PERFORATION OF THE STOMACH IN NEWBORNS

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Gastrointestinal perforation, which has a high mortality rate in neonates, is a serious surgical problem[98]. Despite the fact that most perforations occur in the small and large intestines, gastric damage is also a cause of life-threatening conditions in newborns [10,14,27,38]. The prevalence of gastric perforation (GP) is difficult to assess, since not all cases are recorded by medical statistics. According to the literature, the pancreas ranges from 1:2900 to 1:5000 live births and 7-10% of all gastrointestinal perforations in newborns [25,63,77,100]. Spontaneous gastric perforation in a newborn was first described in 1825 by Siebold [78]. From 1986 to 2018, 438 cases of pancreas were described in the medical literature (Table 1). 69 English-language sources in the electronic databases MEDLINE and SCOPUS present 328 cases of pancreas. Russian-speaking authors in 14 literature sources report 110 cases of pancreas. The purpose of this report is to analyze the available literature, characterize the modern understanding of pancreas, describe risk factors, diagnosis, treatment methods and results, and determine prognostic aspects.

Material and methods A systematic review of the literature was carried out in accordance with the international recommendations PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) [112] and included electronic databases MEDLINE and SCOPUS and Russian-language literature sources. To include a larger number of patients, full-text articles, references to literature sources, abstracts, reports of single cases of pancreas, reports of newborns with gastric perforation were analyzed. Despite its increasing prevalence, pancreas remains a relatively rare disease, with mostly small case series reported. Several aspects of pancreas, from etiology to optimal treatment, are still unclear.

Etiology and pathogenesis Although numerous theories have been proposed, the etiology of pancreas remains unclear [109]. A number of authors believe that gastric perforation in newborns has a different pathogenesis than intestinal perforation. The literature reports on the etiology of gastric perforation from different points of view: congenital local muscle agenesis, high acidity of gastric juice, gastrointestinal ischemia caused by asphyxia, increased intragastric pressure due to distal obstruction or aerophagia, uncoordinated motility of the upper gastrointestinal tract, absence of C-KIT/CD117 (receptor tyrosine kinase) + mast cells or interstitial pacemaker cells. It is also believed that increased intragastric pressure plays a major role, which may be a consequence of the lack of coordination of the upper gastrointestinal tract and the immaturity of the vomiting mechanism in the newborn. It was previously assumed that spontaneous gastric perforation was caused by a congenital absence of gastric musculature [20,50,82,97]. Herbut (1943) examined the site of perforation histologically and found that there was no gastric

musculature near the perforation[50]. Herbut explained these findings by congenital agenesis of the muscles along the greater curvature and the fact that the mucosa is covered with a serous layer without smooth muscle. Shaw et al. (1965) experimentally refuted the theory of congenital muscle agenesis in dogs by tying the ends of the stomach and duodenum and blowing air until the organ was perforated [101]. The author found that all perforations were localized along the greater curvature of the stomach; upon histological examination, all samples showed the absence of muscle near the perforation. Shaw et al. (1965) concluded that the histological appearance of absent muscle at the edge of the perforation was the result of traction caused by increased intragastric pressure [101]. Gastric rupture can occur with sufficiently strong stretching due to the separation of muscle bundles of the stomach wall (usually along the greater curvature). Holgerson in 1981 repeated Shaw's experiment on 10 stomachs of newborns after their death and obtained identical results [51].

Risk factors Given the lack of convincing and reliable data on the causes of gastric perforation, a careful assessment of risk factors is necessary. In addition, taking into account cases (non-idiopathic) with obvious reasons for the pancreas, for example, such as perforation of a feeding tube, distal obstruction, nasal ventilation, one cannot but agree with the fact that this complication is extremely low in patients with tube feeding, high intestinal obstruction or respiratory (nasal) therapy. From this it is possible to make the assumption that an increase in intragastric pressure can serve as a trigger for rupture of the gastric wall. Based on the literature review, a number of general aspects can be identified.

Thus, gastric perforation in newborns remains a rare and life-threatening condition. Over the past 15 years, the survival rate of infants with this pathology has increased from 25 to 50%, which is associated with progress in neonatal intensive care methods. The key to a good result is an early diagnosis to correct metabolic and electrolyte disorders before they become irreversible, and timely adequate surgical intervention.

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