REVIEW ARTICLE

CONTENTS

Anatomical, physiological and morphological features of the liver in newborns with biliary atresia (based on literature data and personal observations)

Olga M. Gorbatyuk
EDUCATIONAL AND SCIENTIFIC INSTITUTE OF MEDICINE, UNIVERSITY KROK, KYIV, UKRAINE

ABSTRACT

Aim: To provide a fundamental understanding of the anatomical and histological structure of the liver in children with BA, which are essential for accurate diagnosis, the development of targeted treatment approaches, and effective surgical correction.

Materials and Methods: A literature review was conducted on embryogenesis, anatomy, and morphology of the disease in newborns and children with biliary atresia (BA). At least 27 scientific sources were analyzed, covering the period from classical descriptions (since 1972) to modern publications (2020s). The selection criteria included studies describing liver embryogenesis, morphological features, and clinical and surgical aspects of BA. The databases PubMed, Medline, Embase, and Google Scholar were searched, covering research areas such as liver anatomy, physiology, and morphology in children, as well as clinical issues related to biliary atresia. In addition, the author's observations were included. These were obtained during diagnostic imaging procedures, intraoperative evaluation of the organ during surgical correction, anatomical studies of 7 deceased children with BA, and histological examination of liver tissue. All patients were under our observation during diagnosis and treatment for BA and demonstrated characteristic clinical, anatomical, physiological, and morphological changes of the liver.

Conclusions: The cause of BA may lie in disruptions or arrest of the embryonic development of the bile ducts and hepatic vascular system. In infants, the liver is in a state of anatomical immaturity and functional insufficiency, and its structure is not fully developed. This must be taken into account when treating children with BA.The anatomical differences in the liver of children with BA have significant practical implications for the surgical management of BA.

KEY WORDS: biliary atresia, infants, anatomy and physiology of liver, treatment

Wiad Lek. 2025;78(10):2151-2159. doi: 10.36740/WLek/213594 DOI

INTRODUCTION

Biliary atresia (or atresia of the bile ducts) occurs in approximately 1 in 8,000 to 1 in 30,000 newborns, according to various studies [1-3]. In biliary atresia (BA), the affected obliterated ducts create a mechanical obstruction to the normal flow of bile, resulting in its retention in the liver and causing chronic inflammation and hepatocellular damage [4]. BA accounts for up to 39% of all liver-related mortality in children [5,6]. For many years, the condition was considered incurable until 1959, when Kasai M. and Suzuki S. introduced the surgical procedure of hepatic portoenterostomy, which made it possible to transform the "uncorrectable" type of BA into a "correctable" one, thus saving the lives of many children [7,8].

Clinically, the condition presents with persistent jaundice of the skin and mucous membranes, pale stools, and dark urine. Jaundice in BA appears within the first days of life, progresses steadily, and is caused by elevated levels of direct bilirubin. The liver becomes enlarged and firm, and by the age of 5-6 months, biliary cirrhosis of the liver develops. By the age of 2 months, the spleen also enlarges [9,10].

The choice of surgical strategy depends on the type of BA. According to the anatomical classification, there are three types of BA: 1) atresia of the extrahepatic (external) bile ducts with preservation of the ducts at the hepatic hilum and a well-developed gallbladder; 2) atresia of the intrahepatic bile ducts; 3) total atresia of the bile ducts. Atresia of the extrahepatic bile ducts (the "correctable type") at the level of the common bile duct or hepatic duct is considered a favorable form of the defect in terms of successful surgical correction. In such cases, it is generally possible to achieve good bile drainage. In contrast, intrahepatic duct atresia and the total form present significant difficulties for correction, which has led to their classification as the "uncorrectable type" [11-14].

BA is the most common cholestatic liver disease in infants and the leading indication for liver transplantation [15].

Understanding BA involves liver embryogenesis along with its anatomical and morphological features, as these contribute to a complex pattern of pathological changes requiring surgical correction. Successful treatment of children with BA greatly depends on the surgeon's thorough understanding of the disease's nature and mechanisms of development. Without up-to-date knowledge of the disease's etiopathogenesis, as well as the anatomical, functional, and morphological features of the liver, achieving effective treatment outcomes is not possible.

AIM

To provide a fundamental understanding of the anatomical and histological structure of the liver in children with BA, which are essential for accurate diagnosis, the development of targeted treatment approaches, and effective surgical correction.

MATERIALS AND METHODS

A literature review was conducted on embryogenesis, anatomy, and morphology of the disease in newborns and children with biliary atresia (BA). At least 27 scientific sources were analyzed, covering the period from classical descriptions (since 1972) to modern publications (2020s). The selection criteria included studies describing liver embryogenesis, morphological features, and clinical and surgical aspects of BA. The databases PubMed, Medline, Embase, and Google Scholar were searched, covering research areas such as liver anatomy, physiology, and morphology in children, as well as clinical issues related to biliary atresia. In addition, the author's observations were included. These were obtained during diagnostic imaging procedures, intraoperative evaluation of the organ during surgical correction, anatomical studies of 7 deceased children with BA, and histological examination of liver tissue. All patients were under our observation during diagnosis and treatment for BA and demonstrated characteristic clinical, anatomical, physiological, and morphological changes of the liver.

REVIEW AND DISCUSSION

Brief overview of liver embryogenesis. The liver develops from the hepatic diverticulum of the primitive gut during the 4th week of intrauterine development. The central part of this diverticulum gives rise to the liver and hepatic ducts. Hepatic cells grow in the form of cords into the mesenchymal tissue of the transverse septum of the diverticulum, forming the hepatic parenchyma, bile canaliculi, and intrahepatic bile ducts. The caudal portion of the hepatic diverticulum gives rise to the gallbladder and the common bile duct, undergoing a solid proliferative stage followed by vacuolization. By the 7th-12th weeks of embryonic development, the gallbladder becomes a hollow organ, and a lumen appears in the cystic duct.

Simultaneously with the formation of the distal parts of the biliary system, hepatic secretory cords develop, and both extrahepatic and intrahepatic segments of the portal and hepatic veins form. During the embryonic period, liver lobules begin to form, and their final differentiation is completed by the end of the 1st month of life. The vascular system of the liver originates from the vitelline and umbilical veins. As the intestines develop, the biliary segment of the vitelline veins regresses, and the umbilical veins of the umbilical cord and embryo merge. The vessel passing through the hepatic parenchyma (the ductus venosus Arantii), which carries oxygenated blood into the inferior vena cava of the fetus, becomes obliterated after birth. Disruptions or arrest in the embryonic development of the biliary ducts and hepatic vasculature may lead to conditions such as biliary atresia, biliary hypoplasia, choledochal cysts, hepatic cysts, and portal vein anomalies, often resulting in portal hypertension [16,17,18].

The main structural element of the liver is the hepatocyte and the hepatic lobule, which ranges in diameter from 0.7 to 2.0 mm. It consists of a single layer of hepatocytes arranged around a central vein. Irregularly organized cells create interconnected cavities known as lacunae. It is within this histological structure that pathological processes such as necrosis and cirrhosis develop, leading to severe consequences [10].

The liver performs a wide range of functions. Its main roles include: 1-regulating metabolic processes by storing, secreting, and processing carbohydrates, fats, and proteins; 2-synthesizing proteins; 3-producing bile; 4-regulating the blood coagulation system; 5-performing hematopoietic functions; 6-participating in water and electrolyte balance; 7-carrying out detoxification; 8-modulating and supporting the immune system [19].

NORMAL LIVER ANATOMY IN NEWBORNS AND INFANTS

In newborns, the liver is one of the largest organs, accounting for more than 4% of total body weight. It is considered normal for the liver to extend 2-3 cm below the costal margin along the midclavicular line.

Typically, the right and left lobes of the liver in newborns are approximately equal in volume, or the left lobe may be larger than the right. This is due to features of intrauterine development, during which the left lobe is better supplied with blood. This fact should be taken into account during liver resection at this age. In the postnatal period, the liver continues to grow, although its mass increases more slowly than body weight. For example, by 10-11 months of age, liver weight doubles, whereas body weight triples.

The liver is covered with peritoneum on almost all sides (a mesoperitoneal organ), except for the posterior part of the diaphragmatic surface and the porta hepatis. The peritoneal covering of the liver, at the points where it extends to the diaphragm, the porta hepatis, and adjacent organs, forms the ligamentous apparatus of the liver. In addition to providing support, these ligaments serve as conduits for blood and lymphatic vessels and nerves. From a surgical standpoint, the most important ligaments are:

1) The coronary ligament, located in the frontal plane. At its lateral edges lie the left and right triangular ligaments. These are subject to surgical division during liver mobilization, cyst dissection, liver resection, or diaphragmatic hernia repair.

2) The fourth ligament – the falciform ligament, also known as the suspensory ligament, which is located in the sagittal plane. At the anterior edge of the liver, it transitions into the round ligament, which contains the umbilical vessels.

The hepatoduodenal ligament extends from the liver to the duodenum and contains vital anatomical structures: the portal vein, the hepatic artery, and the common bile duct. Its fusion with the hepatogastric ligament and the left gastrophrenic ligament forms the anterior wall of the lesser omentum, and the hepatoduodenal ligament itself forms the anterior border of the omental (epiploic) foramen. Within the hepatoduodenal ligament, the common and cystic bile ducts lie superficially (ventrally), the hepatic artery trunk lies slightly deeper and to the left, and the wide portal vein trunk is located dorsally behind them. These anatomical relationships are crucial and must be taken into account during surgical procedures at the porta hepatis.

During liver mobilization, surgical intervention involves the division of hepatic ligaments. The anatomy of these structures must be considered during resective operations. For example, during mobilization of the left lobe of the liver, the left triangular ligament is divided up to the beginning of the falciform ligament. During mobilization of the right lobe, the right triangular ligament, hepatorenal ligament, round ligament, and falciform ligament are divided.

The liver is divided into right and left lobes, separated by the falciform ligament or the left sagittal fissure, in the anterior part of which lies the round ligament of the liver, and in the posterior part – the obliterated ductus venosus (Arantius' duct).

The right sagittal fissure on the inferior surface of the liver runs from the gallbladder bed to the inferior vena cava. Between the sagittal fissures, anterior to the transverse fissure, lies the quadrate lobe, and posteriorly – the caudate lobe.

Couinaud's classification divides the right and left lobes of the liver into 5 sectors and 8 segments, each with its own autonomous blood supply, bile drainage, innervation, and lymphatic circulation. In the right lobe, two sectors are distinguished: lateral (segments 6 and 7) and paramedian (segments 5 and 8). In the left lobe, three sectors are distinguished: lateral (segment 2), paramedian (segments 3 and 4 – i.e., the quadrate lobe and most of the left lobe), and dorsal (segment 1 or the caudate lobe). These variants of dividing the liver into lobes, sectors, and segments are anatomically justified, as they reflect the liver internal structure. They are diagnostically useful for localizing pathological processes using ultrasound, CT, MRI, and angiography, and are widely applied in resective and alternative liver surgery [19].

The liver is the only organ with two systems of supplying vessels – the hepatic artery and the portal vein. The common hepatic artery originates from the celiac trunk. Within the hepatoduodenal ligament, it gives rise to the gastroduodenal, right gastric, and cystic arteries. In nearly half of cases, anomalies in hepatic artery development are found, including atypical branching and additional vascular branches, unusual origins of the hepatic artery (e.g., from the superior mesenteric artery rather than the celiac trunk), and independent origins of the left and right hepatic branches. The hepatic artery provides 10-35% of the liver blood flow and supplies 50% of its oxygen needs, while 65% of hepatic blood flow is provided by the portal vein.

The portal vein is formed by the confluence of the superior mesenteric and splenic veins. Its tributaries include the inferior mesenteric vein and the coronary vein of the stomach, which typically drain into the splenic vein. The portal vein is 3-8 cm long and 0.5-1.5 cm in diameter, but its size may vary depending on normal anatomical variants or pathological changes, such as those seen in portal hypertension. Pathological changes in the portal vein system are identified and considered during splenoportography and when selecting a surgical method for correcting portal hypertension. Venous outflow from the liver is through three hepatic veins: the right, middle, and left. The latter two may merge into a

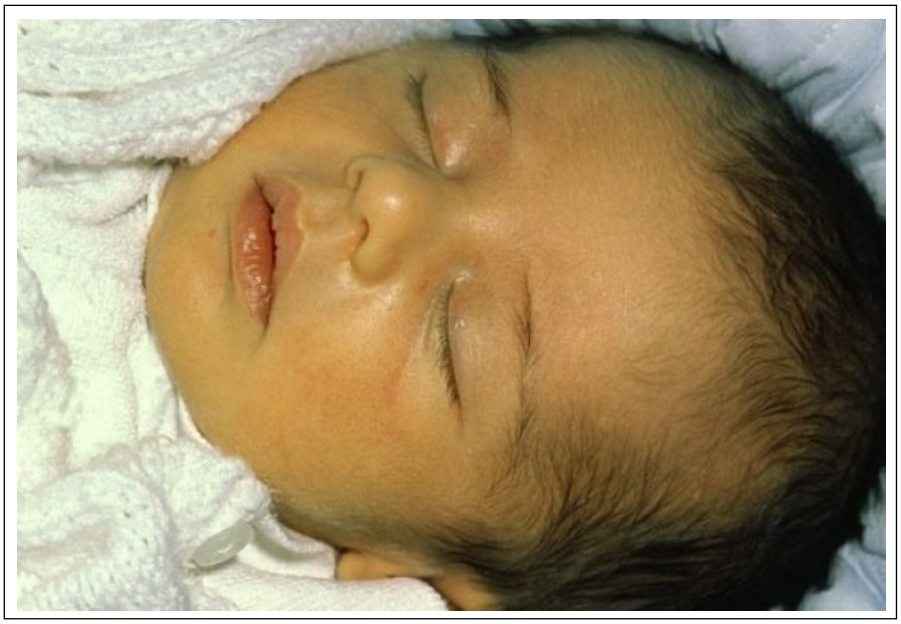


Fig. 1. Jaundice in patient K., 1.5 months old, with BA
Picture taken by the authors

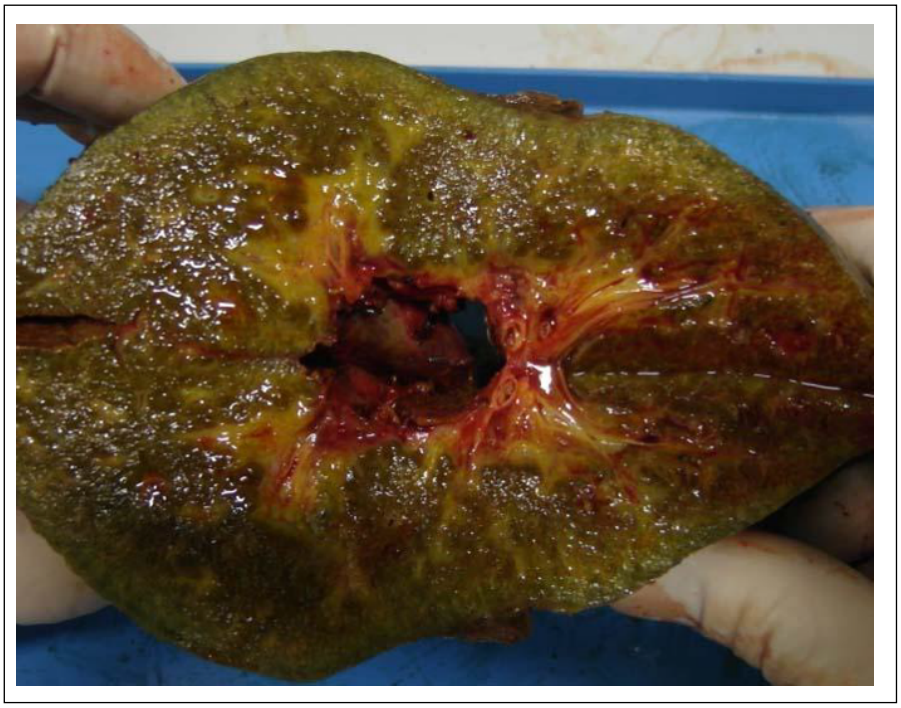


Fig. 2. Macroscopic view of the liver in BA (intraoperative picture)
Picture taken by the authors

single trunk draining into the inferior vena cava at the point where it enters the liver. Obstruction of venous outflow due to thrombosis or the presence of an embryonic membrane at the junction of the inferior vena cava and the right atrium causes severe liver dysfunction.

Lymph drainage from the liver occurs in two directions: from the superior surface of the liver, lymphatic vessels pass through the triangular ligaments and diaphragm to enter the thoracic duct [18]; and from the inferior surface of the liver, lymphatic vessels travel to the porta hepatis, to the aortic lymph nodes, and then to the thoracic duct. The porta hepatis contains lymph nodes – the cystic node and nodes of the hepatoduodenal ligament beneath the right edge of the duodenum. These nodes are consistent landmarks used to locate the cystic artery and the common bile duct [12].

Parasympathetic innervation is provided by the right vagus nerve, and sympathetic innervation comes from branches of the right splanchnic nerve. Together, branches of the vagus and sympathetic nerves form the hepatic and cystic plexuses around the structures of the porta hepatis.

CLINICAL AND ANATOMICAL OBSERVATIONS IN CASES OF BA

A notable feature in newborns is that the right and left liver lobes are approximately equal in volume, and the intrahepatic bile ducts, although formed, exhibit impaired patency due to obliteration or underdevel-

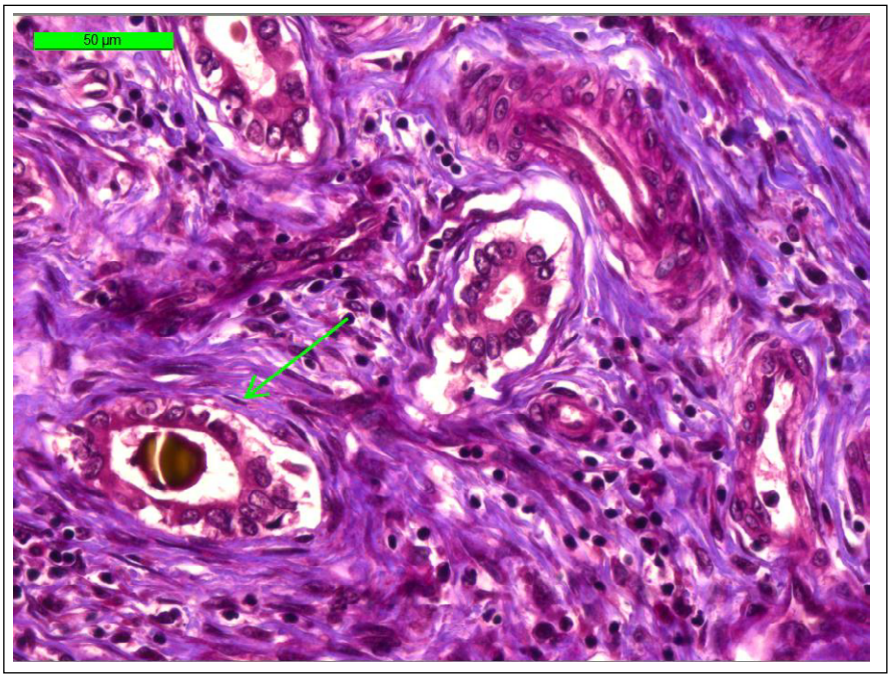


Fig. 3. Gallbladder hypoplasia in BA (intraoperative picture)
Picture taken by the authors

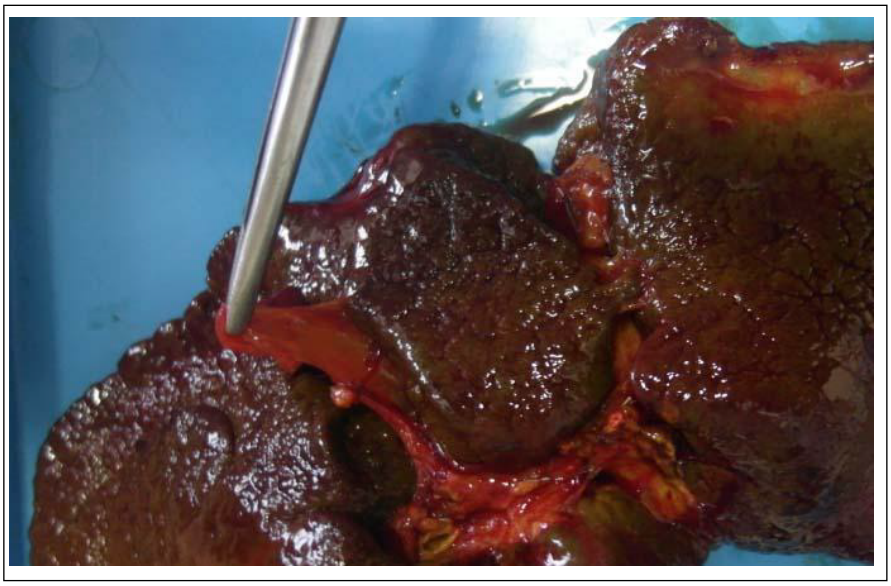


Fig. 4. Proliferation of bile ducts with edema and dystrophic changes in cholangiocytes in BA
Picture taken by the authors

opment. One possible cause of ductal obliteration is neonatal hepatitis with obstructive cholangiopathy, suggesting that atresia can develop after birth and may regress following surgery. BA is not found in embryos, stillbirths, or premature infants – it occurs only in the postnatal period [20].

Currently, several hypotheses have been proposed regarding the origin of BA, including: a viral infection hypothesis, ischemia of bile ducts due to impaired blood supply, disrupted bile acid metabolism, pancreatobiliary maljunction theory, etc.

The symptoms of BA include obstructive jaundice and acholic stools. These may not be noticeable in the first days or weeks of life because bile production begins only after birth, and clinical signs of hyperbilirubinemia require time to develop due to bilirubin accumulation in the serum. Furthermore, physiological jaundice is common and short-lived in newborns. Jaundice in BA typically appears on the 3rd-4th day after birth, does not resolve within 2-3 weeks, and progressively worsens. The direct (conjugated) bilirubin fraction in the serum may reach 100-300 µmol/L. Stools remain acholic, although in the first few days they may be yellow due to partial duct patency and the release of bile pigments from intestinal glands. Urine is dark-colored, resembling the color of dark beer (Fig. 1).

By the end of the first month, the liver begins to enlarge due to the development of cholestatic hepatitis. Intraoperatively, it appears greenish in color and is firm to the touch (Fig. 2). The liver has a yellow-green color, its surface and parenchyma are finely granular, cystic cavities near the porta hepatis contain thick, paste-like bile.

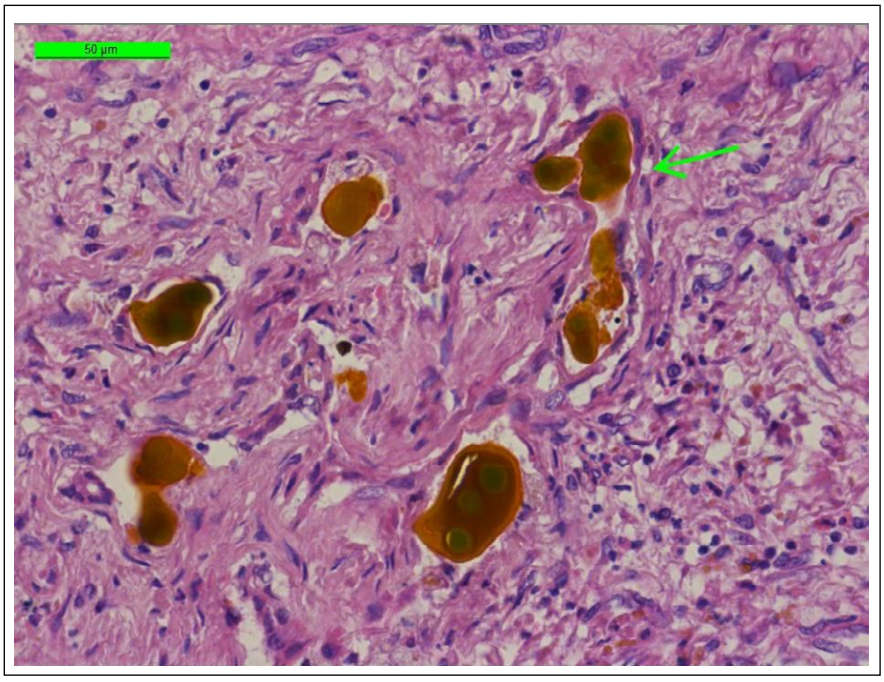


Fig. 5. Bile stasis in the bile ducts in BA
Picture taken by the authors

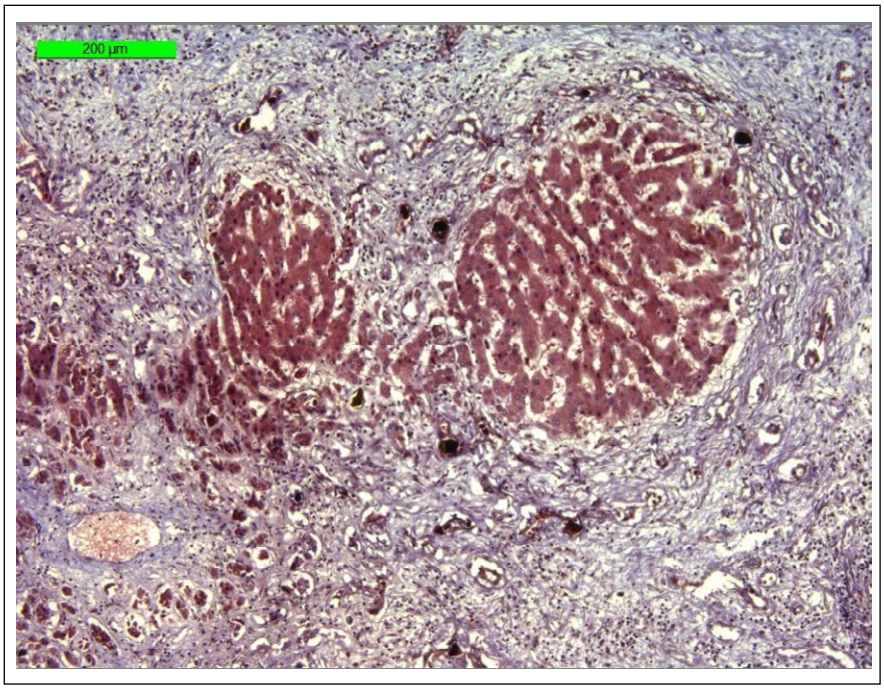


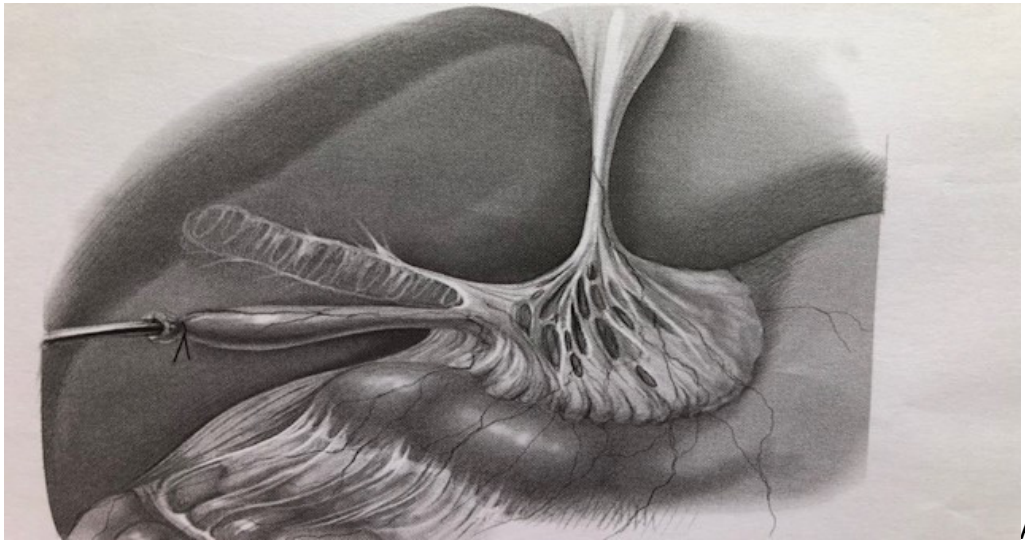
Fig. 6. Liver cirrhosis in an 8-month-old infant with BA
Picture taken by the authors

Over time, portal hypertension develops. Increased pressure in the portal vein leads to its dilation, the development of collateral circulation through the esophageal veins, and the appearance of esophageal varices. As a result of impaired blood flow through the portal vein, the spleen enlarges, and a subcutaneous venous network becomes visible on the anterior abdominal wall. Abdominal distension occurs initially due to flatulence caused by impaired digestion (due to the absence of bile), and later due to ascites from portal hypertension. The average life expectancy is 18 months. Children die from progressive liver failure and gastrointestinal bleeding due to ruptured esophageal varices [21].

Every infant with jaundice should undergo an urgent ultrasound examination. During liver ultrasound, special attention is given to the gallbladder. Since intrahepatic and total BA are more common, the gallbladder is often absent or hypoplastic, appearing as a rudimentary structure. Ultrasound may reveal a triangular cord sign at the porta hepatis, which represents fibrotic remnants of the common bile ducts [22,23] (Fig.3).

The small gallbladder does not change in size after feeding. Gallbladder contraction after feeding excludes BA.

Therefore, the presence of the following signs – persistent jaundice lasting more than one month with elevated direct bilirubin levels that do not show



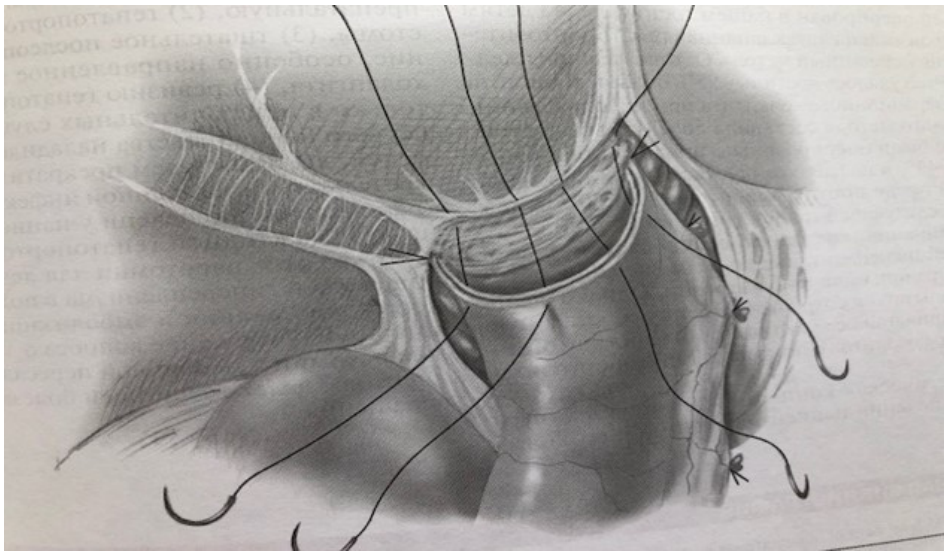


Fig. 7. Schematic representation of the Kasai procedure: a) removal of obliterated bile ducts and exposure of the fibrous cone at the porta hepatis with remnants of bile ducts; b) creation of an anastomosis between the fibrous cone and the end of the jejunum *Picture taken by the authors*

a decreasing trend, acholic (pale) stools, absence of the gallbladder on ultrasound, or a lack of response to conservative "hepatitis" treatment – constitutes an indication for diagnostic laparotomy and intraoperative evaluation of the porta hepatis to determine the type of BA and assess the possibility of surgical correction.

Liver biopsy in cases of BA can be up to 95% accurate, depending on the pathologist's qualifications [24-26]. Histological confirmation of BA is based on typical findings, among which the most characteristic are: bile duct proliferation, bile stasis in the ducts, portal edema, fibrosis, and liver cirrhosis. These histological features were also observed in the analyzed cases (Fig. 4, Fig.5, Fig.6).

The success of treatment largely depends on establishing the diagnosis within the first month of life. A diagnosis of BA made after 3-4 months of age offers almost no hope for successful treatment due to the development of cholestatic hepatitis, fibrosis, and biliary liver cirrhosis. Therefore, the 60-day threshold is considered critical worldwide for achieving a good surgical outcome. Beyond this period, surgery is typically unsuccessful. Many surgical options have been proposed, although some are now of primarily historical interest. The most widely recognized and commonly performed procedures are hepatoportoenterostomy with a Y-shaped Roux-en-Y jejunal loop according to the Kasai technique (with various modifications) and liver transplantation. The former is available in many specialized centers and is most effective when performed on time (within the first 60 days of life). It can also serve as a bridge to subsequent liver transplantation (Fig. 7).

The Kasai procedure is most effective for the treatment of BA when the following five key principles are observed:

1) Timing: the operation must be performed within the first 60 days of life. Under these conditions, bile flow can be achieved in 80% of cases. When surgery is

performed after 90 days of life, bile flow is restored in only 41% of cases.

2) Surgical technique: a) excise the fibrous cone to the appropriate depth (not reaching the liver); b) at the correct anatomical location (posterior wall of the bifurcation of the portal vein); c) use microsurgical magnification devices (surgical loupes); d) form the anastomosis with absorbable sutures, anchoring the bowel to the adventitia of the portal vein posteriorly and to the liver anteriorly.

3) Postoperative management: rational postoperative care includes long-term antibiotics, corticosteroids, and hepatoprotective agents.

4) Timely revision surgery: if bile flow stops postoperatively, a second surgery (revision of the hepatoportoenterostomy with removal of granulation tissue at the porta hepatis) should be performed no later than two months after the initial procedure.

5) Anti-reflux procedures: These should complement the hepatoportoenterostomy to prevent ascending postoperative cholangitis.

Regarding resectional procedures, in children under 1 year of age, the most extensive permissible operation is left-sided hemihepatectomy, although even this is associated with a high risk of liver failure. Notably, in newborns, the right and left liver lobes are approximately equal in volume, which must be taken into account when performing liver resections at this age. An important factor in liver transplantation is the anatomical configuration of the donor segment's blood supply and its size. The volume of the donor's remaining liver must not be less than 40% of the total liver volume [27].

CONCLUSIONS

1. BA is one of the most severe surgical conditions in newborns and infants. Without a thorough understanding of embryogenesis and the anatomical and functional features of the liver and biliary tract, effective treatment outcomes are impossible.
2. The cause of BA may lie in disruptions or arrest of the embryonic development of the bile ducts and hepatic vascular system.
3. In infants, the liver is in a state of anatomical immaturity and functional insufficiency, and its structure is not fully developed. This must be taken into account when treating children with BA.
4. The anatomical differences in the liver of children with BA have significant practical implications for the surgical management of BA.

REFERENCES

1. Hoerning A, Raub S, Dechene A et al. Diversity of disorders causing neonatal cholestasis – the experience of a tertiary pediatric center in Germany. Front Pediatr. 2014;2:65. doi: 10.3389/fped.2014.00065. DOI
2. Lee KJ, Kim JW, Moon JS, Ko JS. Epidemiology of Biliary Atresia in Korea. J. Korean Med. Sci. 2017;32(4):656-660. doi: 10.3346/jkms.2017.32.4.656. DOI
3. Ando H, Inomata Y, Iwanaka T et al. Clinical practice guidelines for Biliary Atresia in Japan – a secondary publication of the abbreviated version translated into English. J. Hepato-biliary-pancreatic Sciences. 2021;28(1):55-61. doi: 10.1002/jhbp.816. DOI
4. Khoei S, Samani PR, Fazelnia F, Pak N. Cholecysto-hepatic duct serving as the only drainage pathway of bile from the intrahepatic to the extrahepatic biliary system in an infant: a case report. BMC Pediatr. 2022;22(1):438. doi: 10.1186/s12887-022-03491-z. DOI
5. De Vries W, de Langen ZJ, Aronson DC et al. Mortality of biliary atresia in children not undergoing liver transplantation in the Netherlands. Pediatr. Transplant. 2011;15:176-183. doi: 10.1111/j.1399-3046.2010.01450.x. DOI
6. de Ville de Goyet J, Illhards T, Chardot Ch et al. Variability of care and access to transplantation for children with biliary atresia who need a liver replacement. J. Clin. Med. 2022;11(8):2142. doi: 10.3390/jcm.11082142. DOI
7. Ohi R. A history of the Kasai operation hepatic portoenterostomy for biliary atresia. World J. Surg. 1988;12: 871-874. doi: 10.1007/BF01655504. DOI
8. Arghya S, Sarma MS. Post-Kasai Portoenteristomy cholangitis, what have we learny so far?. J. Clin. Hepatol. 2025;15(3):102471. doi: 10.1016/j.jceh.2024.102471. DOI
9. Lien TH, Chang MH, Wu JF et al. Effects of the infant stool color card screening program on 5-year outcome of biliary atresia in Taiwan. Hepatol. 2011;53(1):202-208. Doi: 10.1001/hep.24023.
10. Cho SJ, Kim GE. A practical approach to the pathology of neonatal cholestatic liver disease. Semin Diagn. Pathol. 2019;36(6):375-388. doi: 10.1053/j.semdp.2019.07.004. DOI
11. Chiba T, Kasai M, Sasano N. Reconstruction of intrahepatic bile duct in congenital biliary atresia. Tohoku J. Exp. Med. 1975;115(2):99-110. doi: 10.1620/tjem.115.99. DOI
12. Witzleben CL. Extrahepatic biliary atresia : concepts of cause, diagnosis and management. Perspect. Pediatr. Pathol. 1979;5:41-62.
13. Asai A, Miethke A, Bezerra JA. Pathogenesis of biliary atresia defining biology to understand clinical phenotypes. Nat Rev Gastroenterol Hepatol. 2015;12:342-352. doi: 10.1038/nrgastro. DOI

14. Amarachintha SP, Mourya R, Ayabe H et al. Biliary organoids uncover delayed epithelial development and barrier function in biliary atresia. Hepatology. 2022;75(1):89-103. doi: 10.1002/hep.32107. DOI
15. Otte JB. Pediatric liver transplantation personal perspectives on historical achievements and future challenges. Liver Transp. 2016;22(9):1284-94. doi: 10/1002/lt.24470. DOI
16. Skandalalis JE, Gay SW. Embryology for surgeons. 2-nd edition. WB Saunders, Philadelphia. 1972, p.414.
17. Haas JE. Bile duct and liver pathology in biliary atresia. World J. Surg. 1978;2(5):561-569. doi: 10.1007/BF01556047. DOI
18. Hirsig J, Rickham P, Briner J. The importance of hepatic limph drainage in experimental biliary atresia. Effect of omentopexy on prevention of cholangitis. J. Ped. Surg. 1979;14(2):142-145. doi: 10.1016/0022-3468(79)90005-8. DOI
19. Rutkauskas S, Gedrimas V, Pundzius J et al. Clinical and anatomical basis for the classification of the structural parts of liver. Medicina(Kaunas). 2006; 42(2): 98-106.
20. Goor DA, Ebert PA. Anomalies of the biliary tree. Report of a repair of an accessory bile duct and review of the literature. Arch Surg. 1972;104(3):302-309. doi: 10.1001/archsurg.1972.04180030050012. DOI
21. Nizery L, Cardot C, Capito C et al. Biliary Atresia – clinical advances and perspectives. Clin, Res. Hepatol. Gastroenterol. 2016;4(3):281-287. doi : 10.1016/j.clinre. 2015.11.010.
22. Kanegawa K, Akasaka Y, Kitamura E et al. Sonographic diagnosis of biliary atresia in pediatric patients using the @triangular cord@ sing versus gallbladder length and contraction. AJR Am. J Roentgenol. 2003:181(5):1387-90. doi: 10.2214/ajr.181.5.1811387. DOI
23. Miyazaki N, Takami S, Uemura M et al. Impact of gallbladder hypoplasia on hilar hepatic ducts in biliary atresia. Commun Med (Lond). 2024;4(1):111. doi: 10.1038/s43856-024-00544-S. DOI
24. Chiba T. Histopathological studies on the prognosis of biliary atresia. Tohoku J. Exp. Med. 1977;122(3):249-258. doi: 10.1620/tjem.122.249. DOI
25. Lorenz G. Histomorphological liver changes due to extrahepatic biliary atresia. Dtsch Z verdau Stoffwechselkr. 1979;39(5):226- 233.
26. Chardot C. Biliary Atresia. Orphanet J Rare Dis. 2006;1:28. doi: 10/1186/1750-1172-1-28. DOI
27. Au KP, Chok KSN. Anatomical limits in living donor liver transplantation. Korean J Transplant. 2022;36(3):165-172. doi: 10.4285/kjt.22.0023. DOI

CONFLICT OF INTEREST

The Author declare no conflict of interest

CORRESPONDING AUTHOR

Olga M. Gorbatyuk
University KROK
30-32 Tabirna St., 03113 Kyiv, Ukraine
e-mail: ol.gorbatyuk@gmail.com

ORCID AND CONTRIBUTIONSHIP

Olga M. Gorbatyuk: 0000-000303970-8797 A B D E F

A – Work concept and design, B – Data collection and analysis, C – Responsibility for statistical analysis, D – Writing the article, E – Critical review, F – Final approval of the article

RECEIVED: 17.07.2025
ACCEPTED: 28.09.2025