A Rare Cause of Gastric Outlet Obstruction in an Infant

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A 6-week-old female presented with non-bilious vomiting since the age of one week. The vomits were after each feed and consisted of milk of moderate to large in amount. Her stools were of normal color and consistency. The antenatal history was uneventful with a normal antenatal scan. She was born at 37 weeks of gestation with a birth weight of 2.6 kg. She passed meconium within the 24 hours of life. Examination upon admission revealed an emaciated, pale infant with decreased muscle mass and reduced subcutaneous fat. Her weight was 2.6 kg. Abdominal examination revealed a soft abdomen with a liver measuring one cm below the costal margin but no other masses. Other systemic examinations were unremarkable. Investigations revealed hyponatremia, Sodium was 118 mmol/L (ref. range: 135-145), hypokalemia, Potassium was 2.6 mmol/L (3.5-5.5) with hypochloremia, chloride was 77 mmol/L (97-107), metabolic alkalosis and bicarbonate was 42 mmol/L (22-29). The ultrasound abdomen showed a normal partially distended stomach with a pyloric width of 5.5 mm (normal <7 mm) and a pyloric length of 12 mm (normal length <14 mm). The sweat test result and renin and aldosterone levels were normal. Urine sodium, urine, and serum osmolality were normal. Subsequently, the patient underwent a water-soluble upper gastrointestinal meal and followed through [Figure 1].

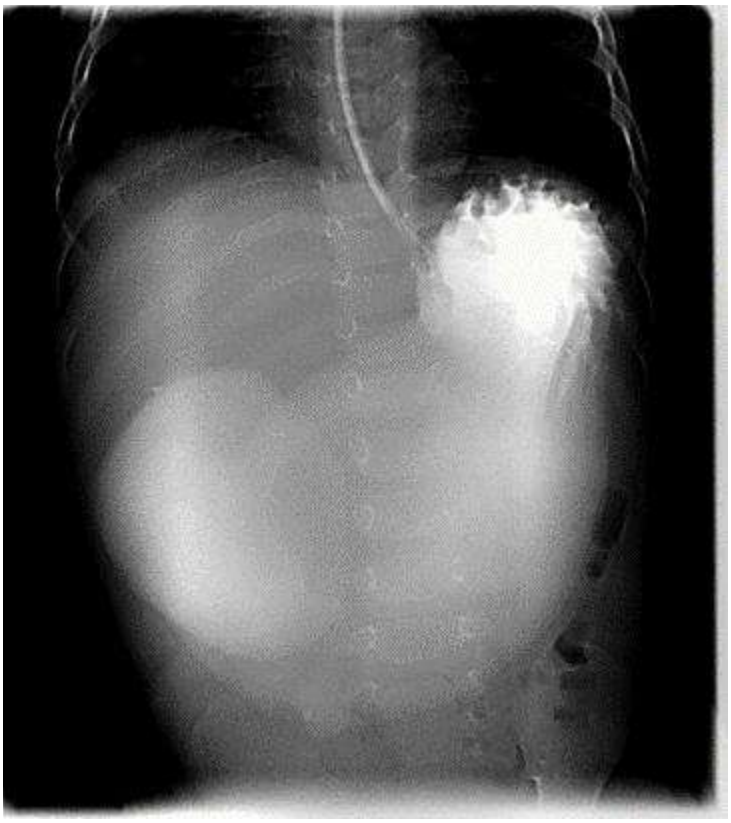

Figure 1: Anterior-posterior image of the upper gastrointestinal study shows a markedly distended stomach filled with water soluble contrast (blue arrow). Oral contrast is seen in the first part of the duodenum (green arrow). No contrast is seen beyond this point.

Questions

1. What is the likely diagnosis?

2. How commonly do patients with this condition present?

3. How would you manage this condition?

4. What is the underlying pathophysiology behind the metabolic alkalosis associated with this condition?

Answers

1. Gastric outlet obstruction due to duodenal web.

2. Either antenatal or postnatally. It can be diagnosed reliably by prenatal ultrasound. They may manifest as polyhydramnios or dilated bowel loops. Postnatally, the age of presentation and degree of obstruction are determined by the size of the duodenal diaphragm's aperture. Therefore, it can present as non-bilious vomiting and abdominal distention in neonates, delayed growth and vomiting or recurrent respiratory infections in infants and toddlers.

3. Gastric decompression, fluid resuscitation, and electrolytes correction, followed by surgical correction. Endoscopic dilatation in certain circumstance can be used.

4. Metabolic alkalosis results from the loss of hydrogen, movement of hydrogen into cells, administration of alkali, or contraction of volume with a constant amount of extracellular bicarbonate.

Discussion

After stabilizing the infant and correcting the electrolyte disturbance, the infant was taken up for laparoscopy. She had a thick duodenal diaphragm just above the ampulla of Vater. Therefore, duodenoduodenostomy was performed. Postoperatively, the patient recovered well and started enteral feeding after a few days, which she tolerated well.

One of the most common congenital anomalies is congenital duodenal obstruction, which accounts for approximately 50% of cases of neonatal intestinal obstruction. The cause is either an intrinsic defect (atresia, diaphragm or stenosis) or extrinsic compression due to annular pancreas, malrotation or pre-duodenal portal vein.[1] Congenital duodenal diaphragm or web is uncommon, and its common location is the second part of the duodenum, followed by the third and fourth parts. The duodenal diaphragm (DD) causes partial or complete obstruction. The age of presentation, degree of obstruction, and radiological findings are determined by the size of the duodenal diaphragm's aperture.[2] Typically neonates present in the first few hours or days of life with vomiting, mostly non-bilious, and abdominal distention. Symptoms are usually less obvious in patients with later-onset clinical symptoms presenting with abdominal distension, delayed growth, or recurrent respiratory infections.[2]

The most prominent clinical finding at presentation in our patient was metabolic alkalosis. This can result from the loss of hydrogen, movement of hydrogen into cells, administration of alkali, or contraction of volume with a constant amount of extracellular bicarbonate. The most common causes are hydrogen ion loss from the gastrointestinal tract or urine, which is usually accompanied by hypokalemia. Hyponatremia was also noted in our patient. During the early stages of DD, sodium wasting is common. This occurs because the sodium bicarbonate levels rapidly rise and exceed the renal threshold, as sodium wasting occurs because the hypovolemic drive to retain sodium is counteracted by excretion of sodium and potassium bicarbonate.[3] The differential diagnoses considered in our patient were adrenal insufficiency, inborn error of metabolism, intestinal obstruction, gastroesophageal reflux disease, milk protein allergy, malrotation, and pyloric stenosis.[4]

DD can be diagnosed prenatally or postnatally. In prenatal ultrasound polyhydramnios or dilated bowel loops can be detected. Postnatal ultrasound can confirm an annular pancreas, duplication cyst, preduodenal portal vein, or pyloric stenosis. The ultrasound of the abdomen was inconclusive in our patient. The other radiologic investigation was abdominal radiography, which demonstrated a "double bubble" sign indicative of gas distension in the stomach and proximal duodenum. Radiograph is usually followed by an upper gastrointestinal series. It may reveal the "windsock" sign or deformity, which occurs when the membrane or web within the duodenum balloons distally into the lumen, creating a windsock-like appearance on imaging. This results from contrast material being trapped behind the obstructive web. This was proved to be pivotal for identifying the approximate obstruction level in our case. Computed tomography (CT) and magnetic resonance imaging (MRI) are not usually required, except for the evaluation of vascular anomalies, if suspected.[5]

For any type of intestinal obstruction, initial management should include gastric decompression, fluid resuscitation, and electrolyte correction. Serum electrolytes and bicarbonate levels should be normal before surgery. The preferred surgical approach for DD is duodenostomy excision while avoiding injury to the ampulla. The survival rate of patients with DD after surgery is approximately 100% in the absence of prematurity and other serious anomalies.[5] We performed duodenoduodenostomy employing laproscopy. However, it can be managed safely and effectively by endoscopic dilatation, however, this method has some limitations. The child's size, for instance, should be considered, because infant weighing less than 5 kg will

probably not be amenable for this technique as the infant's size will not allow the adult endoscope to be utilized. This was the situation with our patient. The anatomy and the location of the web is another drawback, as an endoscopic intervention would not be feasible for a fully occlusive web since it would be unable to pass a guidewire and the balloon safely into place.[6]

In conclusion, DD is a rare cause of intestinal obstruction that requires early recognition and surgical treatment.

Disclosure

Informed consent was obtained from the patient's mother.

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