatation is a rare and poorly documented cause of AGD in medical literature. Various causes have been suggested but non-obstructive AGD is usually the diagnosis of exclusion based on clinical, biochemical and radiological findings. On the other hand, obstruction secondary to gastric atresia, antral webs/stenosis is a rare but well documented cause of AGD.

CASE REPORT

A 34-weeks child was born to a G2P1A0 mother by emergency lower segment Cesarian section (LSCS) elsewhere in view of fetal distress and premature rupture of membranes. There was a history of meconium aspiration and birth asphyxia. The child cried approximately after 3-5 minutes of birth following rigorous efforts of resuscitation. History of cyanosis and groaning was also positive. Antenatal ultrasound done elsewhere was unremarkable for any congenital malformation including gastrointestinal obstruction.

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The child was brought to our hospital after 36 hours of birth. At the time of admission, clinical examination revealed hypothermia, lethargy, altered sensorium with Glasgow coma scale of E1M2V2. Deep tendon reflexes were brisk and pupils were sluggishly reacting to light. The child was tachypnic (RR=60/minute) with irregular and feeble breathing efforts alternating with apneic episodes. There was presence of cutaneous cyanosis and sclerema with a weak cry tone. Auscultation revealed decreased air entry on both sides with presence of conductive breath sounds. Abdomen was soft with few bowel sounds on auscultation. Rest of the clinical examination was unremarkable.

At the time of admission, SPO_2 was only 40%. Laboratory examination revealed high titer of C-reactive protein but the rest of the tests including TLC, DLC, Serum Na^+, K^+ and Ca^{++}, blood urea and serum creatinine were within normal limits.

Immediately after admission, the child was intubated with a noncuffed, endotracheal tube and put on positive pressure ventilation at optimized pressure and flow rates. Proper positioning of endotracheal tube was ensured. This was followed by nasogastric intubation which revealed a bloody aspirate of 3-5 ml. In view of the clinical and biochemical findings, appropriate therapeutic management was instituted.

During the hospital stay, the child could not achieve sufficient oxygen saturation of the arterial blood in spite of assisted ventilation. As there was no significant improvement in mental and respiratory status, the child was advised x-ray chest including abdomen and ultrasound of cranium and abdomen. Intermitted passage of meconium was noted. Urine output was within normal limits and nasogastric aspirate was insignificant.

Radiograph of the chest and abdomen [Figure 1] revealed diffuse infiltrative opacities involving all zones in right lung with presence of few areas of air bronchogram. Left lung was partially visualised but revealed pulmonary infiltrates similar to right lung.

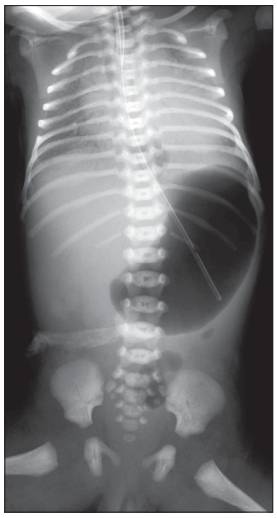


Figure 1: Anteroposterior radiograph of chest and abdomen taken in supine position reveals bilateral pulmonary infiltrates especially on right side along with moderately dilated gas filled stomach and small amount of gas in distal bowel. Nasogastric and endotracheal tube is seen *in situ*

The volume of the lungs, rib cage and cardiac shadow was, however, within normal limits. The abdomen revealed moderately dilated stomach filled with air with nasogastric tube the left upper abdomen. Air shadows were seen in the distal bowel as well. US of the cranium and abdomen were unremarkable and there was no evidence of any antral thickening or diaphragm.

Based on the clinical, biochemical and radiological investigations, the provisional diagnosis of septicemia with acute gastric dilatation, respiratory distress and altered sensorium was made. However, the child developed cardiorespiratory arrest approximately 2 hours after imaging and within 24 hours following admission from which the child could not be revived.

DISCUSSION

Acute gastric distension or dilatation (AGD) in a young neonate is a rare clinical event. The causes can be broadly divided in to obstructive and non-obstructive categories.

The usual cause of obstruction is the presence of gastric atresia, antral stenosis or webs preventing gastric outflow with resultant gastric dilatation.[1] Other rare causes include antral valve, ectopic pyloric pancreas and pyloric duplication cyst.[2-4] Such newborns usually present with projectile, non-bilious vomiting especially after feeds. Clinical examination may reveal distension of abdomen with constant nasogastric aspirate with presence of dilated stomach and absence of gas in distal bowel radiographically. In some cases, there may be a palpable lump as in cases of duplication cyst. However, if the obstruction is incomplete, the obstructive features will be less severe and gases will be seen in distal bowel as well. Contrast upper gastrointestinal studies often aid in diagnosis especially in establishing the obstructive nature of the lesion. Real time ultrasound scanning after saline loading of the newborn stomach often helps in the visualising the obstructing lesions like webs or valves.[1,2] Antenatal ultrasound in cases of gastric outlet obstruction will reveal dilated stomach with presence of polyhydramnios. These features were not seen in our index case which helped us to exclude these causes.

Non-obstructive causes of AGD include excessive air swallowing which may be due to respiratory distress, improper endotracheal tube insertion, and tracheoesophageal fistula with lower segment communicating with trachea.[5] Acute gastric dilatation can also be secondary to metabolic derangements especially hypocalcemia and septic states with resulting gastric hypotonia.[6] AGD has been associated with use of mydriatics in preterm infants.[7] Reports of AGD associated with cerebral palsy or severe mental retardation have also been described in children.[8] The latter may be due to autonomic neuropathy or neuromuscular in-coordination. It is possible in our case that severe birth asphyxia might have affected the autonomic nervous system leading to neuromuscular in-coordination with resulting respiratory distress and acute gastric dilatation.

The importance of recognizing AGD lies in the fact that such cases may progress to spontaneous gastric perforation or apnea due to vagal stimulation caused by profound degree of gastric distension sometimes leading to unexpected and spontaneous death.[1,8,9]

However, the diagnosis of non-obstructive AGD is based upon exclusion of obstructive causes which in our case was done clinically and by imaging. Among the non-obstructive causes, iatrogenic cause i.e., improper endotracheal intubation is an important cause and should always be excluded. Tracheoesophageal fistula is ruled out, once nasogastric intubation is successful. Children with vigorous respiratory efforts can also swallow excessive air leading to gastric and small bowel distension with air subsequently resulting in airy nasogastric aspirate. In our case, the newborn was having feeble spontaneous breathing and nasogastric aspirate was unremarkable. Dyselectrolytemia especially hypocalcemia produced hypotonia of bowel and always

needs exclusion in such cases as was done in our case. Sepsis as described by Cisneros-Garcia *et al.* is a rarely reported cause and should be suspected once other causes has been ruled out.[6] AGD has a good prognosis upon treating the underlying cause. The usual treatment is orogastric drainage. Treatment with antibiotics is indicated in cases of septicemia.

In our case, though the patient could not be saved but the diagnosis of septicemia with acute gastric dilatation was made when all the other obstructive and non-obstructive causes were ruled out.

To summarise, acute gastric distension in a newborn is a rare entity but can occur secondary to sepsis and dyselectrolytemia. Early recognition of condition and its underlying treatable cause is mandatory to reduce the morbidity and mortality associated with this condition.

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