PERSONAL PRACTICE

Intestinal dilatation in the fetus

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Introduction

There are now sufficient data on the sonographic appearance of normal fetal gut to allow intestinal abnormality in the antenatal period to be diagnosed, although as yet relatively little attention has been paid to the implications for the child. We present eight such cases. Six children were found to have midgut atresia, three of whom had evidence of antenatal perforation. Two of the six had intestinal malrotation, which we believe, was responsible for the atresia and two had meconium ileus not associated with cystic fibrosis. One child was found to have intestinal pseudo-obstruction and one a high anorectal malformation.

Intestinal dilatation in the otherwise well newborn child is usually due to obstruction. The differential diagnosis includes mid and hindgut atresia, meconium ileus, Hirschsprung's disease and midgut volvulus. Although the first two conditions soon declare their presence, Hirschsprung's disease and the intestinal malrotation that leads to volvulus may not be apparent at birth.

Structural abnormalities in the fetus are revealed in considerable detail by ultrasonography. The appearances of anomalies in the neural tube, abdominal wall, and renal tract are relatively well established, but description of the pathology affecting the fetal bowel is still far from complete.

The first report of bowel dilatation in the fetus was by Lee and Warren in 1977. They found multiple dilated tubular structures on ultrasound scan at 35 weeks' gestation.[1] Jejunal atresia was found during surgery in the early neonatal period.

Subsequent reports confirmed that fetal intestinal obstruction can be detected by ultrasound scanning in the antenatal period. The pathology included midgut or hindgut atresia, meconium ileus, malrotation and Hirschsprung's disease. Meconium cyst and peritonitis following perforation have also been described.

Eight such instances were noted at St George's Hospital, London, between 1987 and 1994. Each had an ultrasonic diagnosis of intestinal dilatation in the antenatal period with intestinal pathology confirmed after the child was born. Six children had midgut atresia, three of whom had evidence of antenatal perforation. Two of these had intestinal malrotation which we believe was responsible for the atresia. Two had meconium ileus, but no evidence of cystic fibrosis. One child was found to have intestinal pseudo-obstruction and one an anorectal malformation. A ninth child was observed to have an intrauterine volvulus, leading to ileal atresia, but dilated bowel was not reported antenatally. The sonographic appearances

Table 1 Summary of patients at St George's Hospital

Case No	Indication	Gestational age	Appearances	Diagnosis	Outcome
1	Polyhydramnios	33	Dilated bowel (20 mm)	Ileal atresia+meconium peritonitis	Ileal resection, now well
2	Unknown	35	Dilated bowel (20 mm)	Malrotation neuropathic pseudo-obstruction	Ladd's procedure, remains TPN dependent
3	Maternal systemic lupus erythematosus	19	Echogenic small bowel+dilatation (5–6 mm)	Meconium ileus with secondary ileal atresia, segmental volvulus, and meconium cyst	Premature labour, ileal resection, now well, cystic fibrosis not confirmed
		20	Dilated loops (7–8 mm)		
		28	Dilated loops (10 mm)+meconium cyst		
4	Routine	19	Cystic structure	Malrotation ileal atresia ×2	Ladd's procedure ileal resection, now well
		34	Dilated bowel+hyperperistalsis		
5	Routine	21	Dilated bowel	Malrotation apple-peel midgut atresia	Ladd's procedure ileal resection, now well
		32	Progressive dilatation+active peristalsis		
6	Maternal diabetes	19	Echogenic bowel	Meconium ileus with secondary ileal atresia and meconium peritonitis	Ileal resection, now well, cystic fibrosis not confirmed
		31	Dilated bowel (15 mm)		
		33	Dilated bowel (30 mm)		
		35	Aminocentesis bile stained		
7	Routine	20	Echogenic bowel	High anorectal malformation, no other anomalies	Neonatal colostomy awaiting posterior saggital anorectoplasty
		22	Cystic areas in abdomen		
		30	Dilated bowel (20 mm)		
8	Routine	22	Dilated bowel	Jejunal atresia	Jejunal resection, now well
9	Routine	21	Abdominal mass	Intrauterine volvulus ileal atresia	Ileal atresia, now well
		33	Mass resolved		

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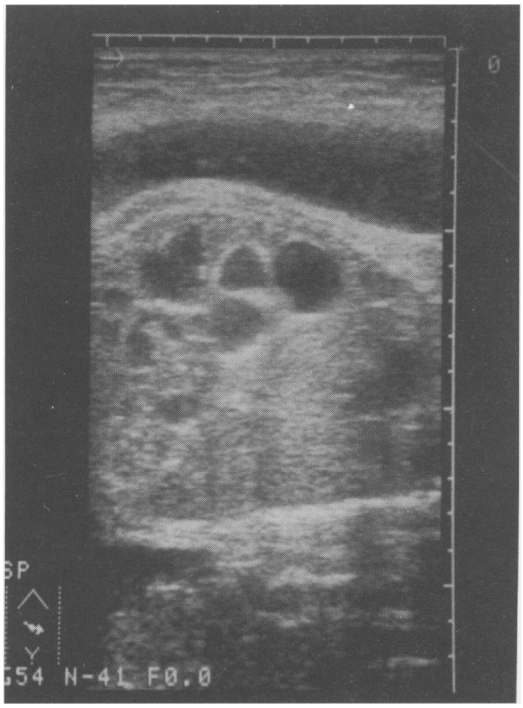

Figure 1 Ultrasound scan at 33 weeks' gestation showing multiple loops of dilated gut measuring 20 mm in cross section (from case 1).

and postnatal diagnoses are summarised in table 1.

The antenatal appearances of ileal atresia, together with the corresponding postnatal contrast enema, are shown in figs 1 and 2.

Published findings

Our experience is consistent with published work and confirms that intestinal pathology may be diagnosed before birth. Detection of abnormality has been facilitated by a number of careful studies of the normal fetus.

Zilanti and Fernandez followed up the changing appearances of the fetal intestine between 26 and 41 weeks in 81 normal pregnancies.[2] Four stages were described, during which the fetal bowel, initially uniformly grey in appearance, became increasingly well defined during the third trimester. These changing appearances were attributed to increasing intestinal content as gestation progressed. Colon was defined earlier than small bowel and had a predictable position.

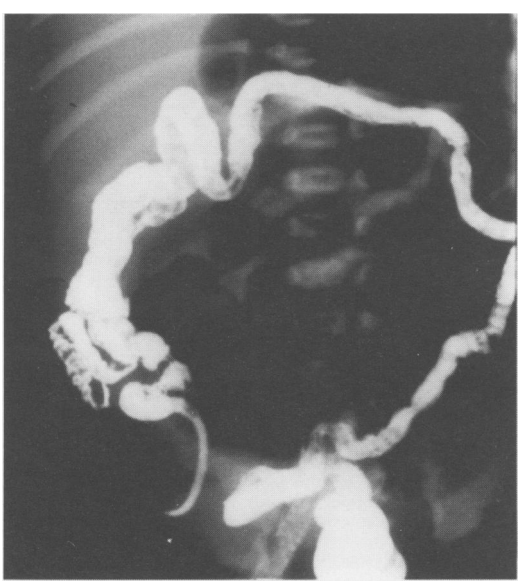

Figure 2 Contrast enemas showing a microcolon without reflux of contrast into the gas filled dilated loops of small gut (from case 1).

Nyberg *et al* were able to identify colon in some fetuses as early as 22 weeks, and in all after 28 weeks.[3] Discrete small bowel segments appeared later, and were seen in only a third of fetuses examined after 34 weeks. Small bowel loops never exceeded 7 mm in diameter or 15 mm in length, while the colonic diameter increased during pregnancy, reaching values of up to 18 mm at term. The observations were based on a study of 130 normal fetuses during the second half of gestation.

Parulekar also detected an increase in bowel diameter with gestation, observing that small bowel rarely exceeded 6 mm and the colon 23 mm in diameter.[4] Individual bowel segments were identified at a much earlier stage than in previous reports. Small bowel was identified in 44% of fetuses between 10–15 weeks and in 100% after 20 weeks. Colon was identified in 44% of fetuses between 18–20 weeks and in 100% after 25 weeks. Increasing small bowel peristalsis was detected after 25 weeks and colonic haustrae were identified in most fetuses over 30 weeks. This study was based on 243 normal fetuses between 10 and 40 or more weeks of gestation.

These studies emphasise the importance of gestational age in the assessment of pathology in the fetal intestine. There are 43 reports of dilated intestine in the fetus associated with pathology in the newborn child (table 2).[5–32] These cases were observed relatively late in pregnancy, frequently only after 30 weeks' gestation. Many of our cases appear exceptional in this respect. Late diagnosis reflects the relatively poor definition of the fetal gastrointestinal tract by ultrasound scanning during the first half of pregnancy. These anomalies will not be detected at the time of routine scanning in most practices.

Complications associated with the obstruction were detected in several cases. Thus intestinal perforation was apparent as fetal ascites, extraluminal calcification, or meconium cyst, and volvulus appeared as a multiloculated mass.

Intestinal dilatation does not always mean obstruction. In our second case the antenatal appearances were probably due to the pseudo-obstruction rather than the malrotation. Congenital chloride diarrhoea may present with dilated bowel.[33] Conversely, mesenteric cysts and polycystic kidneys have been misinterpreted as dilated intestine.

It is appropriate to ask whether diagnosis before birth confers any advantage to the mother or child. Most of the intestinal abnormalities detected in the fetus are readily detected after birth and do not present a life threatening emergency. Furthermore, intestinal dilatation in the fetus is not always associated with pathology in the newborn child.

Table 2 Summary of findings in 43 reported cases

Diagnosis	*Total cases*	*Complications*			
		Perforation	*Volvulus*	*Atresia*	*Polyhydramnios*
Midgut atresia	15	5	4	–	8
Anorectal atresia	9	0	0	0	1
Hirschsprung's disease	2	0	0	0	2
Meconium ileus	11	5	3	3	6
Malrotation	5	1	3	4	4
Duplication cyst	1	0	1	0	1

There is certainly one, and probably two, potentially lethal conditions detectable in the antenatal period which may not be apparent at birth. The first is Hirschsprung's disease and the second midgut malrotation. In both the intestinal obstruction may be intermittent and failure to diagnose either leaves the child exposed to dangerous complications.

Vermesh reported fetal intestinal distension and polyhydramnios in one pregnancy which resulted in the birth of an apparently normal baby.[25] This child was admitted as an emergency at the age of 2 months and subsequently found to have Hirschsprung's disease. The mortality from enterocolitis in Hirschsprung's disease is still 30% and the incidence is higher if the condition is not diagnosed and treated. Malrotation complicated by midgut volvulus results in intestinal obstruction. This obstruction may be intermittent until extensive necrosis occurs following occlusion of the superior mesenteric vessels. To date, antenatal diagnosis of malrotation has depended on detection of complications of the volvulus: atresia or perforation, often with extensive damage. The ability to observe intestine in the fetus by ultrasound scanning means that the obstruction can be detected consequent on volvulus before ischaemic damage occurs.

We believe that an apparently normal child with a history of intestinal dilatation in the antenatal period should have Hirschsprung's disease excluded by suction biopsy of the rectal mucosa, and intestinal malrotation by a limited upper gastrointestinal contrast study.

If a fetus shows an abnormality which may need treatment in the early neonatal period there is a strong case for planning delivery in a unit capable of undertaking all the investigations, support, and treatment the child may need. Mother and baby should not have to be separated soon after birth, and there should be no need for the child to undergo a potentially hazardous ambulance transfer. A scheme for the postnatal management of a child in whom bowel dilatation was detected on antenatal ultrasound scan is set out in fig 3.

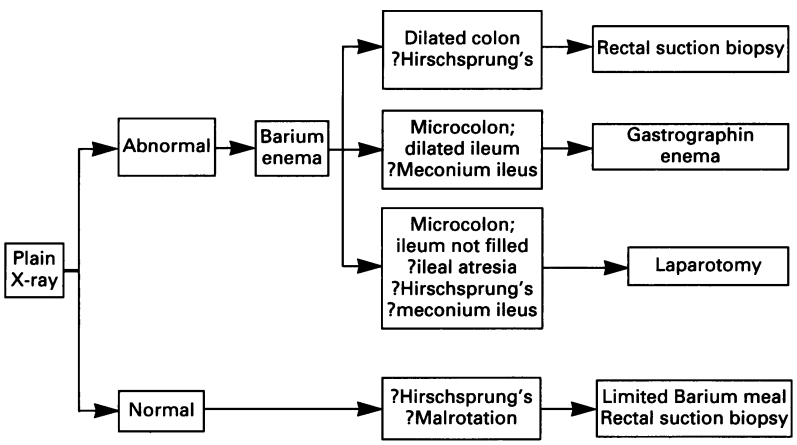

Figure 3 Postnatal management.

Conclusions

Over the past few years, studies giving values for intestinal diameter in the normal fetus have contributed to the objective diagnosis of intestinal pathology in the antenatal period. The absence of intestinal gas and the dynamic nature of the ultrasound examination seem to make the technique more accurate in the fetus than in the newborn child. Uncertainty may still arise due to overlap between normal and abnormal subjects and because of the difficulty in distinguishing small from large intestine. Pathology is more likely in the presence of polyhydramnios, meconium peritonitis, and other structural anomalies and if the dilatation increases with time.

Our proposed scheme of management of suspected fetal intestinal obstruction is as follows:

If dilated bowel loops are seen on antenatal ultrasound permanent copies should be made, with objective measurements of diameter, to permit comparison with normal values. A search should be made for associated features such as fetal ascites, peritoneal calcification, and meconium cyst. Serial scans would give invaluable information on the natural history of the condition. The paediatric surgical team should be involved at an early stage so that the implications of the findings can be discussed with the parents, and to facilitate further management.

We believe that all these babies should be followed up closely after birth. Any who have evidence of intestinal obstruction after delivery would be managed along conventional lines: physical examination, followed by contrast enema, rectal suction biopsy or operation as indicated. Children who appear normal should be investigated for Hirschsprung's disease and intestinal malrotation.

We do not yet have sufficient data to justify early delivery in the hope of preventing intestinal damage. Nevertheless, one of our patients had pronounced dilatation at 33 weeks which then decreased suddenly. Laparotomy after birth revealed a recent perforation.

This proposed scheme of management requires close cooperation between obstetricians, paediatricians, and paediatric surgeons. Only by conscientious monitoring before and after birth will the importance of fetal intestinal dilatation become apparent.

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