UDC 616.361-091.8

MORPHOFUNCTIONAL CHARACTERISATION OF EXTRAHEPATIC BILIARY TRACTS OF MAMMALS

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https://doi.org/10.5281/zenodo.8332190

Abstract: A review of modern ideas about the normal and pathological anatomy and physiology of the extrahepatic biliary tract and the great papilla of the duodenum of mammals is given. Emphasis is placed on the clinical significance of changes in the physiology of the biliary system before and after surgical operations. This paper reviews and analyses the works of a number of scientists and researchers of modern times, founders and their followers on the study of morphological structure of extrahepatic biliary tracts.

Keywords: anatomy of biliary tract, physiology of biliary tract, sphincter of Oddi, surgery of biliary tract.

МОРФОФУНКЦИОНАЛЬНАЯ ХАРАКТЕРИСТИКА ВНЕПЕЧЕНОЧНЫХ ЖЕЛЧЕВЫВОДЯЩИХ ПУТЕЙ МЛЕКОПИТАЮЩИХ

Аннотация: Дан обзор современных представлений о нормальной и патологической анатомии и физиологии внепеченочных желчных путей и большого сосочка двенадцатиперстной кишки млекопитающих. Акцент сделан на клинической значимости изменений физиологии билиарной системы до и после хирургических операций. В статье рассмотрены и проанализированы работы ряда ученых и исследователей современности, основоположников и их последователей по изучению морфологического строения внепеченочных желчных путей.

Ключевые слова: анатомия желчевыводящих путей, физиология желчевыводящих путей, сфинктер Одди, хирургия желчевыводящих путей.

INTRODUCTION

The extrahepatic bile ducts (EBD) are a system connecting the liver parenchyma and duodenum, the function of which is to form the final composition of bile. The epithelium of the human common bile duct (CBD) is poorly studied, because its destruction by bile occurs within several hours after death [44; 45]. Mucous, fibrous and adventitial membranes are distinguished in the wall of the common bile duct [37]. The most detailed description of the histological structure of the ODS was given by V.Burden, 1925 [17]. The duct is covered with cylindrical epithelium. Immediately under the epithelium is the ductal stroma consisting of bundles of elastic fibres. The outer layer of the LSF is represented by loose connective tissue containing blood and lymphatic vessels. There are crypts on the surface of the mucosa of the LSL. Normal crypts of the LSL are devoid of folds, and gland ducts open at their bottom [17]. The surface of the GI tract is covered with insoluble mucus. Mucus is produced by cholangiocytes and peribiliary glands. Thus, the epithelial cover does not directly contact with bile. The appearance of glands in the form of invaginates is found in the prenatal period; acinar structure appears after the 2nd month of life.

MATERIALS AND METHODS

The product of the glands are sialo- and sulphomucins. Some glands are transformed into ectopic pancreatic acinuses, which explains the presence of insignificant concentration of

pancreatic enzymes in normal bile [55]. Heterotopia of the gastric fundal glands into the mucous membrane of the GI with the formation of a chronic ulcer has been described [2]. The number of mucous glands of the GI tract is small, with the exception of the gallbladder neck and the large duodenal papilla (LDP). Age-related proliferation of BSDC mucous glands is characteristic of the elderly [30]. In addition to biliary cells, the mucosa of the BSDC contains bocaloid cells and cells of the enterochromaffin system. Cholangiocytes constitute 3-5% of the entire cellular population of the liver, and the proportion of fluid produced by them is 40% of the total volume of bile. Choleresis is stimulated by secretin by activation of Na+-dependent transport protein, which captures conjugated bile acids from ductal bile, transports them to the cytoplasm of cholangiocytes and then returns to the portal system ("small circle of recirculation") [10]. Biliary epithelium forms the final composition of bile by secretion and absorption of various substances, first of all water, glucose and bicarbonates.

The volume of ductal resorption in humans is unknown. In a rabbit, physiological water transport through the wall of the GI wall is about 0.03 ml/hour [18]. The epithelium of bile ducts produces IgA, one of the factors preventing bacteriocholysis, its amount decreases in GI tracts [36, 38, 53]. Bile acid receptors - "farnesoid-X-receptors", regulating the synthesis of most transport proteins and enzymes involved in cholesterol and bile acids metabolism, have been found in enterocytes [32]. Consequently, it is the ductal component of bile, after cholecystectomy, that should be responsible for the magnitude of hydrostatic pressure in the GI tract and the volume of bile entering the duodenum [34]. Cholangiocytes secrete regulatory peptides, participating in tissue integration of the hepatobiliary system [49].

The intrinsic mucosal lamina of the LSL is represented by collagen fibres and scarce smooth muscle fibres, which give the inner surface of the duct a characteristic cellular appearance [57]. According to R.Henning et al., 1988 [24], the collagen fibres of the fibro-muscular layer of the FPS form a reticular structure. In case of overstretching of the LSL, at mechanical jaundice, the duct structure is irreversibly destroyed. It is not known reliably the threshold value of LVD diameter, after achievement of which restoration of tone is impossible. According to empirical data, this value is 15 mm [24]. At the same time, there are reports in the literature about the normalisation of the diameter of the LSD in case of significant dilatation of the duct [43]. In elderly people, the number of OVF capillaries decreases [4, 40]. It was found in vitro that the contraction force of a fragment of the ODD wall is no more than 3% of the contraction force of the GI wall, which corresponds to a 30-fold lower concentration of smooth muscle fibres in the wall of the ODD compared to the gallbladder [60]. Active peristalsis of the LSD has been found in rodents [24]. In ultrasound studies of healthy humans, it was found that in the interdigestive period, the diameter of the LSD decreases and increases after a meal, which confirms the presence of an active change in tone [33]. Radiological data on the existence of the Mirizzi sphincter at the confluence of the common hepatic and vesicular ducts [46] are not confirmed histologically and have long been outdated [24]. The hepatic veins are surrounded by a peribiliary vascular plexus originating from branches of the hepatic artery. Venous outflow is carried out directly into the sinusoids, which plays the role of a "fast circle of biliary-hepatic recirculation" of lipophilic substances (bile acids, cholesterol, phospholipids, hydrophobic drugs) resorbed by cholangiocytes and transported again to hepatocytes at their excessive concentration in ductal bile [10]. Two sympathetic nerve plexuses and two parasympathetic cholinergic nerve plexuses were found in extrahepatic bile ducts [59].

RESULTS AND DISCUSSION

The portion of the duodenal wall at the confluence of the GI and the main pancreatic duct (MPD), together with the muscular and glandular apparatus, is called the large duodenal papilla (LDP). Its size and shape are subject to considerable functional variation even in the same individual [50]. Soon after the introduction of fibrogastroduodenoscopy (FGDS), an endoscopic classification of the shape of the BSDC was proposed because it was found that the success of cannulation depended on the shape of this entity. Hemispherical, conical and flat forms of BSDC have been identified [29]. In the literature, anatomical variants of the so-called "overhigh" depression of the BSDC have been described, when the BSDC is located in the bulb of the duodenum or the antral part of the stomach, and bile flows directly into the stomach or massive duodenogastric reflux occurs. More than 70% of such individuals have a combination of peptic ulcer disease, duodenogastric ulcer disease and cholecystocholedocholithiasis, or a combination of gastric cancer and cholecystocholedocholithiasis [9, 25, 26]. Argyrophilic fibres are a peculiarity of the BSDC structure; as a result, the BSDC has a significant tendency to posttraumatic swelling. Blood vessels of the submucosal layer have cavernous structure and also increase the swelling of the BSDC at trauma or inflammatory process [30]. The BSDC lumen is filled with mucosa folds, the apexes of which are directed along the bile flow. These formations were first described by A. Vesalius,1543 (cited by [15]). The height of the valves is 2-6 mm and their width is 2-3 mm [1, 31]. The valves prevent duodenobiliary reflux. Obviously, after papillosphincterotomy (PST) they cannot function, and after choledochoduodenostomy (CCD) their function is irrelevant.

If the epithelium of BSDC has no differences from other regions of the biliary system, its underlying layers are organised in a unique way. Numerous tubular acinar glands are located in submucous layer. The BSDC is the site of maximum concentration of mucosal glands in the GI tract. There is no unified point of view on the normal histological structure of the BSDC in the literature. This circumstance serves as a fundamental obstacle for histological diagnostics of the majority of non-tumoural lesions of this organ. The prevailing opinion is that BSDC is characterised by a mixed glandular-muscular-vascular structure resembling the structure of the prostate gland [14, 51, 61, 62]. The opponents of the above authors [30], after thorough research, came to the directly opposite concept of "strict tissue architectonics of the BSDC", when glands and muscular apparatus do not mix in the norm. Consequently, the appearance of mucous glands in the muscular sheath of the BSDC is a pathological condition and is characteristic of age-related involution.

The discussion of morphologists of the 50-70s of the last century remained incomplete and needs to be continued [58]. Apparently, with the advent of "safe" endoscopic papillosphincterotomy in the 80s, the issue of morphological substrate of non-tumoural papillostenosis seems to be unimportant for clinicians. At the same time, complex ideas about pathological changes of BSDC, the system of indications and contraindications to PST were replaced by destruction of the closure apparatus of biliary and pancreatic tracts as a surgical access to biliary tracts for lithoextraction, regardless of the presence of pathology and preservation of the function of this important organ. Currently, the designation "sphincter of Oddi dysfunction" is accepted to denote the impaired capacity of the BSDC, especially associated with pain syndrome after cholecystectomy [21, 28, 35].

"Duodenal window" (DO), first described by H. von Luschka, 1869, and studied in detail by M. Papamiltiades & R. Rettori, 1957 [48], is a muscular diaphragm formed from the outer

longitudinal and inner circular layers of the duodenum, passing the DIO. The distance between the outer and inner slits depends on the magnitude of the angle of ductal implantation. The size of the DO in an adult is 5-7 mm [30, 54]. The oblique and spiral direction of muscle fibres originating from the muscular sheath of the duct allows the duct to move relative to the duct during intestinal peristalsis in all three coordinates. This mobility should be taken into account and used when performing duct catheterisation and endoscopic papillosphincterotomy. When performing transduodenal papillosphincteroplasty (TDPSP), as well as when performing EPST, the BSDC should be maximally protruded into the lumen of the intestine to spread these communicating fibres. This technique, developed by D. Del Valle [23], allows to avoid perforation of the posterior wall of the duodenum. The so-called antegrade papillosphincterotomy, in which the traction of the BSDC is performed in the opposite direction, has not survived the clinical trials [20]. With EPST (because it is sutureless), injury to any edge of the duodenal window results in retroperitoneal perforation of the duodenum. The duodenal window is a weak point of the duodenal wall, defects in which are the necks of parapharyngeal diverticula [12]. The prevalence of parapharyngeal diverticula (PFD) increases with age and represents a serious problem for the endoscopic treatment of choledocholithiasis [3, 42, 56].

Currently, the concept of an autonomous sphincter of Oddi (SO), i.e., not derived from the duodenal musculature, prevails. One of the main proofs of its autonomy is its development from undifferentiated mesenchyme, independent of duodenal musculature [11, 41, 52]. According to classical ideas, the CO is a closing muscle of more or less complex structure, i.e. a sphincter [16, 47, 60]. There are publications advocating a compromise view of the functional anatomy of the sphincter [27]. The competing theory of CO anatomy and physiology states that this muscular formation is an active muscle pump, and the muscular sheath of the duodenum passes onto the intramural portion of the OJP, giving the false impression of its own sphincter. At the same time, the outer intestinal muscular layer formed by longitudinal fibres is directed upwards, surrounding the wall of the intrapancreatic section of the DIC, and the fibres of the inner, circular layer are directed to the intraduodenal section of the duct [51].

"It is a single muscular apparatus," writes H. Schreiber, "having nothing in common with the sphincter described by Oddi and other older authors..., actively pumping bile into the duodenum, just as the heart pumps blood". [51]. B.S. Brikin et al. 2003 [6] came to a similar opinion in their work.

Throughout the recent history of CO function studies, including the study of the effects of various drugs on CO function, the main source of information was experimental studies on rodents. However, according to P. Calabuig [19], there is a fundamental difference of CO functions in rodents and higher mammals. In the former, the LGE is peristaltic and the CO actively transports bile to the duodenum. In higher mammals, the LGE is only able to change the wall tone; the CO functions only as a hydrodynamic resistance. It has been established that the length of the BSDC sphincter zones in humans varies from 2 to 30 mm [13,39]; therefore, PST classifications based on the length of the BSDC cut [5, 22] are of historical significance only.

CONCLUSIONS

The long pause in the study of the morphology of the terminal apparatus of the LVD, which arose after the introduction of EPST, ended at the turn of the century. Interesting works by Yu.M. Shutov, 1998, [8], C. Avisse, 2000 [12]; B.S. Briskin et al., 2003 [6], A.A. Sotnikov, 2003 [7], devoted to the mentioned problem and developing some of its aspects were published.

We could not find newer publications devoted to micromorphology of extrahepatic bile ducts in the available literature. Nowadays due to rapid development of endoscopic and laser methods of research the biliary system has become the place of the greatest use of therapeutic and diagnostic instrumental manipulations. However, the complication rate of some of these manipulations remains rather high. This in turn makes the problem of studying morphofunctional characteristics of extrahepatic biliary tracts in mammals very actual.

Literature

1. Aggarwal S., Kumar A., Roy S. et al. Massive dilatation of the commonbile duct resembling a choledochal cyst // Trop. Gastroenterol. – 2001. – Vol.22, №4. – P.219-220.
2. Alpini G., Glaser S., Baiocchi L., et al. Secretin activation of the apical Na+-dependent bile acid transporter is associated with cholehepatic shunting inrats // Hepatology. – 2005. – Vol.41, №5. – P.1037-1045.
3. Alvaro D. Biliary epithelium: a new chapter in cell biology // Ital. J. Gastroenterol. Hepatol. – 1999. – Vol.31. – P.78-83.
4. Aruin L.I., Ilchenko A.A., Chikunova B.Z. Heterotopia of the mucous membrane of the fundal part of the stomach into adenomatous polyp of the common bile duct with the formation of ulcerous defect in it // Experiment. and clinical gastroenterology. - 2004. - №5. - C.128-132.
5. Avisse C. Ampulla of Vater. Anatomic, embryologic, and surgical aspects // Surg. Clin. North. Am. – 2000. – Vol.80, №1. – P.201-212.
6. Bernard P., Le Borgne J., Dupas B., et al. Double common bile duct withectopic drainage into the stomach. Case report and review of the literature // Surg. Radiol. Anat. – 2001. – Vol.23, №4. – P.269-272.
7. Beryozov V.D., Shimkevich L.L. Internal relief of the large duodenal nipple in cholecystitis and pancreatitis //Archiv Pathologii. - 1983. - № 10.-C. 52-55.
8. Böckl O., Zimmermann G. Kombinierte radiomanometrische und histologische Untersuchungen bei Ergriffen an der Papilla Vateri // Zeitschriftfűr Gastroenterologie. – 1976. – Vol.14, №6. – S.638-644.
9. Boyden E.A. The comparative anatomy of the sphincter of Oddi in mammals, with special reference to the choledochoduodenal junction in man // The Biliary System: Oxford, 1965. – P.15-40.
10. Boyden E.A. The pars intestinalis of the common bile duct, as viewed bythe older anatomists // Anat. Rec. – 1936. Vol.66, №2. – P.217-232.
11. Briskin B.S., Titova G.P., Ektov P.V. et al. A new view on the structure of the locking mechanism of the terminal section of the common bile duct // Annals of Surgical Hepatology. - 2003. - T.8, №1. - C.63- 71.
12. Burden V.G. Observations on the histologic and pathologic anatomy of the hepatic, cystic and common bile ducts // Annals of Surgery. 1925. – Vol.82. – P.584-597.
13. Calabuig R., Weems W.A., Moody F.G. Choledochoduodenal flow: effect of the sphincter of Oddi in opossums and cats // Gastroenterology. – 1990. – Vol.99, №6. – P.1641-1646.
14. Chenderovitch J. Secretory function of the rabbit common bile duct //American Journal of Physiology. – 1972. – Vol.223, №3. – P.695-706.
15. Colp R., Doubilet J.I. Endocholidochal section of the sphincter of Oddi //Ach. Surg. –

1936. – Vol.33. – P.696-707.
16. Corazziari E. Sphincter of Oddi dysfunction // Digestive and Liver Disease. – 2003. – Vol.35, №3. – P.26-29.
17. Dehkanov T.D., Dehkanova N.T., Rakhmanova H.N. Morphology of the nodes of the ventral plexus after experimental removal of the gallbladder //ADVANCED SCIENCE XII International Scientific and Practical Conference | ICNS "Science and Education" P. 100-102.
18. Del Valle D. Papillosphinctérotomie. Indications et résultats // RévueInternationale d'Hépatologie. – 1965. – Vol.15, №5. – P.1017-1029.
19. Delmont J. Le sphincter d'Oddi: Anatomie traditionelle et anatomie fonctionelle // Gastroenterol. Clin. Biol. – 1979. – Vol.3, №2. – P.157-165.
20. Elbrond H., Ostergaard L., Hunicke B., et al. A model for simultaneousstudy of pressure and electrical events in the rabbit's sphincter of Oddi and duodenum // Scand. J. Gastroenterol. – 1988. – Vol.23, №1. – P.1211-1216.
21. Födisch H.J. Feingewebliche Studien für Orthologie und Pathologie derPapilla Vateri - Stuttgardt: G. Thieme, 1972. – 55 s.
22. Geenen J.E. New diagnostic and treatment modalities involving endo- scopic retrograde cholangiopancreatography and esophagogastroduodenoscopy. // Scand. J. Gastroenterol. – 1982. – Vol. 77. – P.93-106/
23. Germain M., Martin E., Gremillet C. Embriology of the sphincter of Oddi // The sphincter of Oddi: 3d Gastroenterological symposium. – Basel:Karger, 1977. – P.1-5.
24. Glaser S., Alvaro D., Ueno Y. et al. Gastrin reverses established cholan- giocyte proliferation and enhanced secretin-stimulated ductal secretion of BDLrats // Liver Int. – 2003. – Vol.23, №2. – P.78-88/
25. Grigoryan R.S., Starkov Yu.G. Endoscopic papillosphincterotomy at papillary diverticula // Surgery. - 2001. - №7. - C.52- 55
26. Groen A.K. Lipid transport into bile and role in bile formation // Curr. Drug Targets Immune Endocr. Metabol. Disord. – 2005. – Vol.5, №2. – P.131-135.
27. Hendrickson W.F. A study of the musculature of the entire extrahepatic biliary system, including than of the duodenal portion of the common bile ductand of the sphincter // Bull. Johns Hopkins. Hosp. – 1898. – Vol.9. – P.221- 232
28. Henning R., Steiner D., Lierse W., et al. Die Biokonstruktion der extrahepatische Gallenwege des Menschen // Aktuel. Chir. – 1988. – Vol. 23. –S.74-78.
29. Hoffmann R.M., Schwarz G., Pohl C., et al. Gallensaure-unabhängigeWirkung von Hymecromon auf die Gallesekretion und die Motilität der Gallenwege // Dtsch. Med. Wochenschr. – 2005. – Vol.130, №34-35. – S.1938-1943.
30. Hopwood D., Wood R., Milne G. The fine structure and histochemistry of human bile duct in obstruction and choledocholithiasis // J. Pathol. – 1988. –Vol.155, n.1. – P.49-59