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The management of bilious vomiting in the neonate

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ABSTRACT

Bilious vomiting is synonymous with intestinal obstruction, be it functional or anatomical. In the neonate it may be due to congenital malformations of the gastrointestinal tract or develop due to acquired conditions, particularly intestinal complications associated with prematurity. This review considers the congenital malformations that may present with bilious vomiting and explores the diagnostic dilemmas faced in the preterm infant. The difficult issue of the need to exclude malrotation in term infants with bilious vomiting and the consequences of time-critical transfer is discussed.

Contents

1. Background

Bile is produced in the gall bladder and drains into the duodenum. It is luminous yellow in colour and is not normally present in vomitus unless flow beyond this point is prevented, when it will appear in the stomach and be turned green by the action of gastric acid. Bilious vomiting should be assumed to be due to intestinal obstruction (mechanical or functional) until proved otherwise. The importance of this sign stems from the knowledge that whatever pathology is causing luminal obstruction may also be causing vascular obstruction. Whilst luminal obstruction might be managed conservatively, obstruction to the vascular supply of the gut, if not relieved within a few hours, may lead to death of the intestine and possibly the patient. Although in neonates there are

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many causes of intestinal obstruction which do not carry a risk of intestinal ischaemia the presence of bilious vomiting associated with abdominal tenderness requires emergency surgical assessment.

Defining which colours represent bile in vomit has proved challenging and although most clinicians would recognise bile in vomiting as green in colour, the vomiting of yellow fluid does not exclude the presence of obstruction [1].

The classic triad of features of intestinal obstruction at any age are bilious vomiting, absolute constipation (in the neonate failure to pass meconium) and abdominal distension. Although this triad can be applied to the neonate with obstruction there are some infants who do not vomit bile, some who may pass meconium, and some who may not be distended.

The initial management of intestinal obstruction includes intravenous access to provide fluid resuscitation, the passage of suitably sized nasogastric tube to achieve decompression of the stomach by regular aspiration and provision of maintenance intravenous fluids plus replacement of nasogastric aspirates. These management steps are applicable to most if not all of the conditions described in this article.

Once initial resuscitation and the above management have been instituted it is then necessary to establish the cause of the bilious vomiting. For the purpose of this review these have been divided into congenital malformations and acquired disorders particularly associated with prematurity.

2. Congenital abnormalities

There are a number of congenital malformations that may occur in the newborn which may present as intestinal obstruction associated with bilious vomiting which are considered below in anatomical order from stomach to anus.

2.1. Duodenal atresia

This occurs in about 1 in 5000 infants and in 30% of cases is associated with Down's syndrome. The condition is diagnosed on antenatal ultrasound scan in about 50% of cases. Infants born with this condition may exhibit all three typical features of intestinal obstruction including bilious vomiting but in a third of infants the obstruction it is proximal to the insertion of the bile duct into the duodenum and so vomiting is non-bilious. Abdominal distension may not be very obvious and if it is it may be confined to the upper abdomen as the rest of intestine downstream of the atresia will be empty. Diagnosis is usually easily made on an abdominal X-ray which will demonstrate a typical double-bubble appearance. Surgery is usually performed in the first few days of life and is not critical emergency in most cases. Post-operative management specific to duodenal atresia relates to the provision of nutrition. Because duodenal motility is usually deranged as a result of long-standing obstruction many practitioners advocate intravenous nutrition. However this may be avoided by siting a feeding tube through the anastomosis at surgery to enable jejunal feeding until duodenal function has returned thus avoiding the need for intravenous feeding and central venous access [2]. With either technique full oral enteral feeding is usually achieved within 2–3 weeks of life.

2.2. Malrotation

The incidence of malrotation presenting in the neonatal period is estimated at 1:6000 live births [3]. Although the anatomy of the intestine can be quite variable, the main concern is that abnormal fixation of the small bowel can result in the root of the mesentery, providing the blood supply to the whole of the small bowel, being on a thin stalk which is prone to twisting (volvulus) (Fig. 1). If the twist occurs and vascular obstruction results from it then the intestine may be dead within a few hours.

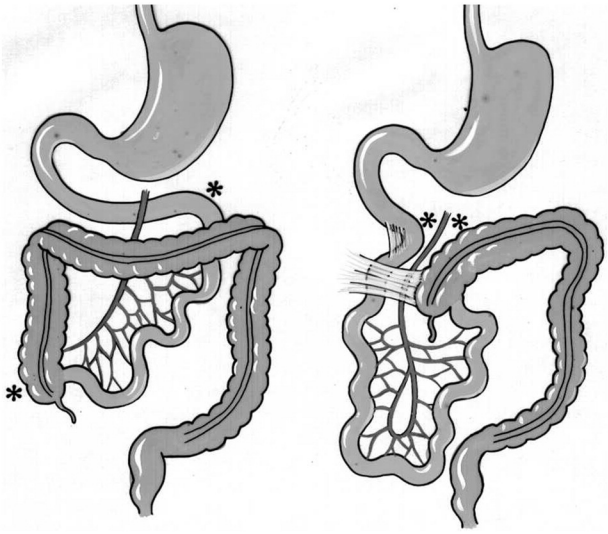

Fig. 1. Intestinal anatomy: normal (left) and malrotation (right). The asterisks represent the points defining the root of the mesentry.
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The clinical scenario is usually that of a term baby in the first 2–3 days of life who presents with bilious vomiting which may be intermittent. The infant may be otherwise very well with no abnormal clinical signs although if volvulus is present there may be associated gastrointestinal bleeding, abdominal tenderness and severe systemic disturbance. The diagnosis may be suspected on the basis of an asymmetric gas pattern on abdominal X-ray (Fig. 2) although in many cases an abdominal X-ray is unhelpful. The diagnosis is confirmed by performing an upper gastrointestinal contrast study at which the position of the duodenum, jejunum and small bowel can be ascertained. Immediate laparotomy is indicated because of the risk of volvulus. In most

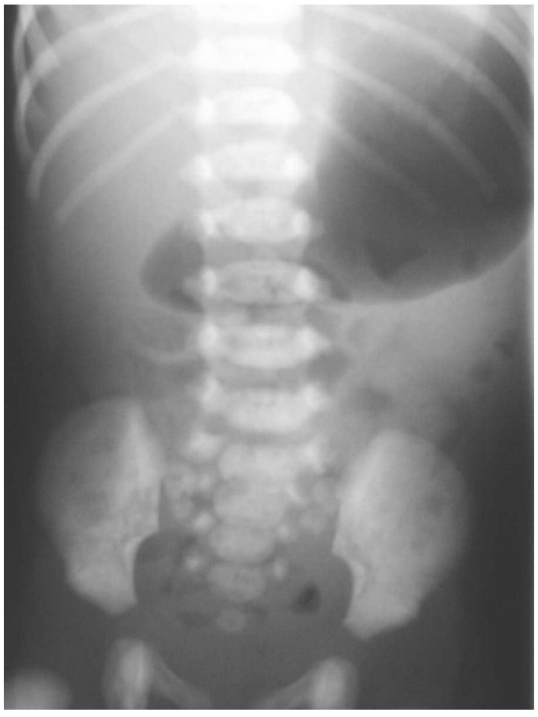

Fig. 2. Abdominal X-ray in a patient with malrotation showing asymmetry of gas pattern.
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