honeycomb. Microscopic hemangioma is composed of blood vessels sinusoidally enlarged, filled with blood. In the later stages of their evolution, there are signs of thrombosis, hemorrhage, fibrosis, calcification, etc.

Among the most serious complications are: a break with intraperitoneal bleeding or bleeding in liver parenchyma, or in the surrounding organs. The mortality in these complications reaches 60-83 %, but these complications appear in fewer than 10% of large hemangiomas of the liver. Hematobilia develops more often in malignant tumors, but also it is described in hemangiomas. In the literature, there are reports of malignant hemangioma of the liver with tumor thrombosis, suppuration and abscess formation. Giant liver tumors and tumors localized in the liver hilum may be accompanied by jaundice, signs of portal hypertension, intestinal obstruction.

Ultrasonography and computer tomography in hemangioma has a sensitivity of 60-80%. Typical ultrasound signs of hemangioma include hyperechoic formation, with uniform, clear edges.

On computer tomography examination, the typical signs of liver hemangioma are hypodense lesion, moving from the periphery to the center of contrast substance, complete opacification on delayed images, which is defined as isodense or hyperdense formation.

Liver scintigraphy indicates a defect of blood supply and determines the focus of greater than 2 cm, but does not specify the nature of these defects.

Nuclear magnetic resonance is an exam for lesions less than 2 cm, and for those who are in close proximity to the suprahepatic veins.

Surgical treatment remains the treatment of choice in some cases. Indications for surgery include the presence of clinical symptoms, changes in the size and nature of the tumor, rupture, subcapsular or intratumoral hemorrhage, central necrosis, the presence of haematological disorders or diagnostic uncertainty.

Conclusions:

1. The absence of pathognomonic symptoms and poor clinical manifestations of hepatic hemangioma does not allow accurate and early diagnosis.
2. The indications for surgical correction is the presence of clinical and laboratory signs, complications and diagnostic uncertainty.
3. Liver hemangiomas do not have specific scintigraphic picture.

REFERENCES

1. Angelescu Nicolae "Tratat de patologie chiricală", Bucureşti, 2003
2. Алимпиев С. В. "Современные тенденции хирургической тактики при гемангиомах печени"//Анналы хирургической гепатологии. 1999. т. 4. №1 с. 97-103
3. Ашкрафт К.У., Холдер Т.М. . „Детская хирургия”, Санкт-Петербург, 1997;
4. Дурнов Л.А.,Голдобенко Г. В. „Детская онкология”, Москва, 2002, стр. 540;
5. Шерлок Ш., ДулиДж. "Заболевания печени и жёлчных путей", М, 1999.

Babuci S., Malai A., Negru I.

CONTEMPORARY CONCEPTS IN LAPAROSCHIZIS

(Literature review)

State University of Medicine and Pharmacy „Nicolae Testemiţanu”
„Surgical pediatric infections” Laboratory
MSPI Institute of Mother and Child Care
Laboratory of surgical correction of congenital defects in children

Summary

This article examines contemporary issues of etiopathogenesis, clinical picture, diagnosis and treatment methods in laparoschizis which are reflected in literature. Despite the progress of surgical correction strategies, principles of surgical treatment of congenital abdominal wall defects are still the same: to reduce the eviscerated abdominal cavity organs in safe conditions, closure of the abdominal wall defect with an acceptable cosmetic appearance, identification and treatment of associated malformations, establishing an independent nutrition support until switching to a complete enteral nutrition and detection and adequate treatment of abdominal, intestinal complications or wound. The prognosis of this malformation is severe.

Laparoschizis is a congenital structural defect of the anterior abdominal wall, located adjacent to and usually on the right of the insertion of the umbilical cord, through which abdominal viscera herniate. The abdominal wall defect is relatively small (less than 5 cm in diameter) compared to the size of eviscerated organs and it is shaped as a paraumbilical slot immediately adjacent to the umbilicus or separated from it by a leather band [13]. Although in most cases the parietal defect is located on the right side of the umbilicus [14], the abdominal parietal defect can rarely be found on the left side of the cord inserted normally [74, 90]. Eviscerated organs are not covered by any embryonic membrane or intrauterine sac [44],

being exposed to amniotic fluid, which determines the development of morphopathological changes (edema, shortening of intestinal loops and so on).

The Babylonian records certified these malformations in 1056, but it is considered that for the first time gastroschisis was described by Lycosthenes in 1557 [79]. A number of fetal specimens of different gestational ages collected by John Hunter (1728-1793) are exposed in the Hunterian Museum of the Royal College of Surgeons of England, including a child aged 97 days of gestation with gastroschisis [70]. There are communications that support that the first description of gastroschisis belongs to J. Calder (1733) [13, 15, 70]. In 1775 Hei W. described a newborn whose "small intestine was located on the abdomen, having no sac." He immediately reduced the bowel into the abdominal cavity, but the child died within a few hours. The first cases of survival were reported by Vissick and Fear (1878) (1873), that in retrospect was a rupture of the omphalocele minor [70].

The term "gastroschisis" was first used by Taruffli in 1894 [49, 70] and comes from the Greek roots, meaning " split stomach ", though the correct term would be "laparoschisis" (split abdomen) [8].

The incidence of gastroschisis varies from 0.5 to 7.0 cases per 10,000 births, with an average of 1/2700 births [79, 89]. Incidence of associated primary congenital malformations in patients with gastroschisis is considered low, representing about 17% - 27% [71, 87]. Intestinal atresia, cryptorchidism and persistence of fetal circulation are considered secondary deformities. It is to be noted that intestinal atresia can complicate the development of gastroschisis in 10-20% of cases. Multiple congenital malformations are detected relatively rarely in case of gastroschisis. In 10-20% of cases they are associated with bowel malformations [34], urinary tract malformations - 6%, heart defects - 1%. It was found that fetuses with gastroschisis have an increased risk of prematurity (22-40%), oligohydramnios (36%), intrauterine retardation (38-77%) or abortion (7%) [3, 63, 69].

Cardiac malformations associated with gastroschisis include: septal defects, Fallot tetralogy, Ebstein anomaly, coarctation of the aorta, the major artery transposition etc. [41]. There were also reported concomitant facial, musculoskeletal malformations [47], associated chromosomal abnormalities, including trisomies and Turner syndrome [28, 47, 88]. Other reported associations include: neural tube defects [35], hydrocephalus [41, 72], anorectal malformations [31, 47, 57].

Embryology. In the 3th week of development embryo represents a tri-laminar disc consisting of dorsal ectoderm, ventral endoderm, mesoderm which lies between these laminas and the vitelline sac which is much larger than the embryo. In this period neural folds begin to form from ectoderm, which when growing, close to form the neural tube. At the same time, the lateral sides of the disc start to fold ventrally to form the wall of the body [24].

The abdominal wall is formed by the fusion of embryonic cephalic, caudal and two lateral folds. In the 6th week of pregnancy, during the abdominal wall formation, the rapid development of the intestinal tract determines its migration through the umbilical ring outside the abdominal cavity – in the umbilical cord. In the 10 to 12 week – the abdominal wall is well formed, and the abdominal cavity is spacious enough for the intestine to return, subsequently the normal rotation and fixation occur [26, 80].

Etiology and pathogenesis. It is believed that malformation develops in the 4 - 8 week of gestation, the etiology of gastroschisis being a subject of debate, based on the role of intrauterine stroke in the pathogenesis of this malformation. Duhamel (1963) considers that early disturbances of the embryonic (somatopleuric) mesoderm differentiation may be due to developmental defects in the abdominal wall.[21] Later Vermeji-Keers C. and coauthors (1996) presented a modified theory based on the mesoderm dysplasia of the umbilical ring with development of a lateral defect [80].

Shaw (1975) suggested that gastroschisis is caused by the rupture of the amniotic membrane from the basis of the umbilical cord that occurs during the physiological hernia or delay of the umbilical ring closure. This theory does not explain the rupture production and the mode of existence of the normal skin located between the umbilical cord and parietal defect [66].

DeVries P.A. (1980) sustained the fact that premature involution of the right umbilical vein leads to adverse effects on the adjacent mesenchyme, causing necrosis of the abdominal wall with the subsequent development of the abdominal wall rupture [18].

Hoyme H.E. et al. (1981) theorized that the disruption of the right vitelline (omphalomesenteric) artery in the umbilical region causes infarction and necrosis of the umbilical cord base with the subsequent rupture of the abdominal wall and herniation of the bowel contents through the defect. This hypothesis is not anymore accepted because it has been found that the vitelline arteries supply the intestinal and vitelline sac, but not the abdominal wall, which is vascularized from the dorsolateral branches of the aorta [33].

Komuro H. et al. (2010) support the hypothesis of association of developmental disorders of the vitelline sac and fusion of related vitelline structures embedded in the umbilical cord with pathogenesis of the abdominal wall defect in gastroschisis. There is an opinion that paraumbilical bands derived from the vitelline structures could be a possible cause of the intestinal ischemia during prenatal or postnatal period [40].

In recent years the risk factors in the development of gastroschisis have been identified, including: maternal and paternal age, socioeconomic status, drug

use and smoking during pregnancy, alcohol use, maternal infections, contact with chemicals, irradiation [79]. It was retrospectively found the role of constrictive preparations (ephedrine, cocaine, etc.), non-steroidal anti-inflammatory drugs (ibuprofen) and smoking in the development of gastroschisis and intestinal atresia [87]. The incidence of gastroschisis is fairly large in the group of pregnant women aged 15-19 years, constituting 1:400 of newborns [91].

The role of genetic factors in the etiology of gastroschisis is unclear. Although familial cases are reported, gastroschisis occurs in sporadic cases [78].

Recent studies have identified a positive association for NOS3, ANP, ADD1, ICAM1 genes with gastroschisis. These genes are linked to the mechanism of angiogenesis, dermal and epidermal resistance, the integrity of the vessels, supporting the hypothesis of vascular compromising in the aetiology of gastroschisis [77].

It was suggested the idea that cadmium and CO2 content in tobacco induce the expression of inflammatory factors (TNF and NFK-A), activating NOS3 and ICAM1, which could be involved in the pathophysiology of gastroschisis [25]. Gastroschisis more frequently appears as an isolated defect (83.3 to 93%), but in 12.2 to 35% of cases it may be part of syndromes and chromosomal abnormalities (trisomy 13, 18, 21 etc.).

Clinical picture and diagnosis do not present any great difficulties. The anterior wall defect of the abdomen is 2-8 cm, through which the eventration of the stomach, small intestine, a part of the large intestine, sometimes urinary bladder, uterus with its adjacent structures occur [49]. Although some authors claim that in the case of real gastroschisis the liver is always located in a normal position [56], there are reports which indicate presence of the liver herniation from 2.3 to 16% [10, 48]. It is not surprising that prenatal diagnosis of gastroschisis is often questioned when there is herniation of the liver, since liver eventration is more commonly associated with omphalocele [48]. Parietal defect can rarely be less than 2 cm and it involves all layers of the abdominal wall. Minor gastroschisis is considered a minor abdominal defect in the right side and adjacent to the umbilical cord with only a small protrusion of the omentum [89].

Eventrated organs are edematous, covered by gelatinous exudate or by a thick layer of fibrin, formed in the intrauterine period in response to the amniotic waters action and disturbances of mesenteric blood supply to the defect. Intestinal loops have a gray or cyanotic color, the peristalsis being absent. Intrauterine retardation and increase of mortality may be associated with the syndrome of malabsorption and loss of amino acids through the amniotic fluid [38, 79].

During the postnatal period gastroschisis is classified into two groups: simple and complex [6, 17]. The incidence of complex gastroschisis ranges from 11% to 31% [6, 22, 50]. Concomitant intestinal pathology is detected in the complex forms of gastroschisis: malrotation, vitelline duct remnants, volvulus, atresia or stenosis, infarction, perforation. This spectrum can contribute to occurrence of the short bowel syndrome, increasing essentially the incidence of mortality [32, 50].

In addition to mechanical occlusions, a severe dysmotility syndrome is determined in patients with gastroschisis, which is manifested by pseudo-obstruction [55].

"Closed" gastroschisis is a form of malformation conditioned by a significant closure of the abdominal wall defect around the eviscerated medium intestine, causing a series of sequels: intestinal infarction, intestinal reabsorption, intestine mummification with complete closure of the parietal defect ring [16, 67]. The simple intracavitary occlusion without vascular deficiency is considered less severe. But a remnant of extra-abdominal bowel is most often identified, which is located on the right side of the umbilic as an unviable retracted mass or the intestine has a normal length with gangrenous changes. Proximal intra-abdominal intestine is distended, having a variable length [89].

Prenatal diagnosis is determined by ultrasonography, beginning with 12 - 14 week of pregnancy after completion of the physiological hernia phase of the medium intestine [9, 85]. It should be noted that the pre-natal ultrasound is not always able to identify gastroschisis along with intestinal atresia, having an efficiency of 70-80%. Assessment of alpha-fetoprotein level, chorionic gonadotropin in maternal serum and index of acetylcholinesterase/ pseudocholinesterase is useful in the prenatal diagnosis of gastroschisis [79].

Differential diagnosis is made with: omphalocele, bladder extrophy, amniotic band syndrome, cord ectopia, Cantrell pentalogy [71].

Treatment. The first gastroschisis surgery was reported by Fear W. (1878) [23], Watkins (1943) being the first to perform the operation successfully [84]. In 1953 Moore T.C. and Stokes G.E. proposed the method of abdominal sealing with skin folds [51].

Preoperative stabilization and manangement of the newborn with gastroschisis should consider several factors. Significant water loss through evaporation and heat loss take place in gastroschisis from the exposed intestine, with the subsequent occurrence of metabolic changes. Immediately after birth it is necessary to find an adequate intravenous access and to initiate the restoration of the fluid volume. The child should be monitored for signs of hypothermia, respiratory distress and shock. Stomach decompression and prevention of the bowel distension are important.

Eventrated intestine must be wrapped with gauze dressings soaked with warm physiological saline solution, then it is placed centrally on the abdominal wall, the child being positioned on the right side to prevent twisting of the mesentery . It is necessary to

carry out a thorough examination of the child to exclude the coexistence of other malformations. Examination of the eventrated bowel requires particular attention to highlight the presence of intestinal atresia, bowel necrosis or perforation [9].

Contemporary strategies of surgical correction of gastroschisis have considerably evolved in the last years. However the principles of surgical treatment of congenital abdominal wall defects are the same: to reduce eviscerated organs in the abdominal cavity in safety, closing the abdominal wall defect with an acceptable cosmetic appearance, identification and treatment of associated malformations, establishing an independent nutrition support until switching to full enteral nutrition, detection and appropriate treatment of abdominal, intestinal or wound complications [46, 54].

Primary reduction with defect closure with fascial sutures represents the initial standard of the surgical strategy, while the gradual reduction is commonly used as a rescue strategy in cases of visceroabdominal disproportion. Unimomental primary closure of gastroschisis should be the priority goal, because it provides the best protection of the viscera. At the same time, this principle may be resolved in two-thirds of cases [54].

Closure of the abdominal wall with skin flaps (bimomental grafting) was the only method of gastroschisis treatment until the late 60s. Deliberate distension of the abdominal wall, described for the first time by Izant and Brown (1966) [36] and complete evacuation of the bowel meconium are essential for the success of this technique [45, 76]. A ventral iatrogenic hernia formation occurs, removal of the ventral hernia after 6 - 12 months being the second stage of the surgery [91].

In some cases when the primary closure is not possible, temporary or permanent management of the parietal defect can be achieved by the use of some synthetic or biological patches. The synthetic ones (nonabsorbable) are used for the temporary closure of the abdominal parietal defect until the fascia surface allows effective closure of the abdomen. Absorbable meshes (Poliglactin, Vicril) proved to be inert, nonantigenic, nonpyrogenic, being used for long-term or permanent closure of the abdominal cavity.

The group of synthetic materials includes: Marlex, silastic sheets, monofilament polypropylene mesh, Gore.Tex. Synthetic material sutured to the fascia can cause some complications, including infection, visceral erosions, fistulae, skin erosions, adhesion development [7, 27 , 46]. Porcine dermal collagen and porcine intestinal submucosa are used from acellular xenographic biomaterials (Surgis). Human acellular allografts have proven to be quite effective in the plastic surgery of abdominal wall defects (AlloDerm) [46 , 58].

The success of primary fascial closure is dependent upon the dimensions of abdominal visceral disparity and subsequent effects of intra-abdominal hypertension, which is still the biggest challenge for the pediatric surgeons. Syndrome of the abdominal compartment has been recognized to be an important cause of morbidity and mortality in the late `80s. Slight increase in the intra-abdominal pressure can cause kidney and respiratory failure, as well as deficiencies and bowel ischemia [43].

Measures designed to resolve the abdominal visceral disproportion ranged from organs resection to distension of the abdominal wall and closure with skin flaps [39, 46, 86]. Evaluation of pressure in the bladder is of great importance in the choice of tactics of primary closure of the abdominal wall in conditions of complete safety, being determined that these values should not be more than 20 mmHg after reducing the contents of the abdominal cavity [53]. It was found that the primary closure is often associated with a temporary but significant deterioration of the respiratory function caused by the decrease of respiratory compliance [19].

The advantages of primary fascial closure are: shortening of the development period of the ileus, a shorter time interval for switching to peroral nutrition, reduction of hospital stay, elimination of the need for a repeated surgery [8, 64].

The concept of gradual reduction of eviscerated organs using gauze bags was described for the first time by Schuster S.R. in 1967. Later, Allen and Wrenn (1969) proposed to use silastic bags [65]. Use of this method along with parenteral feeding allowed a significant reduction of the number of patients with gastroschisis [37, 74].

Special silastic bags have been widely used since 1997 in order to gradually introduce the eviscerated contents into the abdomen. Although it leads to a longer hospital stay, this method has decreased the risc of long-term intestinal disturbances and the need for a new surgery [39]. Some risks are still present such as infection, suture line dehiscence [43], development of intestinal adhesions, development of fistulas caused by the synthetic material or mechanical complications caused by the parietal defect ring [59].

The umbilical cord is an autogenous material that has proved to be useful in the treatment of abdominal wall defects [29, 61], including temporary covering of the eventrated viscera in gastroschisis [86]. This technique along with the method of dividing the rectus abdominis muscle were described as methods that facilitate primary closure of the abdomen, but that lead to a significant increase in the incidence of ventral hernias [42].

Postoperative complications include: intestinal ischemia, bowel infarction, development of enterocutaneous fistulas, necrotizing enterocolitis, intestinal obstruction, short bowel syndrome, prolonged intestinal disorders, septic complications, renal ischemia [12].

The prognosis of malformation is severe. Mortality in gastroschisis has 3 phases: 1) prenatal and terminal

deaths, 2) early neonatal deaths, 3) late deaths caused by adhesive intestinal obstructions, association of liver disturbances. Hypothermia, fluid loss and sepsis continue to influence significantly the risk of mortality [5, 46, 62, 78]. Some authors report a quite acceptable postnatal mortality index of 5.9 - 8% [20, 62], while in some parts of the world the values of this index are disturbing, constituting 28% [22] or 50% [81]. Most reports indicate a survival of approximately 90% - 97% in simple gastroschisis [1, 50] and about 10% - in complex gastroschisis [46].

REFERENCES

1. Abdullah F., Arnold M.A., Nabaweesi R. et al. Gastroschisis in the United States 1988-2003: analysis and risk categorization of 4344 patients. J. Perinatol. 2007. 27:50-5.
2. Agarwal R. Prenatal diagnosis of anterior abdominal wall defects: Pictorial essay. Ind. J. Radiol. Imag. 2005. 15(3):361-72.
3. Akhtar J., Skarsgard E.D. Associated malformations and the "hidden mortality" of gastroschisis. J. Pediatr. Surg. 2012. 47:911-6.
4. Allen R.G., Wrenn Jr. E.L. Silon as a sac in the treatment of omphalocele and gastroschisis. J. Pediatr. Surg. 1969.; 4:3 – 8.
5. Ameh E.A., Chirdan L.B. Ruptured exomphalos and gastroschisis: a retrospective analysis of morbidity and mortality in Nigerian children. Pediatr. Surg. Int. 2000. 16:23-5.
6. Arnold M.A., Chang D.C., Nabaweesi R. et al. Risk stratification of 4344 patients with gastroschisis into simple and complex categories. J Pediatr Surg 2007:42:1520-5.
7. Beres A., Christison-Lagay E.R., Romao R., Langer J.C. Evaluation of Surgis for patch repair of abdominal wall defects in children. J. Pediatr. Surg. 2012. 47:917-9.
8. Bianchi D.W., Crombleholme T.M., Alton M.E. Gastroschisis; in Fetology: Diagnosis and Management of the Fetal Patient. New York. McGraw-Hill. 2000. pp 473-82.
9. Boia E.S., Iacob R.E., Adam O. et al. Surgical treatment of gastroschisis using silimed gastroschisis container – case report. J. Pediatr. 2006. 9 (35-36):39-45.
10. Brantberg A., Blaas H.G.K., Salvesen K.Å. et al. Surveillance and outcome of fetuses with gastroschisis. Ultrasound Obstet. Gynecol. 2004. 23:4-13.
11. Calder J. Two examples of children born with preternatural conformations of the guts. Med. Essays Observations Edinb. 1733:203-4.
12. Chabra S. Management of gastroschisis: prenatal, perinatal, and neonatal. NeoReview. 2006. 7:e419.
13. Chabra S., Gleason C.A. Gastroschisis: embryology, pathogenesis, epidemiology. NeoReviews. 2005. 6(11):e493-e498.
14. Ciobanu O., Stoica A., Zavate A. ş.a. Laparoschisis – Consideraţii clinico-terapeutice. Craiova Medicală. 2007. 9(4):314-7.
15. Curry J.I., McKinney P., Thornton J.G., Stringer M.D. The aetiology of gastroschisis. Br. J. Obster. Gynecol. 2000. 107:1339-46.
16. Davenport M., Haugen S., Greenough A. et al. Closed gastroschisis: Antenatal and postnatal features. J. Pediatr. Surg. 2001. 36:1834-7.
17. Davis R.P., Treadwell M.C., Drongowski R.A. et al. Risk stratification in gastroschisis: can prenatal evaluation or early postnatal factors predict outcome? Pediatr. Surg. Int. 2009. 25:319-25.
18. deVries P.A. The pathogenesis of gastroschisis and omphalocele. J. Pediatr. Surg. 1980. 15:245e51.
19. Dimitriou G., Greenough A., Giffin F. et al. Temporary impairment of lung function in infants with anterior abdominal wall defects who have undergone surgery. J. Pediatr. Surg. 1996. 31:670-2.
20. Driver C.P., Bruce J., Bianchi A. et al. The contemporary outcome of gastroschisis. J. Pediatr. Surg. 2000. 35:1719-23.
21. Duhamel B. Embryology of exomphalos and allied malformations. Arch. Dis. Child. 1963. 38:142-7.
22. Eggink B.H., Richardson C.J., Malloy M.H. et al. Outcome of gastroschisis: a 20-year case review of infants with gastroschisis born in Galveston, Texas. J. Pediatr. Surg. 2006. 41:1103-8.
23. FearW. Congenital extrusion of abdominal viscera: return: recovery. Br. Med. J. 1878. 2:518-20.
24. Feldkamp M.L., Carey J.C., Sadler T.W. Development of gastroschisis: Review of hypotheses, a novel jypotheses, and implications for research. Am. J. Med. Gen. 2007. 143A:639-52.
25. Green R.F., Moore C. Incorporating genetic analyses into birth defects cluster investigations: strategies for identifying candidate genes. Birth. Defects Res. A Clin. Mol. Teratol. 2006. 76:798-10.
26. Grevious M.A., Cjhen M., Shah S.R., Rodriguez P. Structural and functional anatomy of the abdominal wall. Clin. Plastic. Surg. 2006. 33:169-79.
27. Grevious M.A., Cohen M., Jean-Pierre F., Herrmann G.E. The use of prosthetic in abdominal reconstruction. Clin. Plastic Surg. 2006. 33:181-97.
28. Guler I., Erdem A., Biri A. et al. Gastroschisis with fetal chromosomal abnormality: a case report. Fetal Diagn. Ther. 2007. 22:274-6.
29. Heaton F.C., Thomas Jr C.G., Owen J. The use of umbilical cord for reconstruction of abdominal wall defects. Surg. Forum. 1970. 21:56-7.
30. Hey W. Practical observations in surgery. London: Cadell and Davis; 1803. p. 228.
31. Hipolito R., Haight M., Dubois J. et al. Gastroschisis and Hirschsprung's disease: a rare combination. J. Pediatr. Surg. 2001. 36:638-40.
32. Houben C., Davenport M., Ade-Ajayi N. et al. Closing gastroschisis: diagnosis, management, and outcomes. J. Pediatr. Surg. 2009. 44:343-7.
33. Hoyme H.E., Higginbottom M.C., Jones K.L. The vascular pathogenesis of gastroschisis: intrauterine interruption of the omphalomesenteric artery. J. Pediatr. 1981. 98:228-31.
34. Hunter A.G., Stevenson R.E. Gastroschisis: clinical presentation and associations. Am. J. Med. Genet. C. Semin. Med. Genet. 2008. 148C:219-230.
35. Hwang P.J., Kousseff B.G. Omphalocele and gastroschisis: an 18-year review study. Genet. Med. 2004. 6:232-6.
36. Izant R.J., Brown F. Current embryology and treatment of gastroschisis and omphalocele. Arch. Surg. 1966. 93:49-53.
37. Jona J.Z. The bgentle touch technique in the treatment of gastroschisis. J. Pediatr. Surg. 2003. 38(7):1036- 8.
38. Kale A., Kale E., Akdeniz N., Canoruc N. Elevated amniotic fluid amino acid levels in fetuses with gastroschisis. Braz. J. Med. Biol. Res. 2006. 39:1021-5.
39. Kidd J. N., Jackson R.J., Smith S.D., Wagner C.W. Evolution of staged versus primary closure of gastroschisis. Ann. Surg. 2003. 237(6):759-65,
40. Komuro H., Hoshino N., Urita Z. et al. Patogenic implications of remnant vitelline structures in gastroschisis. J. Pediatr. Surg. 2010. 45(10):2025-9.
41. Kunz L.H., Gilbert W.M., Towner D.R. Increased incidence of cardiac anomalies in pregnancies complicated by gastroschisis. Am. J. Obstet. Gynecol. 2005. 193:1248-52.
42. Lafer D.J. Rectus muscle transection for visceral replacement in gastroschisis. *Surgery*. 1968. 63:988-90.
43. Lansdale N., Hill R., Gull-Zamir S. et al. Staged reduction of gastroschisis using preformed silos: practicalities and problems. J. Pediatr. Surg. 2009. 44:2126 – 9.
44. Ledbetter D.J. Gastroschisis and omphalocele. Surg. Clin. N. Am. 2006. 86:249-60.
45. Luck S.R., Sherman J.O., Raffensperger J.G., Goldstein I.R. Gastroschisis in 106 consecutive newborn infants. Surgery. 1985. 98:677-83.

46. Marven S., Owen A. Contemporary postnatal surgical management strategies for congenital abdominal wall defects. Semin. Pediatr. Surg. 2008. 17:222-35.
47. Mastroiacovo P., Lisi A., Castilla E.E. et al. Gastroschisis and associated defects: an international study. Am. J. Med. Genet. A 2007. 143:660-71.
48. McClellan E.B., Shew S.B., Lee S. et al. Liver herniation in gastroschisis: incidence and prognosis. J. Pediatr. Surg. 2011. 46:2115-8.
49. Mirza B., Sheikh A. An unusual case of gastroschisis. APSP J. Case Rep. 2010. 1:2.
50. Molik K.A., Gingalewski C.A., West K.W. et al. Gastroschisis: A plea for risk categorization. J. Pediatr. Surg. 2001. 36:51-5.
51. Moore T.C., Stokes G.E. Gastroschisis. Report of two cases treated by a modification of the gross operation for omphalocele. Surgery. 1953. 33:112-20.
52. Nichol P.F., Byrne J.L., Dodgion C., Saijoh Y. Clinical considerations in gastroschisis: incremental advances against a congenital anomaly with severe secondary effects. Am. J. Med. Genet. C. Semin. Med. Genet. 2008. 148C:231-40.
53. Olesevich M., Alexander F., Khan M., Cotman K. Gastroschisis revisted: role of intraoperative measurement of abdominal pressure. J. Pediatr. Surg. 2005. 40:789-92.
54. Owen A., Maeven S., Johnson P. et al. Gastroschisis: a national cohort study to describe contemporary surgical strategies and outcomes. J. Pediatr. Surg. 2010. 45:1808-16.
55. Phillips J.D., Raval M.V., Redden C. et al. Gastroschisis, atresia, dysmotility: surgical treatment strategies for a distinct clinical entity. J. Pediatr. Surg. 2008. 43:2208-12.
56. Redford D.H., McNay M.B., Whittle M.J. Gastroschisis and exomphalos: precise diagnosis by midpregnancy ultrasound. Br. J. Obstet. Gynaecol. 1985. 92:54-9.
57. Rex D., Chowdhury M.M., O'Malley A.J. et al. Gastroschisis with Hirschsprung's disease: a therapeutic dilemma. J. Pediatr. Surg. 2010. 45:1724-6.
58. Rice R.D., Ayubi F.S., Shaub Z.J. et al. Comparison of Surgisis, AlloDerm, and Vicryl Woven Mesh grafts for abdominal wall defect repair in an animal model. Aesth. Plast. Surg. 2010. 34:290-6.
59. Ryckman J., Aspirot A., Laberge J.M. et al. Intestinal venous congestion as a complication of elective silo placement for gastroschisis. Semin. Pediatr. Surg. 2009. 18:109-12.
60. Sabine Punzmann. Omphalozele und gastroschisis. Innaugural-Dissertation zur Erlangung des Doctorgrades der Medizin. 2011. 121 p.
61. Samii A.M., Jafroudi Y. Gastroschisis: use of umbilical cord in its repair. J. Med. Liban. 1974. 27:473-7.
62. Sangkhathat S., Patrapinyokul S., Chiengkriwate P. et al. Infectious complications in infants with gastroschisis: an 11 year review from a referral hospital in southern Thailand. J. Pediatr. Surg. 2008. 43:473-8.
63. Santiago-Munoz P.C., McIntire D.D., Barber R.G. et al. Outcomes of pregnancies with fetal gastroschisis. Obstet. Gynecol. 2007. 110:663-8.
64. Schmidt A.F., Goncalves A., Bustorff-Silva J.M. et al. Does staged closure have a worse prognosis in gastroschisis? Clinics. 2011. 66(4):563-6.
65. Schuster S.R. A new method for the staged repair of large omphalocele. Surg. Gynecol. Obstet. 1967. 125:837- 50.
66. Shaw A. The myth of gastroschisis. J. Pediatr. Surg. 1975. 10:235-44.
67. Sherif E., Noah C., Tiffany Ch. et al. Contemporary 2-year outcomes of complex gastroschisis. J. Pediatr. Surg. 2012. 47:1521-8.
68. Shi Y., Farinelli C.K., Chang M.S., Carpenter Ph.M. Left-sided gastroschisis with placenta findings: case report and literature review. Int. J. Clin. Exp. Pathol. 2012. 5(3):243-6.
69. Skandalakis J.E., Gray S.W., Ricketts R. The anterior body wall. In: Embryology for Surgeons. 4th ed Philadelphia: Lippincott Williams & Wilkins. 2006. pp.605-89.
70. Spitz L. A historical vignette: gastroschisis. J. Pediatr. Surg. 2008. 43:1396-7.
71. Stevenson R.E., Hall J.G., Goodman R.M. Human Malformations and Related Anomalies. New. York: Oxford University Press. 1993. pp. 882-5.
72. Stoll C., Alembik Y., Dott B. et al. Omphalocele and gastroschisis and associated malformations. Am. J. Med. Genet. A 2008. 146A:1280-5.
73. Suver D., Lee S.L., Shekherdimian S. et al. Left-sided gastroschisis: higher incidence of extraintestinal congenital anomalies. Am. J. Surg. 2008. 195(5):663-6.
74. Sydorak R.M., Nijagel A., Hirose S. et al. Gastroschisis: small hole, big cost. J. Pediatr. Surg. 2002. 37(12):1669- 72.
75. Taruffi C. Storia della teratologia, Vol. VII. Bologna, Italy: Regia Tipografia; 1894.
76. Thepcharoennirund S. Primary fascial closure in 112 infants with gastroschisis. J. Med. Assoc. Thai. 2005. 88(4):492-7.
77. Torfs C.P., Christianson R.E., Iovannisci D.M. et al. Selected gene polymorphisms and their interaction with maternal smoking, as risk factors for gastroschisis. Birth. Defects Res. A Clin. Mol. Teratol. 2006. 76:723-30.
78. Tsai M.C., Chen S.N. Hypothermia and sepsis: the major causes of mortality in gastroschisis. Zhonghua Min. Guo. Xiao Er. Ke Yi Xue Hui Za Zhi. 1995. 36:328-30.
79. Valdez J.A.L., Coyolt D.M.C., Vega C.A.V. New embryological hypothesis, genetics an epidemiology of gastroschisis. Bol. Med. Hosp. Infant. Mex. 2011. 68(3):224-31.
80. Vermeij-Keers C., Hartwig N.G., van der Werff J.F. Embryonic development of the ventral body wall and its congenital malformations. Semin. Pediatr. Surg 1996. 5:82-9.
81. Vilela P.C., Ramos de Amorim M.M., Falbo G.H. et al. Risk factors for adverse outcome of newborns with gastroschisis in a Brazilian hospital. J. Pediatr. Surg. 2001. 36:559-64.
82. Vissick C. An umbilical hernia in a newly born child. Lancet. 1873.1:829.
83. Waldhausen J.H.T. Surgical management of gastroschisis. Neoreviews. 2005. 6:e500.
84. Watkins D.E. Gastroschisis. Va. Med. 1943. 70:42-5.
85. Weir E. Congenital abdominal wall defects. CMAJ. 2003. 169:809-10.
86. Werbeck R., Koltai J. Umbilical cord as temporary coverage in gastroschisis. Eur. J. Pediatr. Surg. 2011. 21:292-5.
87. Werler M.M., Sheehan J.E., Mitchell A.A. Association of vasoconstrictive exposures with risks of gastroschisis and small intestinal atresia. Epidemiology. 2003. 14:349e54.
88. Williams L.J., Kucik J.E., Alverson C.J. et al. Epidemiology of gastroschisis in metropolitan Atlanta, 1968 through 2000. Birth Defects Res. A Clin. Mol. Teratol. 2005. 73:177-83.
89. Wilson R.D., Johnson M.P. Congenital abdominal wall defects: an update. Fetal. Diagn. Ther. 2004. 19:385-98.
90. Yoshioka H., Ayoama K., Iwamura Y., Muguruma T. Two cases of left-sided gastroschisis: review of the literature. Pediatr. Surg. Inter. 2004. 20(6):472-3.
91. Веселый С.В., Грона В.Н., Перунский В.П. и др. Методы диагностики и лечения гастрошизиса и омфалоцеле у детей. Перинатол. и Педиаир. 2006. 27(3):1-6.
92. Грона В.Н., Перунский В.П., Веселый С.В. и др. Оптимизация лечения врожденных расщелин передней юрюшной стенки у детей. Укр. Ж. Хир. 2008. №1. С.105-12.