

## Basic Study

**Predictive factors at birth of the severity of gastroschisis**

Anthony S de Buys Roessingh, Amélie Damphousse, Pierluigi Ballabeni, Josée Dubois, Sarah Bouchard

Anthony S de Buys Roessingh, Sarah Bouchard, Department of Pediatric Surgery, University Hospital, Hôpital Sainte-Justine, H3T1C5 Montréal, Canada

Amélie Damphousse, Josée Dubois, Department of Radiology, University Hospital, Hôpital Sainte-Justine, H3T1C5 Montréal, Canada

Anthony S de Buys Roessingh, Pierluigi Ballabeni, Department of Biostatistic, Centre Hospitalier Universitaire Vaudois, CH -1011 Lausanne, Switzerland

**Author contributions:** de Buys Roessingh AS and Bouchard S wrote the paper; Damphousse A and Dubois J did all the radiological lecture and Ballabeni P made the statistics.

**Institutional review board statement:** None.

**Informed consent statement:** All human studies have been reviewed by the appropriate ethics committee and all persons gave their informed consent prior to their inclusion in the study. Details that might disclose the identity of the subjects under study were omitted. Authors drew attention to the Code of Ethics of the World Medical Association (Declaration of Helsinki, 1964, as revised in 2004).

**Conflict-of-interest statement:** None of the authors have any financial or other relationships with other people or organizations that could inappropriately influence their work. There is no grant support for this research.

**Data sharing statement:** I declare data sharing statement in World Journal Gastrointestinal Pathophysiology; dataset available from the corresponding author at Email address or URL. Participants gave informed consent for data sharing.

**Open-Access:** This article is an open-access article which was selected by an in-house editor and fully peer-reviewed by external reviewers. It is distributed in accordance with the Creative Commons Attribution Non Commercial (CC BY-NC 4.0) license, which permits others to distribute, remix, adapt, build upon this work non-commercially, and license their derivative works on different terms, provided the original work is properly cited and the use is non-commercial. See: <http://creativecommons.org/licenses/by-nc/4.0/>

**Correspondence to:** Anthony S de Buys Roessingh, MD, PhD, Department of Biostatistic, Centre Hospitalier Universitaire Vaudois, Rue du Bugnon 21, CH -1011 Lausanne, Switzerland. [anthony.debuys-roessingh@Chuv.ch](mailto:anthony.debuys-roessingh@Chuv.ch)  
Telephone: +41-21-3143126  
Fax: +41-21-3143076

Received: June 5, 2015

Peer-review started: June 6, 2015

First decision: August 10, 2015

Revised: October 12, 2015

Accepted: October 23, 2015

Article in press: October 27, 2015

Published online: November 15, 2015

**Abstract**

**AIM:** To establish children born with gastroschisis (GS).

**METHODS:** We performed a retrospective study covering the period from January 2000 to December 2007. The following variables were analyzed for each child: Weight, sex, apgar, perforations, atresia, volvulus, bowel length, subjective description of perivisceritis, duration of parenteral nutrition, first nasogastric milk feeding, total milk feeding, necrotizing enterocolitis, average period of hospitalization and mortality. For statistical analysis, descriptive data are reported as mean  $\pm$  standard deviation and median (range). The non parametric test of Mann-Whitney was used. The threshold for statistical significance was  $P < 0.05$  (Two-Tailed).

**RESULTS:** Sixty-eight cases of GS were studied. We found nine cases of perforations, eight of volvulus, 12 of atresia and 49 children with subjective description of perivisceritis (72%). The mortality rate was 12% (eight deaths). Average duration of total parenteral nutrition was 56.7 d (8-950; median: 22), with five cases of necrotizing enterocolitis. Average length of hospitalization for 60 of our patients was 54.7 d (2-370;

median: 25.5). The presence of intestinal atresia was the only factor correlated with prolonged parenteral nutrition, delayed total oral milk feeding and longer hospitalization.

**CONCLUSION:** In our study, intestinal atresia was our predictive factor of the severity of GS.

**Key words:** Gastroschisis; Perivisceritis; Bowel atresia; Volvulus

© **The Author(s) 2015.** Published by Baishideng Publishing Group Inc. All rights reserved.

**Core tip:** Gastroschisis (GS) is defined as a full-thickness congenital abdominal wall defect usually situated on the right side of the umbilicus, with intestines protruding into the amniotic fluid without any protective membrane. The amniotic fluid creates an inflammation of the bowel wall, called perivisceritis. Associated with intestinal abnormalities are malrotation and a degree of short bowel: Volvulus, perforation and atresia may also be found. Our study shows that for babies born with GS, intestinal atresia is the only factor of prediction of the need for early and full enteral feeding, for its duration, and for the length of hospitalization.

de Buys Roessingh AS, Damphousse A, Ballabeni P, Dubois J, Bouchard S. Predictive factors at birth of the severity of gastroschisis. *World J Gastrointest Pathophysiol* 2015; 6(4): 228-234 Available from: URL: <http://www.wjgnet.com/2150-5330/full/v6/i4/228.htm> DOI: <http://dx.doi.org/10.4291/wjgp.v6.i4.228>

## INTRODUCTION

Gastroschisis (GS) is defined as a full-thickness congenital abdominal wall defect usually situated on the right side of the umbilicus, with intestines protruding into the amniotic fluid without any protective membrane<sup>[1]</sup>. The amniotic fluid creates an inflammation of the bowel wall, called perivisceritis<sup>[2]</sup>. Associated with intestinal abnormalities are malrotation and a degree of short bowel: Volvulus, perforation and atresia may also be found<sup>[3]</sup>. The degree of inflammation of the bowel and the presence of intestinal abnormalities are supposed to reflect the severity of the malformation, determining the surgical procedure and affecting the clinical outcome<sup>[4]</sup>. The duration of total parenteral nutrition (TPN), the timing of the introduction of normal feeding, the average length of hospitalization and in some cases death is all dependent on the severity of the malformation.

A prenatal ultrasound (US) diagnosis makes it possible to inform and prepare the parents, even though it remains difficult to predict whether the affected children will have a high or a low risk of abdominal complications, and how long they will need to be hospitalized<sup>[5,6]</sup>. If the defect closes by itself before birth, ischemia of the bowel may provoke total bowel necrosis. The prenatal closure

of the abdominal wall is therefore not always a good sign and may be associated with midgut infarction, short bowel or even vanishing midgut<sup>[7,8]</sup>.

Surgical management starts with the clinical observation of the anatomical anomalies accompanying GS and the search for associated malformations. Primary or delayed repair of the abdominal wall is then discussed<sup>[9]</sup>. Primary repair entails returning the bowel into its cavity soon after birth with a surgical procedure. But this primary abdominal closure is not always possible and depends on the age, weight and clinical condition of the baby, as well as on the amount of viscera protruding from the abdominal cavity: If it is too important, the immediate closure of the abdominal wall may cause excessive pressure. Delayed repair entails the use of a suspended pouch, called a "silo", containing the externalized bowel loops, with a gradual return of the bowel into the abdominal cavity and the closure of the abdominal wall a few days later<sup>[10]</sup>. The "silo" reduces the risk of excessive pressure, hypothermia and dehydration.

Volvulus, perforation and atresia may require resection, anastomosis or the creation of a stoma. The presence of these associated intestinal anomalies influences surgical and post surgical clinical management, for instance regarding the decision to perform a stoma or delay oral feeding because of the risk of necrotizing enterocolitis (NEC).

We performed a retrospective review of all children born with GS in our hospital over an eight-year period. The aim of this study is to establish which parameters such as bowel volvulus and/or atresia may help to predict the duration of TPN before the initiation of milk intake and eventual total oral milk feeding, and also the length of hospitalization.

## MATERIALS AND METHODS

We reviewed the files of all children admitted to our hospital with a diagnosis of GS from January 2000 to December 2007. Our records on the mothers indicate the duration of the pregnancy and the method of delivery. In our records on the children, we registered: weight, sex, apgar scores, acidosis at birth, length of intubation, bowel aspect, presence of necrosis, of perforation, of volvulus without necrosis, length of TPN, first milk feeding, total milk feeding, NEC, average length of hospitalization and, in some cases, time of death. Signed informed consent for this study was obtained from the appropriate local institutional Human Research Board.

Mothers were followed during the prenatal period by means of regular ultrasound exams. Bowel dilation, thickness of the abdominal wall, motility of the bowel, quantity of amniotic fluid, and fetal development were controlled during gestation. Vaginal delivery of the babies was proposed in our maternity department to avoid post-natal transfer. Scheduled vaginal provocation was planned, unless obstetric considerations led us to opt for caesarian delivery. When faced with fetal indications such

as worsening fetal status, progressive bowel dilation or loss of bowel movement, or with maternal problems, we performed an early delivery. Prenatal discussion sessions were organized between physicians of different specializations, and parents had the opportunity to meet with pediatric surgeons, neonatologists and geneticists.

The diagnosis of GS was confirmed immediately after delivery by neonatologists and pediatric surgeons. Primary surgery, if possible within the few hours following delivery, was our preferred mode of wall repair. After a rapid physical assessment, a nasogastric tube was placed in the stomach and intravenous fluid and antibiotics were given. The child was rapidly brought to the operating theater after routine resuscitation. The colon was irrigated with 5% n-acetyl cysteine diluted in warm normal saline solution to evacuate meconium. A Foley catheter was placed in the bladder for urinary drainage and for measurement of intra-abdominal pressure after closure of the abdominal wall.

Under total anaesthesia, the bowel loops were reduced gently into the abdominal cavity, fascia were separated from the skin and repaired with absorbable sutures. The abdominal wall was closed layer by layer. Urine production, absence of a compartment syndrome and good perfusion of both legs were controlled throughout. A broviac catheter was placed for TPN. The babies were then taken to the intensive care ward with intubation/ventilation, and given pain killers and drugs for wall relaxation for as long as necessary. Bladder pressure was measured continuously and maintained below 15 mmHg. Pulse oxymetry was measured on the feet. Nasogastric suction was maintained until bowel function returned. TPN was provided until adequate oral nutrition became possible.

Primary closure was not always indicated, and the decision not to close the abdominal wall was taken either during surgery, when the introduction of the bowel into the abdominal cavity induced a compartment syndrome, or if the baby could not be brought to the operating room because of his weight or the presence of associated malformations. In these cases, we used a protective "silo" to allow staged reduction of the bowel over a period of several days. After this period of progressive bowel reintegration into the abdominal cavity, the abdominal wall was closed layer by layer as described above.

Short bowel syndrome normally defines a functional state dependent on the degree to which the normal absorptive capacity of the small intestine is compromised<sup>[11]</sup>. In our paper, short bowel is defined according to either the length of small intestine present after abdominal closure, or the length left after intestinal resections in cases of intestinal atresia. We defined two categories: more than 100 cm or less than 50 cm.

Inflammation of the bowel was defined subjectively at birth and during surgery on the basis of the aspect of the bowel wall, the presence of large amount of fibrin, the abnormal thickness of the bowel wall and the absence of bowel movement or contraction after

stimulation. A less inflamed bowel was defined subjectively, on the basis of the presence of a small amount of fibrin, a practically normal appearance and thickness of the bowel wall, and bowel movement under stimulation.

Oral feeding was started as soon as possible through a gastric catheter. One milliliter per hour was given at the beginning, and this amount was gradually increased depending on the color and quantity of fluid aspiration, the abdominal distention and stools production. The amount needed for full feeding was determined by the weight of the baby.

For statistical analysis, descriptive data are reported as mean  $\pm$  standard deviation and median (range). The non parametric test of Mann-Whitney was used as the distribution of different variables was not always regular. The threshold for statistical significance was  $P < 0.05$  (Two-Tailed). All statistical analyses were performed by a biostatistician using the statistical software SAS for Windows (SAS release 8.2, 2002, Cupertino, California, United States).

## RESULTS

From January 2000 to December 2007, 72 babies were deemed eligible for the study. But four were immediately excluded because their records were incomplete. We therefore retained 68 cases of GS ( $n = 68$ ) for our study.

The mean age of the mothers was 23.1 years, with a range from 15 to 34 years. The mean delivery time was 35.6 wk (median: 36 wk), with vaginal delivery in 67% of cases. A diagnosis of intrauterine growth restriction was made in 22.4% ( $n = 15$ ) of cases, and confirmed at birth in all cases. No intrauterine deaths were reported for fetuses with intrauterine growth restriction. Oligohydramnios was observed in 24% ( $n = 16$ ) of mothers. No complications were reported during delivery. Average weight at birth was 2501 g and 53% were girls.

We found eight volvulus, 12 atresia, nine perforations and six stenosis, 39 bowels less than 100 cm and 22 less than 50 cm (Table 1). Subjective description has done by the surgeon of the presence of a perivisceritis were found in 49 children (72%). We used silos in 17 cases (25%), for an average period of 5.94 d. Average age at surgery was 2.45 d.

A post-natal mortality rate of 12% (8/68) was observed. Three of these babies (3/68) showed concomitant fetal abnormalities incompatible with life; three (3/68) died, respectively from short bowel syndrome requiring a bowel transplant, NEC with fistula, and after surgery for a chylothorax; and two more (2/68), who presented small bowel necroses requiring multiple surgery, also died shortly after birth.

Mean duration of TPN was 56.7 d (range: 8-950 d; median: 22 d) with five NEC; four newborns (6%) received TPN for less than ten days; 20 patients (29.9%) required TPN for 30 d or more, including nine patients ( $n = 9$ ) who presented no other complications besides GS. Of those 20 patients, eight ( $n = 8$ ) required multiple

**Table 1 Summary of clinical conditions for the 68 cases of gastroschisis**

	Mean	Median
Birth weight	2501 g	
Gestational age	35.6 wk	36 wk
Female sex	53%	
Mother's age	23 yr	
Timing of closure	2.5 d	
Silo	25% (17)	
Severely inflamed	72% (49)	
Perforations	13.2% (9)	
Volvulus	11.7% (8)	
Atresia	17.6% (12)	
Bowel less than 100 cm	66% (39)	
Short bowel less than 50 cm	30% (21)	
Stenosis	8.8% (6)	
NEC	7.35% (5)	
Mortality	11.7% (8)	
For 60 children (68 minus eight deaths)		
Intubation in days	8.5 d	5 d
1 <sup>st</sup> feeding	17.3 d	11 d
Timing of total milk feeding		
Days of TPN	56.7 d	22 d
Hospitalization	54.7 d	25.5 d

NEC: Necrotizing enterocolitis; TPN: Total parenteral nutrition.

surgery, six had atresia ( $n = 6$ ), three NEC ( $n = 3$ ), and three eventually died ( $n = 3$ ).

Mean duration of intubation was 8.47 d (median: 5 d). Mean duration of hospitalization was 54.7 d (2-370; median: 25.5) for our 60 cases of GS (Table 1). Twenty-six patients (23.9%) were hospitalized for 50 d or more, among whom nine required ( $n = 9$ ) multiple surgery, eight were cases of atresia ( $n = 8$ ), three of volvulus ( $n = 3$ ), three of NEC ( $n = 3$ ), and the three who eventually died ( $n = 3$ ).

The median period of time before initiation of nasogastric milk, the median duration of TPN, the median period of time until the start of total oral milk intake and the median duration of hospitalization are summarized in Table 2.

There were a total of 12 cases with intestinal atresia and 48 without atresia. The median period of time before initiation of nasogastric milk, the median duration of TPN, the median period of time until the start of total oral milk intake and the median duration of hospitalization are summarized in Table 2.

There were eight cases of volvulus and 52 children without volvulus. The median period of time before initiation of nasogastric milk, the median duration of TPN, the median period of time until the start of total oral milk intake and the median duration of hospitalization are summarized in Table 2.

## DISCUSSION

Our study shows that for babies born with GS, intestinal atresia is the only factor of prediction of the need for early and full enteral feeding, for its duration, and for the length of hospitalization.

The etiology of GS has not yet been ascertained, but low socioeconomic status, poor maternal education, drug abuse, in particular with cocaine, abuse of tobacco and alcohol, and young maternal age (less than 20 years old) are associated with GS<sup>[11-14]</sup>. Although the survival rate for babies born with GS has improved and is now practically 85%, short and long-term morbidity is still a serious problem. The mortality rate reported in recent literature varies greatly (2.4% to 11%)<sup>[15,16]</sup>, but the mortality rate reported in our study (11.7%) tallies with the values reported in most studies. Prematurity, intrauterine growth retardation and the presence of a congenital circulatory or pulmonary anomaly are external factors associated with a poorer outcome for children born with GS<sup>[3]</sup>. In our study, three babies (3/68) showed concomitant fetal abnormalities incompatible with life; finally, five babies died from GS and associated bowel complications (5/68, 7.3%).

Many authors have attempted to establish a prognosis of post-natal morbidity in cases of GS by studying various prenatal US findings, such as amniotic fluid volume, small bowel diameter, maximum bowel diameter, maximum thickness of the bowel wall, intrauterine growth restriction, Doppler velocimetry of the superior mesenteric artery, and the presence of other anomalies. Other studies have tried, based either on prenatal US or on bowel examination at birth, to establish a prognosis for the length of hospitalization, the duration of TPN or the timing of introduction of normal feeding. It appears, however, that antenatal screening cannot reliably predict morbidity, and the fact that no consensus emerges from the different studies is due mainly to the small sample size, but also to the difficulty in correlating imaging findings with clinical outcome. Some studies found a significant association between intra-abdominal bowel dilation and bowel atresia<sup>[5,17,18]</sup>. Japaraj *et al*<sup>[6]</sup> found that the occurrence of polyhydramnios was significantly associated with a higher rate of severe bowel complications such as atresia, perforation and necrosis. Intestinal atresia has been described as a significant risk factor of morbidity and mortality, due to the fact that the dilated bowel causes increased abdominal pressure during and after abdominal closure<sup>[15]</sup>. While, in both our groups (with atresia and without atresia), nasogastric milk feeding was initiated after an equal number of days following surgery (11 d and 10 d respectively), the median duration of TPN was clearly and significantly greater in the "atresia group", and the period of time before total milk feeding could be introduced was longer. The mortality rate in the "atresia group" was not higher, even though, in 12 cases, atresia was associated with three perforations and intestinal necrosis which required several surgical procedures. In one case, atresia was also associated with a chronic intestinal pseudo obstruction. The higher morbidity in this group also influenced the duration of hospitalization, which was definitely longer, due to surgical complications, prolonged TPN and delayed total milk feeding.

Short-term and long-term outcomes of GS are

**Table 2 Comparison in days between two groups of 60 children depending on the presence of atresia, volvulus and perivisceritis (68 minus eight deaths)**

	Atresia (n = 12)	Non atresia (n = 48)	Mann-Whitney test	Volvulus (n = 8)	No volvulus (n = 52)	Mann-Whitney test	Subjective perivisceritis (n = 49)	Subjective low perivisceritis (n = 19)	Mann-Whitney test
TPN duration	60	20	0.005	36	20	0.140	34.5	22	0.564
Start of total oral milk intake	10	11	0.809	11	7.5	0.212	11	10.5	0.569
Total oral milk intake	56	20	0.05	22	17	0.468	36	21	0.196
Mean hospitalization	64	24.5	0.021	48	25.5	0.309	39	25.5	0.505

TPN: Total parenteral nutrition.

well-known but difficult to predict. Many studies have demonstrated that the presence of a compromised bowel is associated with a significant increase in the number of surgical procedures, a longer period of full enteral feeding and a prolonged hospitalization<sup>[15,19]</sup>. Prolonged TPN, with its risk of sepsis, is directly related to intestinal recuperation, and the morbidity of GS is closely related to intestinal damage<sup>[20]</sup>. While gastrointestinal complications such as matting between the loops, malrotation, volvulus, perforations and atresia increase the complexity of early management<sup>[19,21]</sup>, later management may be complicated by the presence of problems of absorption, intestinal dysmotility, obstruction, NEC, infarction and stenosis<sup>[1,3,9]</sup>.

The pathogenesis of secondary bowel lesions is not fully understood, but both chemical and mechanical origins are concerned<sup>[2]</sup>. We know that prolonged contact with the amniotic fluid is deleterious for the bowel and may lead to inflammation of the bowel wall resulting in the production of a yellow fibrous tissue named perivisceritis<sup>[21,22]</sup>. This perivisceritis is accompanied by edema, cellular infiltration of epithelial cells and the presence of macrophages in the bowel wall. Specific therapeutic strategies, including amnio-exchange as a prenatal treatment, may be developed to prevent the resulting more serious bowel damage. Amnio-exchange has been tried for many years in some centers<sup>[22]</sup>. Amnio-infusion during pregnancy consists in replacing the amniotic fluid with a saline solution in order to reduce the inflammation of the bowel due to its contact with the amniotic fluid. In animal studies, amnio-exchange reduces the inflammation of the bowel wall by eliminating inflammatory compounds. However, since no prospective and randomized studies with human fetuses have yet been realized, we do not use this technique and none of our babies benefited from amnio-infusion. Inflammation of the bowel at birth, on the basis of the aspect of the bowel wall, the presence of large amount of fibrin, the abnormal thickness of the bowel wall and the absence of bowel movement or contraction after stimulation, does not help to predict the outcome of GS, and does not seem to correlate with the degree of bowel recuperation or bowel damage. The condition of the externalized bowel loops can be difficult to evaluate, and its appreciation is largely subjective and without

predictive value.

The recommended mode and timing of delivery remains a subject of debate<sup>[23-26]</sup>. Labor may be deleterious to the externalized bowel loops, and may entail the risk of membrane rupture and of infection. However, most authors found that caesarean delivery presented no significant benefit and did not improve the outcome of infants with GS. It is therefore reserved for obstetric indications or acute fetal emergencies often related to other organ failure. Preterm delivery, in order to limit the period of intrauterine damage of the bowel due to contact with the amniotic fluid, was of no benefit and did not lessen the morbidity of GS<sup>[25]</sup>.

Surgery is performed in our hospital under emergency conditions, in order to close the abdominal wall as quickly as possible<sup>[9,27]</sup>. We think that early repair leads to a lower incidence of perivisceritis<sup>[28]</sup>. Coughlin JP *et al.*<sup>[29]</sup> also observed an absence of inflammatory desquamation on the bowels of babies operated immediately after birth. Nevertheless, the surgical procedure may have to be delayed if further investigation for associated anomalies is required or if the child is too small for the operation. The surgical procedure may also have to be postponed if the intra-abdominal pressure during reintegration of the loops is too high (more than 20 mmHg) and would require high ventilation pressure, myorelaxant drugs and diuretics<sup>[30]</sup>. In these cases, abdominal closure is deferred, and the intestinal loops are protected with a silo during their progressive reintegration into the abdominal cavity<sup>[7]</sup>. The use of a suspended "silo" for a few days, allowing the gradual return of the viscera into the naturally growing abdominal cavity, makes it possible to close the abdominal wall without undue pressure and with a relatively low risk of intestinal damage.

All our patients required TPN for at least 10 d. In the course of prenatal counseling, parents should be made aware that their newborn will need TPN, and therefore the placement of a central venous line to provide adequate intake until oral nutrition is possible.

Our study also shows that a long period of hospitalization should be expected (mean hospitalization time of 54.7 d) and that, not surprisingly, a prolonged hospitalization is associated with a less favorable outcome. Parents should also be made aware that the length of the hospital stay will depend on how long the

bowel needs to rest and on the duration of total enteral feeding, two elements which depend primarily on clinical conditions.

## ACKNOWLEDGMENTS

The authors are grateful to Annette Wagnière for reviewing the English text.

## COMMENTS

### Background

The authors performed a retrospective study covering the period from January 2000 to December 2007.

### Research frontiers

The median duration of total parenteral nutrition (TPN) was clearly and significantly greater in the "atresia group", and the period of time before total milk feeding could be introduced was longer.

### Innovations and breakthroughs

Associated with intestinal abnormalities are malrotation and a degree of short bowel: Volvulus, perforation and atresia may also be found.

### Applications

This study also shows that a long period of hospitalization should be expected (mean hospitalization time of 54.7 d) and a prolonged hospitalization is associated with a less favorable outcome.

### Terminology

The duration of TPN or the timing of introduction of normal feeding.

### Peer-review

The authors showed that the predictive factor of babies with gastroschisis (GS) is intestinal atresia by analysing 60 babies with GS.

## REFERENCES

- Axt R, Quijano F, Boos R, Hendrik HJ, Jessberger HJ, Schwaiger C, Schmidt W. Omphalocele and gastroschisis: prenatal diagnosis and periparturient management. A case analysis of the years 1989-1997 at the Department of Obstetrics and Gynecology, University of Homburg/Saar. *Eur J Obstet Gynecol Reprod Biol* 1999; **87**: 47-54 [PMID: 10579616 DOI: 10.1016/S0301-2115(99)00078-0]
- Langer JC, Longaker MT, Crombleholme TM, Bond SJ, Finkbeiner WE, Rudolph CA, Verrier ED, Harrison MR. Etiology of intestinal damage in gastroschisis. I: Effects of amniotic fluid exposure and bowel constriction in a fetal lamb model. *J Pediatr Surg* 1989; **24**: 992-997 [PMID: 2530329 DOI: 10.1016/S0022-3468(89)80200-3]
- Lao OB, Larison C, Garrison MM, Waldhausen JH, Goldin AB. Outcomes in neonates with gastroschisis in U.S. children's hospitals. *Am J Perinatol* 2010; **27**: 97-101 [PMID: 19866404 DOI: 10.1055/s-0029-1241729]
- Snyder CL. Outcome analysis for gastroschisis. *J Pediatr Surg* 1999; **34**: 1253-1256 [PMID: 10466606 DOI: 10.1016/S0022-3468(99)90162-8]
- Bond SJ, Harrison MR, Filly RA, Callen PW, Anderson RA, Golbus MS. Severity of intestinal damage in gastroschisis: correlation with prenatal sonographic findings. *J Pediatr Surg* 1988; **23**: 520-525 [PMID: 2971103 DOI: 10.1016/S0022-3468(88)80360-9]
- Japaraj RP, Hockey R, Chan FY. Gastroschisis: can prenatal sonography predict neonatal outcome? *Ultrasound Obstet Gynecol* 2003; **21**: 329-333 [PMID: 12704738 DOI: 10.1002/uog.85]
- Houben C, Davenport M, Ade-Ajayi N, Flack N, Patel S. Closing gastroschisis: diagnosis, management, and outcomes. *J Pediatr Surg* 2009; **44**: 343-347 [PMID: 19231531 DOI: 10.1016/j.jpedsurg.2008.10.084]
- Vegunta RK, Wallace LJ, Leonardi MR, Gross TL, Renfroe Y, Marshall JS, Cohen HS, Hocker JR, Macwan KS, Clark SE, Ramiro S, Pearl RH. Perinatal management of gastroschisis: analysis of a newly established clinical pathway. *J Pediatr Surg* 2005; **40**: 528-534 [PMID: 15793730 DOI: 10.1016/j.jpedsurg.2004.11.037]
- Page R, Ferraro ZM, Moretti F, Fung KF. Gastroschisis: antenatal sonographic predictors of adverse neonatal outcome. *J Pregnancy* 2014; **2014**: 239406 [PMID: 25587450 DOI: 10.1155/2014/239406]
- Schwartz MZ, Tyson KR, Milliorn K, Lobe TE. Staged reduction using a Silastic sac is the treatment of choice for large congenital abdominal wall defects. *J Pediatr Surg* 1983; **18**: 713-719 [PMID: 6229620 DOI: 10.1016/S0022-3468(83)80010-4]
- Goldbaum G, Daling J, Milham S. Risk factors for gastroschisis. *Teratology* 1990; **42**: 397-403 [PMID: 2147789 DOI: 10.1002/tera.1420420408]
- Curry JJ, McKinney P, Thornton JG, Stringer MD. The aetiology of gastroschisis. *BJOG* 2000; **107**: 1339-1346 [PMID: 11117759 DOI: 10.1111/j.1471-0528.2000.tb11645.x]
- Loane M, Dolk H, Bradbury I. Increasing prevalence of gastroschisis in Europe 1980-2002: a phenomenon restricted to younger mothers? *Paediatr Perinat Epidemiol* 2007; **21**: 363-369 [PMID: 17564594 DOI: 10.1111/j.1365-3016.2007.00820.x]
- Zamakhshary M, Yanchar NL. Complicated gastroschisis and maternal smoking: a causal association? *Pediatr Surg Int* 2007; **23**: 841-844 [PMID: 17618440 DOI: 10.1007/s00383-007-1926-6]
- Driver CP, Bruce J, Bianchi A, Doig CM, Dickson AP, Bowen J. The contemporary outcome of gastroschisis. *J Pediatr Surg* 2000; **35**: 1719-1723 [PMID: 11101722 DOI: 10.1053/jpsu.2000.19221]
- Fratelli N, Papageorgiou AT, Bhide A, Sharma A, Okoye B, Thilaganathan B. Outcome of antenatally diagnosed abdominal wall defects. *Ultrasound Obstet Gynecol* 2007; **30**: 266-270 [PMID: 17674424 DOI: 10.1002/uog.4086]
- Heinig J, Müller V, Schmitz R, Lohse K, Klockenbusch W, Steinhilber J. Sonographic assessment of the extra-abdominal fetal small bowel in gastroschisis: a retrospective longitudinal study in relation to prenatal complications. *Prenat Diagn* 2008; **28**: 109-114 [PMID: 18186152 DOI: 10.1002/pd.1907]
- Piper HG, Jaksic T. The impact of prenatal bowel dilation on clinical outcomes in neonates with gastroschisis. *J Pediatr Surg* 2006; **41**: 897-900 [PMID: 16677878 DOI: 10.1016/j.jpedsurg.2006.01.005]
- Henrich K, Huemmer HP, Reingruber B, Weber PG. Gastroschisis and omphalocele: treatments and long-term outcomes. *Pediatr Surg Int* 2008; **24**: 167-173 [PMID: 17985136 DOI: 10.1007/s00383-007-2055-y]
- Carvalho NS, Helfer TM, Serni PO, Terasaka OA, Boute T, Araujo Júnior E, Nardoza LM, Moron AF, Rolo LC. Postnatal outcomes of infants with gastroschisis: a 5-year follow-up in a tertiary referral center in Brazil. *J Matern Fetal Neonatal Med* 2015; **1-5** [PMID: 25747953 DOI: 10.3109/14767058.2014.1002764]
- Goulet O, Ruemmele F, Lacaille F, Colomb V. Irreversible intestinal failure. *J Pediatr Gastroenterol Nutr* 2004; **38**: 250-269 [PMID: 15076623 DOI: 10.1097/00005176-200403000-00006]
- Luton D, de Lagausie P, Guibourdenche J, Oury J, Sibony O, Vuillard E, Boissinot C, Aigrain Y, Beauflis F, Navarro J, Blot P. Effect of amnioinfusion on the outcome of prenatally diagnosed gastroschisis. *Fetal Diagn Ther* 1999; **14**: 152-155 [PMID: 10364666 DOI: 10.1159/000020910]
- Ergün O, Barksdale E, Ergün FS, Prosen T, Qureshi FG, Reblock KR, Ford H, Hackam DJ. The timing of delivery of infants with gastroschisis influences outcome. *J Pediatr Surg* 2005; **40**: 424-428 [PMID: 15750942 DOI: 10.1016/j.jpedsurg.2004.10.013]
- Baud D, Lausman A, Alfaraj MA, Seaward G, Kingdom J, Windrim R, Langer JC, Kelly EN, Ryan G. Expectant management compared with elective delivery at 37 weeks for gastroschisis. *Obstet Gynecol* 2013; **121**: 990-998 [PMID: 23635735 DOI: 10.1097/AOG.0b013e31828ec299]
- Lausman AY, Langer JC, Tai M, Seaward PG, Windrim RC, Kelly EN, Ryan G. Gastroschisis: what is the average gestational age of spontaneous delivery? *J Pediatr Surg* 2007; **42**: 1816-1821 [PMID:

18022429 DOI: 10.1016/j.jpedsurg.2007.07.005]

- 26 **Logghe HL**, Mason GC, Thornton JG, Stringer MD. A randomized controlled trial of elective preterm delivery of fetuses with gastroschisis. *J Pediatr Surg* 2005; **40**: 1726-1731 [PMID: 16291160 DOI: 10.1016/j.jpedsurg.2005.07.047]
- 27 **Savoie KB**, Huang EY, Aziz SK, Blakely ML, Dassinger S, Dorale AR, Duggan EM, Harting MT, Markel TA, Moore-Olufemi SD, Shah SR, St Peter SD, Tsao K, Wyrick DL, Williams RF. Improving gastroschisis outcomes: does birth place matter? *J Pediatr Surg* 2014; **49**: 1771-1775 [PMID: 25487481 DOI: 10.1016/j.jpedsurg.2014.09.019]
- 28 **Blessed WB**, Coughlin JP, Johnson MP, Evans MI, Jewell MR, Goyert GL, Schwartz DB, Klein MD. Immediate delivery room repair of fetal abdominal wall defects. *Fetal Diagn Ther* 1993; **8**: 203-208 [PMID: 8240695 DOI: 10.1159/000263826]
- 29 **Coughlin JP**, Drucker DE, Jewell MR, Evans MJ, Klein MD. Delivery room repair of gastroschisis. *Surgery* 1993; **114**: 822-826; discussion 826-827 [PMID: 8211700]
- 30 **Kandasamy Y**, Whitehall J, Gill A, Stalewski H. Surgical management of gastroschisis in North Queensland from 1988 to 2007. *J Paediatr Child Health* 2010; **46**: 40-44 [PMID: 19943863 DOI: 10.1111/j.1440-1754.2009.01615.x]

**P- Reviewer:** Cordon JP, Germer CT, Woo SY **S- Editor:** Qiu S  
**L- Editor:** A **E- Editor:** Liu SQ





Published by **Baishideng Publishing Group Inc**

8226 Regency Drive, Pleasanton, CA 94588, USA

Telephone: +1-925-223-8242

Fax: +1-925-223-8243

E-mail: [bpgoffice@wjgnet.com](mailto:bpgoffice@wjgnet.com)

Help Desk: <http://www.wjgnet.com/esps/helpdesk.aspx>

<http://www.wjgnet.com>

