

THE ACUTE ABDOMEN IN THE NEWBORN

Gustavo ROCHA, Carla COSTA, J. CORREIA-PINTO, Joaquim MONTEIRO,
Hercília GUIMARÃES

SUMMARY

Introduction: clinicians must be aware of the wide range of disorders causing acute abdomen in the newborn, a frequent condition that causes challenging problems from many aspects.

Aims: to evaluate our incidence of acute abdomen in the newborn, main aetiologies, antenatal diagnosis, clinical presentation and evolution.

Methods: a retrospective chart review at a tertiary centre neonatal intensive care unit, from 1997 to 2006.

Results: 233 (4.9%) out of 4743 newborns had acute abdomen. Conditions causing abdominal distension were the most frequent (39.5%), followed by conditions causing peritonitis (33.9%), abdominal wall anomalies (23.6%), functional obstructions (1.7%), and bleeding disorders (1.3%); 158 (67.8%) patients underwent surgical intervention, and 39 (16.7%) were deceased. Antenatal diagnosis rate was 50%.

Conclusions: some conditions associated to acute abdomen in the newborn are obvious, but others are rare and provide challenging problems in respect to diagnosis and treatment. Antenatal diagnosis, early recognition and timely transfer for surgery may avoid deterioration and loss of functioning bowel.

RESUMO

ABDÓMEN AGUDO NO RECÉM-NASCIDO

Introdução: o abdómen agudo é uma condição relativamente frequente no recém-nascido, muitas vezes coloca problemas no diagnóstico e tratamento, pelo que os clínicos devem estar familiarizados com o grande número de etiologias possíveis.

Objectivos: avaliar a nossa incidência de abdómen agudo no recém-nascido, principais causas, existência de diagnóstico pré-natal, apresentação clínica e evolução.

Métodos: estudo retrospectivo numa unidade de cuidados intensivos neonatais de um centro terciário, de 1997 a 2006.

Resultados: em 4743 recém-nascidos, 233 (4,9%) apresentaram abdómen agudo. As condições associadas a distensão abdominal foram as mais frequentes (39,5%), seguindo-se as condições associadas a peritonite (33,9%), anomalias da parede abdominal (23,6%), obstruções funcionais (1,7%) e associadas a sangramento (1,3%); 158 (67,8%) foram submetidos a intervenção cirúrgica e 39 (16,7%) faleceram. A taxa de diagnóstico pré-natal foi de 50%.

Conclusões: algumas condições associadas a abdómen agudo no recém-nascido são óbvias, no entanto, outras são raras e frequentemente colocam problemas de diagnóstico e tratamento. O diagnóstico pré-natal, o reconhecimento e transferência atempados para um centro com cirurgia pediátrica são importantes para evitar o agravamento clínico e perda de intestino funcional.

G.R., C.S., J.C-P, J.M., H.G.:
Serviços de Neonatologia e Cirurgia Pediátrica. Departamento de Pediatria. Hospital de São João. Porto
J.C-P.: Escola de Ciências da Saúde da Universidade do Minho. Braga

© 2009 CELOM

INTRODUCTION

The term *acute abdomen* stands for a group of abdominal symptoms which rapidly get worse and therefore require immediate treatment¹. It usually presents with acute peritonitis, abdominal distension, a tumour, bleeding and, in the newborn, with abdominal wall abnormalities².

The acute abdomen in the newborn provides challenging problems from many aspects, not only with regard to diagnosis, resuscitation and treatment, but also with prenatal management. Most conditions are uncommon and treatment in especially dedicated centres enables concentration of appropriate resources and expertise. Co-morbidity is common, particularly in the preterm of low birth weight. A multi-disciplinary team of surgeons, anaesthesiologists, neonatologists, radiologists, cardiologists, obstetricians, nurses, physiotherapists and other health professionals will provide the best outcome².

This study was conducted in order to evaluate our incidence of acute abdomen, as well as the main causes, clinical presentation, treatment and outcome of the affected newborns.

MATERIAL AND METHODS

We included in this study all newborns admitted to the neonatal intensive care unit of Hospital de São João, a tertiary medical centre from the north of Portugal, between 1997 and 2006. All newborns who had, at discharge, the diagnosis of any condition associated to acute abdomen were identified. All of the data were sought from medical records. Those with abdominal symptoms which rapidly get worse and therefore require immediate treatment¹ were considered to have had acute abdomen. All clinical, abdominal roentgenograms, laboratory, pathological and autopsy data were analyzed.

We have categorized five groups of clinical conditions: (1) conditions causing peritonitis; (2)

conditions causing abdominal distension; (3) functional obstruction; (4) abdominal wall anomalies; (5) bleeding conditions.

Gestational age was assessed by menstrual age, ultrasound examination or the New Ballard Score³.

RESULTS

A total of 4743 newborns were admitted to the intensive care unit between 1997 and 2006. Of these, 233 (4.9%) had acute abdomen, M = 137 (58.7%)/F = 96 (41.2%), median birth weight 2590 g (535-446), median gestational age 37 weeks (22-41), preterm 110 (47.2%). The acute abdomen was the reason of admission in 149 (63.9%) cases. The annual incidence ranged from 2.8% in 1999 to 8.4% in 2006.

The most common conditions were those associated to abdominal distension (39.5%), table 1.

Table 1 – Clinical conditions associated to acute abdomen (n = 233)

Diagnostic Category	Number of Cases (%)
Conditions causing peritonitis	79 (33.9)
Necrotizing enterocolitis	70 (30) [†23]
Focal small bowel perforation	2 (0.8)
Ileus perforation secondary to strangulated hernia	1 (0.4)
Incarcerated inguinal hernia	1 (0.4)
Meconial peritonitis	2 (0.8) [†1]
Focal perforation of the colon	1 (0.4)
Focal perforation of the rectum	1 (0.4)
Peritonitis secondary to gastric content extravasation from gastrostomy	1 (0.4)
Conditions causing abdominal distension	92 (39.5)
Duodenal atresia	15 (6.4)
Duodenal stenosis	10 (4.3) [†1]
Malrotation of the midgut	5 (2.1)
Volvulus of the midgut	5 (2.1)
Atresia of the midgut	20 (8.6) [†2]
Duplication of the sigmoid	1 (0.4)
Meckel's diverticulum	2 (0.8)
Meconium ileus	11 (4.7)
Hirschsprung's disease	11 (4.7)
Atresia of the colon	2 (0.8)
Anorectal anomalies	7 (3)
Meconium plug syndrome	1 (0.4)
Colon stenosis secondary to necrotizing enterocolitis	1 (0.4)
Functional obstructions	4 (1.7)
Electrolyte disturbances	2 (0.8)
Sepsis	1 (0.4)
Mesenteric cyst	1 (0.4)
Abdominal wall anomalies	55 (23.6)
Gastroschisis	29 (12.4) [†2]
Omphalocele	26 (11.1) [†8]
Bleeding conditions	3 (1.3)
Laceration of the liver	1 (0.4) [†1]
Laceration of the spleen	2 (0.8) [†1]

† – deceased because of the condition

Table 2 – Antenatal diagnosis and delivery

Condition	Antenatal Diagnosis n (%)	Oligohydramnios n (%)	Polyhydramnios n (%)	Gestational Age of Antenatal Diagnosis Median (min-max)	Mode of Delivery
Meconial Peritonitis (n = 2)	1 (50)	0	0	20	C = 1
Duodenal Atresia (n = 15)	8 (53)	1 (6.6)	2 (13.3)	31 (25-37)	C = 5 V = 10
Duodenal Stenosis (n = 10)	4 (40)	0	0	28 (21-35)	C = 2 V = 8
Intestinal Malrotation (n = 5)	1 (20)	0	0	32	C = 1 V = 4
Atresia of the Midgut (n = 20)	8 (40)	0	2 (10)	29 (24-33)	C = 6 V = 14
Intestinal Intraluminal Obstruction (n = 11)	3 (27)	0	3 (27.2)	34 (33-35)	C = 6 V = 5
Mesenteric Cyst (n = 1)	1 (100)	0	0	32	C = 1
Gastroschisis (n = 29)	16 (55)	1 (3.4)	1 (3.4)	28 (12-36)	C = 26 V = 3
Omphalocele (n = 26)	17 (65)	1 (3.8)	1 (3.8)	28 (12-36)	C = 6 V = 20

C – C-section; V – vaginal delivery

There was antenatal diagnosis in eight (53%) cases of duodenal atresia, four (40%) cases of duodenal stenosis and in eight (40%) cases of midgut atresia, table 2. Duodenal atresia was associated to congenital heart disease in four (27%) cases, to Down's syndrome in three (20%) cases, and to ambiguous genitalia in one (7%) case. Duodenal stenosis was associated to Down's syndrome in five (50%) cases, congenital heart disease in two (20%), and three (33%) cases of duodenal stenosis were due to anelar pancreas. A male newborn (gestational age 35 weeks/birthweight 1000 g) with duodenal stenosis due to anelar pancreas was deceased because of a fulminant sepsis after surgical treatment. Two preterm neonates with midgut atresia (a male 34 weeks gestational age/2160 g birthweight with an unclassified dysmorphic syndrome; a female 26 weeks gestational age/600g birthweight) were deceased in the post-operative period with infectious complications.

Three neonates with meconium ileus were affected by cystic fibrosis ($\Delta F508$ (exon 10)/A561 E (exon 12); N 1303k-N 1303k (homozygotic); and Del. AA2183 (exon 13)/683-E).

A neonate with Down's syndrome, including a congenital heart disease, was also affected by Hirschsprung's disease. A neonate with Hirschsprung's disease was submitted to colostomy.

The most frequent condition associated to peritonitis was necrotizing enterocolitis, representing 30% of the cases of acute abdomen in this study. It occurred mainly in the preterm neonate (52/110, 47.2%), and in very low birth weight infant (< 1500 g) (40/56, 71.4%), table 3. In four very tiny neonates [birthweight 635 g (542-720)/gestational age 25 weeks (22-26)] abdominal drainage was unsuccessfully used along with medical treatment. Twenty three patients with necrotizing enterocolitis

were deceased [birthweight 890 g (535-3868)/gestational age 26 weeks (22-38)], six with small bowel syndrome.

Table 3 – Epidemiological and clinical characteristics of the patients presenting with necrotizing enterocolitis (n = 70).

Characteristics	
Male/Female n (%)	40(57)/30 (43)
Gestational Age (weeks) (median, min-max)	32 (22-41)
Preterm n (%)	52 (74)
Birth Weight (g) (median, min-max)	1280 (535-4190)
< 1500 g n (%)	40 (57)
Intra-Uterine Growth Retardation n (%)	16 (23)
Diagnosis of Admission n (%)	24 (34)
Medical Treatment n (%)	49 (70)
Abdominal Drainage n (%)	4 (6)
Surgical Treatment n (%)	17 (24)
Small Bowel Syndrome n (%)	11 (16)
Deceased n (%)	23 (33)
Days of total parenteral nutrition (median, min-max)*	17 (1-129)
Day of total enteric nutrition (median, min-max)*	19 (6-95)
Hospitalization (days) (median, min-max)	26 (1-129)

* deceased excluded

Focal small bowel perforation occurred in two preterm male neonates (gestational age 32 weeks/birthweight 1450 g; gestational age 25 weeks/birthweight 840 g) in days three and 12 of life, respectively. The clinical evolution was favourable after surgery. All the other cases associated to peritonitis underwent surgical treatment. A male neonate (gestational age 37 weeks/birthweight 1650 g) affected by a chromosomal anomaly [46,XY inv(9)(p11,q13)] was deceased two days after surgery with an overwhelming sepsis.

A functional obstruction was diagnosed in two severely ill neonates with important electrolytic disturbances (including hypokalemia) and hypotension, and in another neonate with severe sepsis. All had favourable clinical evolution with medical therapy. Another case of obstruction occurred in a neonate with a mesenteric cyst of 11 cm, successfully extracted.

Gastroschisis and omphaloceles were frequent (23.6%)

in this study and were associated to mortality in 10 (4.3%) cases, table 4. Antenatal diagnosis was performed in the majority (60%) of the cases, at a median age of 28 weeks of gestation (12-36), for both pathologies.

Three cases of acute abdomen were associated with bleeding secondary to spleen (two cases) and liver (one case) traumatic injury. Two neonates were born after emergent C-section for abruptio placentae. One had a favourable evolution after splenectomy, the other deceased after unsuccessful hepatic packing. The third neonate, born after a traumatic vaginal delivery, was deceased with extensive bleeding fractures of the spleen.

In this study, surgical treatment was needed in 158 (67.8%) of the cases. Thirty nine (16.7%) of the newborns were deceased because of the condition that the acute abdomen or of a post-surgical complication, mainly of infectious aetiology.

Table 4 – Epidemiological and clinical characteristics of the patients presenting with abdominal wall anomalies (n = 55).

Characteristics	Gastroschisis (n = 29)	Omphalocele (n = 26)
Male/female n (%)	14 (48)/15 (52)	14 (54)/12 (46)
Gestational age (weeks) (median, min-max)	37 (33-39)	38 (30-40)
Preterm n (%)	11 (38)	9 (35)
Birth weight (g) (median, min-max)	2500 (2010-3550)	2795 (1030-4175)
Antenatal diagnosis n (%)	26 (90)	17 (65)
Associated anomalies	Ventricular septal defect = 1	Trisomy 13 = 1 Cloacal exstrophy = 1 Congenital heart defect = 4 Exstrophy of the bladder = 1 Persistence of omphalomesenteric duct = 1 Pierre-Robin syndrome with congenital heart defect = 1 Diaphragmatic eventration = 1 Ectopia cordis = 1
Surgical treatment	Primary closure = 25 Silastic silo = 4	Manual reduction = 2 Primary closure = 22 Silastic silo = 2
Deceased n (%)	2 (7)	8 (31)
Days of total parenteral nutrition (median, min-max)*	12 (5-100)	4 (1-227)
Day of total enteric nutrition (median, min-max)*	13 (8-101)	8 (4-58)
Hospitalization (days) (median, min-max)	19 (1-110)	10 (1-317)

* deceased excluded

DISCUSSION

There are many clinical conditions associated to acute abdomen in the newborn. Antenatal diagnosis enables fetal intervention and delivery at a centre where neonatal surgical facilities are available, reducing the risks of transfer and unnecessary separation from the mother⁴⁻⁶.

In this study we verified that antenatal diagnosis can still be improved, since about 50% (60/ 119) of detectable conditions were not diagnosed by fetal sonography.

Conditions causing peritonitis

The most common cause of peritonitis in the absence of obstruction is necrotizing enterocolitis. The epidemiology is changing⁷⁻⁹. This can be explained by the changing population and prevalence of risk factors with advances in obstetrical and neonatal care. Earlier reports included a higher proportion of more mature infants following severe perinatal hypoxia. Today, in the very low birth weight infant, necrotizing enterocolitis usually presents latter suggesting a more complex aetiology^{8,10,11}. Peritoneal drainage at the bedside under local anaesthetic will often improve the condition of the patient and in some patients might even avert the need for laparotomy^{12,13}. Mortality remains high but long term outcome for survivors is good in terms of surgical disease, although many have serious medical comorbidity^{14,15}. Prevention is better than cure and many strategies have been studied to reduce the incidence¹⁶.

In this study necrotizing enterocolitis was a frequent cause of acute abdomen, mostly in the preterm neonate, pointing to a multifactorial aetiology where immaturity plays a significant role. Mortality was high, according to that described in literature.

Focal small bowel perforation, usually included in studies of necrotizing enterocolitis, is likely to be a different disease of the preterm neonate. Presentation is often late and usually accompanied by less severe acute inflammatory changes and toxic effects¹⁷. Many infants do very well with localized resection and primary anastomosis, as did the two patients in this study.

Other conditions causing peritonitis are rare in the newborn. Primary peritonitis is rare and remains a diagnosis of exclusion².

Conditions causing abdominal distension

Prenatally, high gastrointestinal obstruction can give rise to polyhydramnios⁴, and in some, the dilated upper pouch is detectable by sonography. Bile stained vomiting is relatively frequent in the newborn, and it may be a sign of intestinal obstruction¹⁸.

Abdominal distension can be caused by mechanical or functional intestinal obstruction, ascites or abdominal masses. Gaseous abdominal distension is also common in the preterm with nasal continuous positive airway pressure².

Duodenal atresia presents in early life with vomiting, often without significant distension. It can be associated to other congenital anomalies and to trisomy 21 in 30-60% of the cases¹⁹. In the absence of associated anomalies the long-term outlook is excellent. In this study, the association to congenital heart disease was high (27%) and the outlook was good in all cases.

In duodenal stenosis, duodenal emptying may be slow for several days, and the association to other anomalies is not uncommon. In this study, the association of duodenal stenosis to Down's syndrome was high (50%).

Malrotation of the midgut results from a failure of rotation and peritoneal fixation when the intestines return from the physiologic hernia to the abdominal cavity at about the tenth week of intrauterine life²⁰. Volvulus of the entire midgut is a serious complication that manifests often in the first week of life²¹. In this study malrotation and midgut volvulus represented 11% of the cases presenting with obstruction, all with good outcome.

Midgut atresias occur mainly in the small bowel, but about 10% involve the colon, like in this study. Some, particularly multiple atresias, are familial and have a more complex aetiology²². The possibility of underlying cystic fibrosis or Hirschprung's disease should always be considered²³.

Duplications are tubular or cystic accessory enteric formations^{24,25}. In this study only one case of sigmoid duplication was diagnosed, and obstruction resulted from lumen compression.

Meckel's diverticulum is a vitello-intestinal duct remnant, located in the distal ileum. Only about 10% cause problems. Approximately one third present with bleeding from peptic ulceration, one third with obstruction – as a result of a volvulus, band or intussusception – and the remaining third with inflammation or abdominal wall fistula²⁶. In this study, intussusception was the cause of obstruction.

Meconium ileus is the most common form of intraluminal obstruction, and in this study it was associated to cystic fibrosis in three cases.

Hirschprung's disease and neuronal intestinal dysplasias constitute a group of conditions that usually cause functional obstruction as a result of abnormal bowel motility and peristalsis. Hirschprung's disease is usually an isolated condition but genetic abnormalities have been

identified²⁷. In this study a case occurred in association with trisomy 21. Most infants can be managed conservatively. Ileostomy or colostomy is still a useful initial treatment when there are complications, such as poorly controlled enterocolitis or long-segment disease involving all the colon or small bowel. In our study a patient with long-segment disease underwent colostomy.

Other condition that can mimic Hirschprung's disease is meconium plug syndrome that is more common in patients born from mothers with diabetes mellitus. This usually run a more benign course after decompression but underlying abnormalities, particularly cystic fibrosis and Hirschprung's disease, should be excluded.

Anorectal anomalies are a not infrequent cause of obstruction and can easily be missed if patency of the anus is not tested. In this study all cases, except one that was managed with dilatations after a cutaneous fistula had manifested, underwent colostomy.

Functional obstructions

Pseudo-obstruction can occur and lead to perforation of the large bowel or caecum. This usually occurs in severely ill patients. Many factors contribute, including electrolyte disturbances, gut dysmotility, hypoperfusion, ileus associated to infection, and drugs⁴. A intra-abdominal mass or tumour can compress the digestive tract and be a cause also of obstruction.

Abdominal wall anomalies

Gastroschisis and omphalocele are the most common abdominal wall anomalies in the newborn and are usually diagnosed antenatally^{28,29}. In this study, although the majority of newborns have had antenatal diagnosis, it can still be improved.

Significant associated congenital anomalies occur in infants with omphalocele. In this study an association occurred in 42% of the affected patients.

Bleeding conditions

They are rare in the neonate and usually related to traumatic injury, vascular abnormalities or coagulation disorders. They can be life threatening especially when massive bleeding follows traumatic, usually birth related, lacerations of liver and/or spleen.

CONCLUSIONS

Acute abdomen occurred in 4.9% of our population of newborns and was associated to a wide range of disorders. Some conditions were obvious but others are rare and

provide challenging problems in regard to diagnosis and treatment. Antenatal diagnosis of congenital anomalies as gastroschisis and omphalocele may still be improved and early recognition and timely transfer for surgery may avoid deterioration and loss of functioning bowel.

Conflito de interesses:

Os autores declaram não ter nenhum conflito de interesses relativamente ao presente artigo.

Fontes de financiamento:

Não existiram fontes externas de financiamento para a realização deste artigo.

REFERENCES

1. HOFMANN-VON KAP-HERR S: The concept of the acute abdomen. *Langenbecks Arch Chir Suppl Kongressbd* 1991;1:113-5
2. DE LA HUNT MN: The acute abdomen in the newborn. *Semin Fetal Neonatal Med* 2006;11:191-7
3. BALLARD JL, KHOURY JC, WEDIG K, WANG L, EILERS-WALSMAN BL, LIPP R: New Ballard Score, expanded to include extremely premature infants. *J Pediatr* 1991;119:417-423
4. LLOYD JR, CLATWORTHY JR HW: Hydramnios as an aid to the early diagnosis of congenital obstruction of the alimentary tract: a study of the maternal and fetal factors. *Pediatrics* 1958; 21:903-9
5. HANCOCK BJ, WISEMAN NE: Congenital duodenal obstruction: the impact of antenatal diagnosis. *J Pediatr Surg* 1989; 24:1027-31
6. BASU R, BURGE DM: The effect of antenatal diagnosis on the management of small bowel atresia. *Pediatr Surg Int* 2004;20:177-9
7. POKORNY WJ, GARCIA-PRATS JA, BARRY YN: Necrotizing enterocolitis: incidence, operative care, and outcome. *J Pediatr Surg* 1986;21:1149-54
8. BLAKELY ML, LALLY KP, MCDONALD S et al: Postoperative outcomes of extremely low birth-weight infants with necrotizing enterocolitis or isolated intestinal perforation. A prospective cohort study by the NICHD Neonatal Research Network. *Ann Surg* 2005;241:984-9
9. FLEET MS, DE LA HUNT MN: The changing face of necrotizing enterocolitis. *Proceedings of the British Association of Paediatric Surgeons 40th Annual Congress* 1993
10. REES CM, HALL NJ, EATON S, PIERRO A: Surgical strategies for necrotizing enterocolitis: a survey of practice in the United Kingdom. *Arch Dis Child Fetal Neonatal Ed* 2005;90:F152-5
11. KOSLOSKE AM: Indications for operation in necrotizing enterocolitis revisited. *J Pediatr Surg* 1994;29:663-6
12. EIN SH, SHANDLING B, WESSON D, FILLER RM: A 13-year experience with peritoneal drainage under local anaesthesia for necrotizing enterocolitis perforation. *J Pediatr Surg* 1990;25:1034-7
13. MOSS RL, DIMMIT RA, HENRY MC, GERAGHTY N, EFRON B: A meta-analysis of peritoneal drainage versus laparotomy for perforated necrotizing enterocolitis. *J Pediatr Surg* 2001;36:1210-3
14. RICKETTS RR, JERLES ML: neonatal necrotizing enterocolitis: experience with 100 consecutive surgical patients. *World J Surg* 1990;14:600-5
15. HINTZ SR, KENDRICK DE, STOLL BJ et al: Neurodevelopmental and growth outcomes of extremely low birth weight

infants after necrotizing enterocolitis. *Pediatrics* 2005;115:696-703

16. REBER KM, NANKERVIS CA: Necrotizing enterocolitis: preventive strategies. *Clin Perinatol* 2004;31:157-167

17. OKUYAMA H, KUBOTA A, OUE T, KURODA S, IKEGAMI R, KAMIYAMA M: A comparison of the clinical presentation and outcome of focal intestinal perforation and necrotizing enterocolitis in very low birth weight neonates. *Pediatr Surg Int* 2002; 18:704-6

18. GODBOLE P, STRINGER MD: Bilious vomiting in the newborn: how often is it pathologic? *J Pediatr Surg* 2002;37:901-911

19. AKHTAR J, GUINEY EJ: Congenital duodenal obstruction. *Br J Surg* 1992;79:133-5

20. DOTT NM: Anomalies of intestinal rotation: their embryology and surgical aspects with report of 5 cases. *Br J Surg* 1923; 11:251-286

21. POWELL DM, OTHERSEN HB, SMITH CD: Malrotation of the intestines in children: the effect of age on presentation and therapy. *J Pediatr Surg* 1989;24:777-780

22. BILODEAU A, PRASIL P, CLOUTIER R et al: Hereditary

multiple intestinal atresia: thirty years later. *J Pediatr Surg* 2004; 39:726-730

23. KUMARAN N, SHANKAR KR, LLOYD DA, LOSTY PD: Trends in the management and outcome of jejuno-ileal atresia. *Eur J Pediatr Surg* 2002;12:63-167

24. FORSHALL I: Duplication of the intestinal tract. *Postgrad Med J* 1961;37:570-589

25. CORREIA-PINTO J, TAVARES ML, MONTEIRO J et al: Prenatal diagnosis of abdominal enteric duplications. *Prenat Diagn* 2000;20:163-7

26. MACKEY WC, DINNEN P: A fifty year experience with Meckel's diverticulum. *Surg Gynecol Obstet* 1983;156:56-64

27. PURI P, SHINKAI T: Pathogenesis of Hirschprung's disease and its variants: recent progress. *Semin Pediatr Surg* 2004;13:18-24

28. CORREIA-PINTO J, TAVARES ML, BAPTISTA MJ et al: Meconium dependence of bowel damage in gastroschisis. *J Pediatr Surg* 2002;37:31-5

29. CORREIA-PINTO J, ESTEVÃO-COSTA J: Prolonged intestinal exposure to amniotic fluid does not result in peel formation in gastroschisis. *J Pediatr Surg* 1999;34:975-6



Hospital São João, Porto