Sys Rev Pharm 2020;11(12):1000-1006
A multifaceted review journal in the field of pharmacy

Corrective Surgery for Complete Destruction of the Hepaticocholedochus Wall and Obstructive Jaundice of Benign Genesis

Nazarbek Omarov *, Meyrbek Aimagambetov, Medet Auenov, Samatbek Abdrakhmanov , Tolkyn Bulegenov

Department of Hospital Surgery, Semey State Medical University, Semey, 103 Abay Kunanbayev str. Republic of Kazakhstan

*Corresponding author: Nazarbek Omarov, omarov.nazarbek@rambler.ru

ABSTRACT

The treatment of Mirizzi syndrome has attracted much attention from surgeons these days since the incidence of this syndrome is progressively increasing. An increase in the frequency of the disease can be associated with high progress in diagnosing the disease. With complete destruction of the hepaticocholedochus wall and the inability to form a biliodigestive anastomosis, in certain situations, the newly developed corrective surgery on the bile ducts, cholecystohepatic choledochoplasty, can increase the likelihood of a favorable outcome. Development and implementation of a new innovative corrective surgery with complete destruction of the wall of hepaticoholedochus with obstructive jaundice. Based on the clinical case, we trace the postsurgical period, which proceeded without complications; the drain tube from the subhepatic space was removed on the 4th day; fistulocholangiography was done on the 7th day; the diameter of the hepatocholedochus was not narrowed; the contrast freely entered the duodenum. The sutures were removed on the 9th day, the drain tube from the common bile duct was removed on the 11th day. The result is full recovery. As a result of our observations and work with clinical cases, we came to the conclusion that, although diagnostic measures have improved today, the issue of complex presurgical diagnosis of pathological changes in Mirizzi syndrome remains open. The imperfection of the diagnosis must be considered every time with surgery. The proposed option of the corrective operation of cholecystohepatic choledochoplasty with complete destruction of the hepatic choledochus wall expands the possibilities of surgical treatment of Mirizzi syndrome.

Keywords: gallstone disease; Mirizzi syndrome; cholecystohepatic choledochoplasty.

Correspondence:
Nazarbek Omarov
1 Department of Hospital Surgery, Semey State Medical University, Semey, 103 Abay Kunanbayev str. Republic of Kazakhstan
*Corresponding author: Nazarbek Omarov email-address: omarov.nazarbek@rambler.ru

INTRODUCTION

Mirizzi syndrome (MS) is one of the serious complications with gallstone disease, the initial morphological sign, which is compression of the proximal hepatic choledochus, culminating in either stricture formation or cholecystobiliary fistula formation. MS occurs in 0.2–5.0% of patients. From the moment of describing the first case of MS to the present day, the topic hasn't lost its relevance. More and more often MS develops as a complication of gallstone disease; this occurs when the inflammatory process spreads from the gallbladder to the bile ducts, resulting in compression or the formation of a cholecystobiliary fistula through which gallbladder calculi migrate to the main bile ducts [1, 2].

According to Alyanov [3], there has been a parallel progressive increase in the number of patients with complicated forms of cholelithiasis; such a persistent tendency persists for 5 years. With an increase in the frequency of occurrence of cholelithiasis, the number of complications such as choledocholithiasis, obstructive jaundice, cholangitis and biliary pancreatitis also increases. Among the rare complications of gallstone disease, Mirizzi syndrome occupies a special place, which develops when the inflammatory destructive process spreads from the gallbladder to the bile ducts, resulting in compression of the ducts or the formation of a cholecystobiliary fistula, through which calculi from the gallbladder migrate to the bile ducts[4]. There is a growing tendency to the progress of biliary tract surgery and an increase in the incidence of cholelithiasis [5, 6].

In 1948, Professor Mirizzi in his article "Syndromedelconducto Hepatico" described the syndrome and characterized it as biliary retention in the common hepatic duct, the reasons for which are the functional component and the anatomical background [7, 8].

In 1989, Csendes, having the experience of observing MS and proceeding from the fact that various types of the syndrome are stages of one process, he introduced his own classification based on the degree of destruction of the wall of the common hepatic duct by the cholecystocholedoheal fistula. Type I MS is a compression of the bile duct wedged into the neck of the gallbladder or cystic duct by the bile calculus. Type II MS is a cholecystobiliary fistula, resulting from erosion of the bile duct wall by means of the bile calculus, occupying no more than one third of the circumference of the bile duct. Type III MS is a cholecystobiliary fistula, occupying up to two-thirds of the circumference of the bile duct. Type IV MS is a cholecystobiliary fistula with complete destruction of the bile duct wall and completely merging with the gall bladder, forming a single structure without any recognizable landmarks between both structures of the gall tree. In 2007, Csendes added another type to its classification, which was later supported by Beltran in 2008. This classification included Mirizzi V type syndrome, which at the same time combines cholecystic-enteric fistula and another type of Mirizzi syndrome. In

this case, type Mirizzi Va syndrome a cholecystic-small intestinal fistula without gallstone obstruction of the intestine. Mirizzi Vb syndrome is a cholecystic-small intestinal fistula complicated by gallstone intestinal obstruction [9, 10].

The search for appropriate methods for diagnosing diseases of the hepato-pancreato-duodenal region has been a critical task of abdominal surgery for many years. The results of the diagnosis of some diseases are not satisfactory despite significant achievements in this area, especially in recent decades. A convincing confirmation of this is the situation regarding the MS. Presurgical diagnosis of MS is quite difficult. The clinical performance of the disease proceeds without symptoms of differential diagnostic value. Using ultrasound and computed tomography plays the role of an initial study [11, 12]. Most often, MS is diagnosed during surgery, which leads to the risk of developing a common bile duct injury [13, 14].

According to various authors, postsurgical mortality in type I Mirizzi syndrome is 4% with the presence of a vesico-choledochal fistula and a complicated course of the disease, which is up to 23% [15, 16]

The postsurgical period is characterized by the frequency of arctations in the proximal part of the common bile duct, which reaches up to 20% of all treated cases. Thus, Mirizzi syndrome is a complication of gallstone disease with a number of unresolved issues regarding the determination of the optimal treatment and diagnostic tactics in the diagnosis and surgical treatment. At the present stage of development of medical science, MS remains an urgent, little-studied problem in the field of physiology of the biliary tract [17-20].

METHODS

The results show surgical treatment of 4,012 patients with cholelithiasis (gallstone disease) treated in the clinic of the University Hospital of the Non-profit Joint-Stock Company “Semey State Medical University” (Kazakhstan) since January 2013 until August 2019.

A retrospective analysis of case histories of patients with calculous cholecystitis was carried out; 1,703 (42.4%) were admitted as planned for surgical treatment; 2,309 (57.6%) patients were hospitalized according to emergency indications. Mirizzi syndrome was found in 120 patients, which in relation to the number of patients with calculous cholecystitis is 3%.

According to the classification of Csendes (2007), I, II, III, IV, and Vtype were diagnosed among patients with MS. A number of 29 (24.2%) patients were hospitalized on a planned basis and 91 (75.8%) according to emergency indications. During the surgery, type I MS was detected in 60 (50%) patients, type II MS in 24 (20%), type III in 18 (15%), type IV in 12 (10%), type V in 4 (3.3%), Vb type in 2 (1.7%) patients.

There were 35 men (29.2%) and 85 (70.8%) women. The age of patients ranged from 32 to 90 years, averaging 63.8±5.6 years. Patients were admitted with symptoms of obstructive jaundice – 49 (40.8%) patients, without obstructive jaundice – 71 (59.2%). The duration of obstructive jaundice in patients ranged from 3 to 24 days.

We described a case of Mirizzi type IV syndrome, where hepatic choledochus wall was completely destroyed to the mouth of the right and left hepatic ducts.

Medical history

Patient V., 78 years old, was admitted to the surgical department on an emergency basis diagnosed with an acute phlegmonous calculous cholecystitis. Cholecystohepatic choledochio fistula – Mirizzi syndrome type IV. Obstructive jaundice. Purulent cholangitis. Biliary cirrhosis. Hepatic insufficiency, decompensated form. Perivisical infiltrate. III degree arterial hypertension, IV risk. XCH – 1. Chronic bronchitis. Chronic pancreatitis, exacerbation. Erosive esophagitis. Focal atrophic gastritis.

On admission the patient complained of periodic pain in the right hypochondrium with radiation to the back, nausea, vomiting, yellowness, dry mouth, fever up to 38.5°C. She became acutely ill when pain occurred in the right hypochondrium, nausea, vomiting, dry mouth, and an increase in body temperature to 38.5°C on April 25, 2018. 3 days after the stroke, yellowness of the skin and sclera appeared. Over time, the pain intensified. Due to the intensification of the pain syndrome, she turned to the emergency clinic of the University Hospital NAO Semey State Medical University on 03.05.2018. She examined by surgeons and admitted in the surgical department of the University Hospital NAO "Semey State Medical University".

After examination, the general condition of the patient turned out to be extremely acute. Consciousness is inhibited. The skin and visible mucous membranes are clearly icteric. Lymph nodes are not enlarged. Subcutaneous fat is moderately developed. The bone-articular and muscular system are without visible pathology. Free breathing through the nose. The breast is pineal. Extensive pulmonary sound is by percussion above the lungs. Auscultatory vesicular respiration over the lungs without wheezing. RR 24 min. There is no visible pulsation in the heart and large vessels area. Auscultatory heart sounds are muffled, no noises, sinus heart rhythm. Heart rate 96 beats per min., arterial blood pressure 150/90 mmHg. Pulsed peripheral arteries of satisfactory properties. The tongue is dry and covered with a brownish coating. Free swallowing. The abdomen is of the correct form and symmetrical, is uneven in breathing. Palpatory of the abdomen is soft, painful right hypochondrium. Positive symptoms: Ortner, Kehr, Murphy. Shyotkin-Blumberg sign is weakly positive. The kidneys are not palpable. Urination is free and painless. The urine is dark in color. The symptom of "striking" is negative on both sides. The bowel movement is light in color.

Laboratory and instrumental studies:

Blood type as of 05/03/2018 is A (II), positive Rh (+).

General blood test as of 05/03/2018:

Hemoglobin – 119g/l;

erythrocytes – 4.8x1012/l;

MCH – 1.0;

leukocytes – 19.4x109/l;

hematocrit – 34%;

stab – 7%;

mcroxyphil – 80%;

monocytes – 3%;

lymphocytes – 10%;

ESR – 67 mm/h.

Simple urine test as of 05/03/2018:

Color – light yellow;

transparency – transparent;

relative density – 1017;

the reaction is acidic;

protein – 0.066 g/l;

leucocytes – 3-2-2 visual field;

mucus +.

Biochemical blood test as of 05/03/2018:

BUN – 6.4 mmol/l;

glucose – 5.8 mmol/l;

total bilirubin – 224.0 mmol/l;

direct bilirubin – 164.4 mmol/l;

indirect bilirubin – 59.6 mmol/l;

total amylase – 40.6 g starch/h;

residual nitrogen – 34.7 mmol/l.

Coagulogram as of 05/03/2018:

International normalized ratio – 2.4;

fibrinogen – 4.17;

thrombotest – VI;

partial thromboplastin time – 35.6;

thrombin time – 13.2;

solvable fibrin-monomeric complexes – 4.0.

Stool ova and parasites test as of 05/06/2018. *Report:* not detected.

EIA blood anti-HIV test as of 07.05.2018. *Report:* negative.

EIA for HbsAg and HCV test as of 09.05.2018. *Report:* negative.

Blood test for microreaction as of 10.05.2018. *Report:* negative.

Ultrasound examination of the abdominal cavity as of 05/03/2018:

Liver: the right lobe is 161x88 mm, the left is 82x74 mm, the contours are even, the echostructure is medium-grained, moderately diffusely inhomogeneous; the echo density is increased. Intrahepatic ducts are expanded to 4 mm.

Gall bladder: deformed shape, dimensions 67x38 mm. The wall of the bladder is thickened and even. There is a large fixed calculus of 60 mm in the cavity of the bladder in the neck area, with a wide acoustic path. Common bile duct is 10.0mm.

Pancreas is 32x21x29 mm; contours are fuzzy; the echostructure is heterogeneous; echo density is increased. Spleen is 134x48mm. The spleen contour is smooth, the echostructure is fine-grained.

Report: Calculous cholecystitis. Obstructive jaundice. Diffuse changes in the parenchyma of the liver, pancreas. Hepatosplenomegaly.

Plain radiography of the thoracic organs as of 05/03/2018 *Report*: Signs of chronic bronchitis.

Electrocardiography as of 05/03/2018. Sinus rhythm, heart rate is 96 beats per min. The horizontal position of the electrical cardiac axis. Supraventricular extrasystole is registered. Scars in the posterior wall of the left ventricle. Myocardial ischemia.

Fibrogastroduodenoscopy as of 05/04/2018 *Report*: Erosive esophagitis. Focal atrophic gastritis.

Therapist's examination as of 05/03/2018. Diagnosis: III-degree arterial hypertension, risk IV. XCH – I. Acute calculous cholecystitis. Choledocholithiasis. Obstructive jaundice. Liver decompensation form.

The patient was operated on after presurgical preparation. The following has been performed: an upper-mid laparotomy, atypical cholecystectomy with compilation of the gallbladder walls, fibro-choledochoscopy, cholecystohepatic choledochoplasty with U-shaped interrupted sutures by Vishnevsky drain, as well as abdominal drain. Following laparotomy, a universal retractor of Aimagambetova was installed in the wound, developed in the clinic (patent of the Republic of Kazakhstan No. 90060).

During the inspection: the liver with rounded edges, dense, of dark greenish color. A liver biopsy was drawn. The projection of the gallbladder is a dense infiltrate formed by the lock of the greater omentum, the hepatic angle of the colon, the stomach, the duodenum, and the liver. Adhesions are phased by a coagulator.

At the same time, a huge calculus with dimensions of 60x50x45mm is palpated in the cavity. The hepatoduodenal ligament does not differentiate. The bladder was opened after taking on the holders, the calculus was removed, while purulent bile with fibrin was released from the bile ducts. The cavity is washed. An atypical cholecystectomy was performed with the anterior, posterior and lateral walls of the gallbladder remaining, while maintaining the blood supply to the cystic artery (Fig. 1).

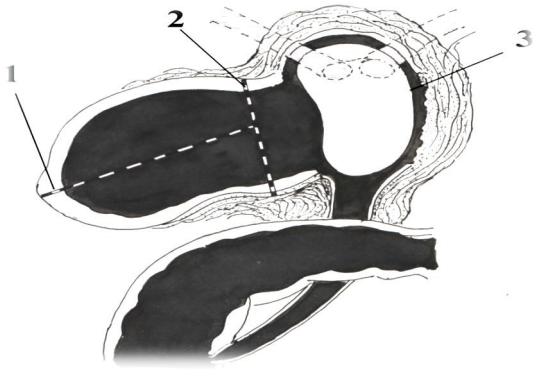

Figure 1. Atypical cholecystectomy, scheme of the line of opening of the gallbladder (1) and cut-off line (2), obturating calculus (3)

Upon examination it is determined that the common bile duct wall is destroyed by a length of 85% (80mm) (Fig. 2); it is determined that bifurcation of the mouth of the right and left ducts, and the mouth of the bile duct is 8mm distally (Fig. 3).

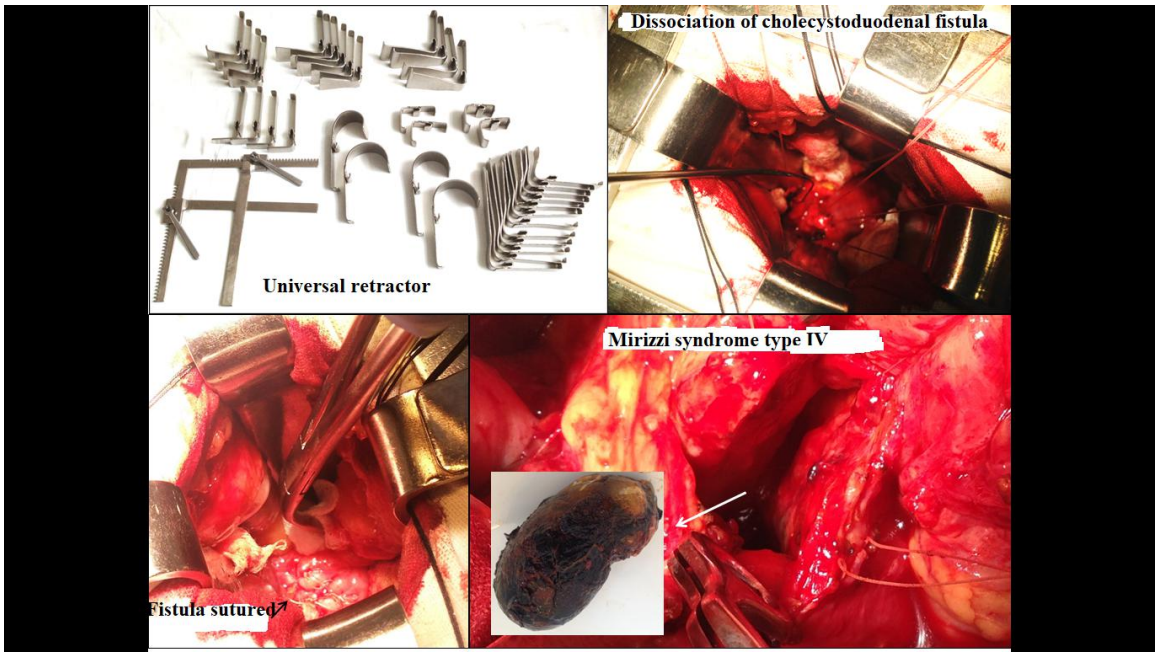

Figure 2. Atypical cholecystectomy, opening the gallbladder. In the lumen of the gallbladder obstructing hepatic choledochus calculus

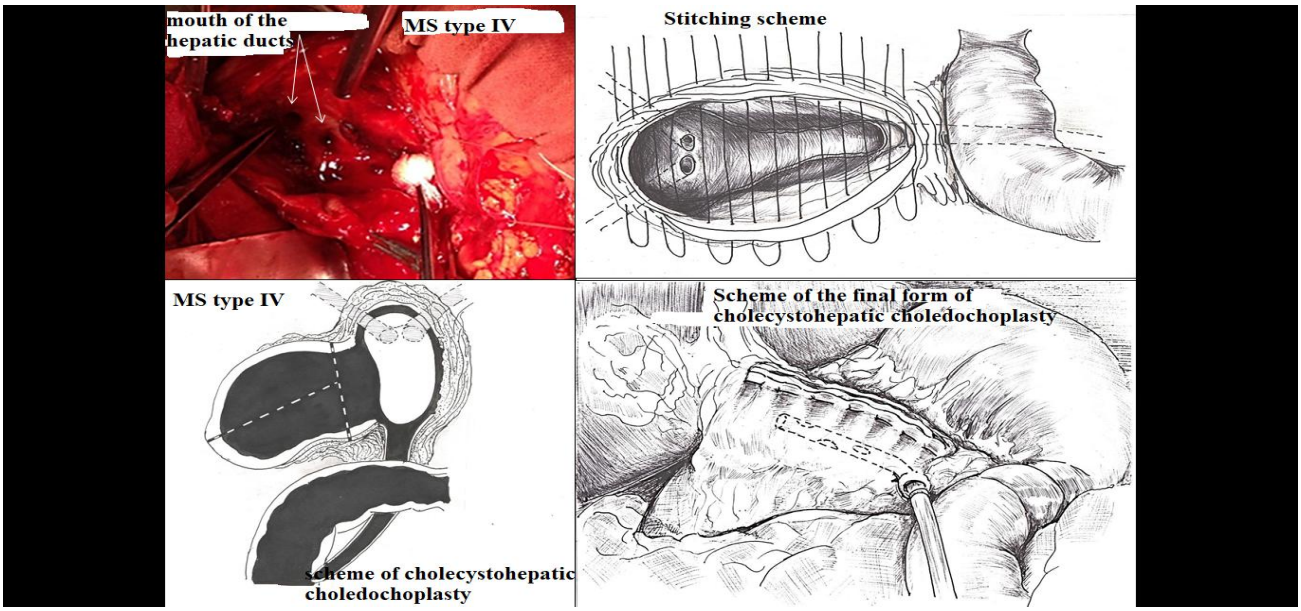

Figure 3. The complete destruction of the bile duct wall – to the mouth of the right (1) and left (2) hepatic ducts

Fibrocholechoscopy was performed using a guide developed in the clinic through the fistulous opening in the proximal direction: purulent bile with fibrin in the lumens of the right and left hepatic ducts and segmental ducts, the ducts were washed with an antiseptic and physiological saline solution. The distal segment of the common bile duct is free, calculi are not determined; the major duodenal papilla is passable. It was decided to perform cholecystohepatic choledochoplasty (patent of the Republic of Kazakhstan No. 1077801) with the remaining sections of the walls of the gallbladder with U-shaped interrupted stitches with lavsan 3-0, on the Vishnevskiy drain (Fig. 4), located below the plasty site. The hermeticism check by rinsing through the drain, the joints are well-grounded, fluid intake is not observed.

Cholangiography: contrast freely enters the duodenum, 12mm diameter of hepatic choledochus; no contrast leakage outside the duct. Hemostasis was performed. Number of wipes. Subhepatic space drain. Drainage from the formed hepatic choledochus is removed through a separate wound in the right hypochondrium. The universal retractor is removed from the wound. Layer-by-layer sutures on the wound. Aseptic dressing. Blood loss is 100 ml.

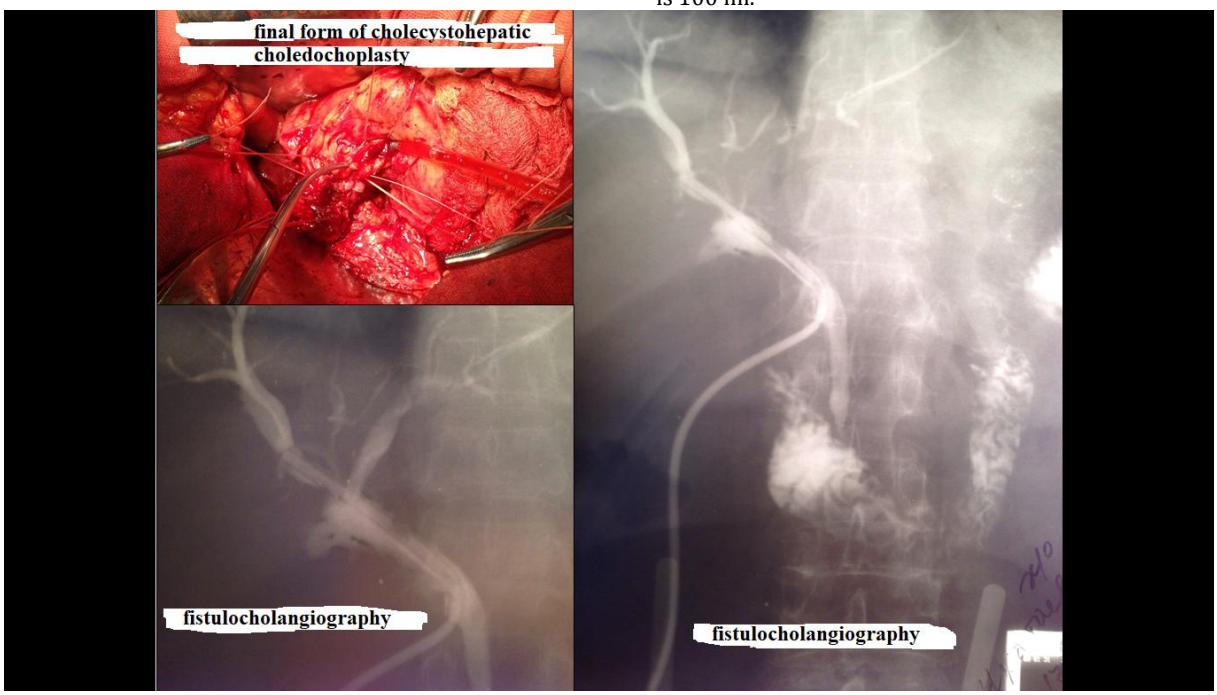

Figure 4. Plastic surgery of hepatic choledoch using the remainder of the gallbladder with U-shaped sutures and drain of hepatic ductus hepaticocholedochus according to Vishnevskiy and postsurgical fistulocholangiogram: the diameter of the hepatic choledochus is not narrowed, the contrast freely enters the duodenum.

RESULTS

The postsurgical period had no complications; the drain tube from the subhepatic space was removed on the 4th day. Fistulocholangiography was performed on the 7th day, the diameter of the hepatic choledochus was not narrowed, the contrast freely entered the duodenum. The sutures were removed on the 9th day, the drain tube from the choledochus was removed on the 11th day. The outcome is full recovery.

Figures of a method to eliminate a defect in the wall of ductus hepaticocholedochus to the proposed methodology of the University Hospital NAO "Semey State Medical University".

The study was within the framework of ethical standards and didn't violate the rights of patients. Participation in the study was voluntary. Written consent was obtained from patients to collect, process personal information, diagnostic data, photographs and treatment outcomes, while maintaining anonymity.

DISCUSSION

In the works of Batvinkov [21, 22], with type IV Mirizzi syndrome with complete destruction of the walls of the hepatic choledoch from confluence to the retroduodenal part of the common bile duct, he suggests that the operation be completed by transpapillary drain of the bile ducts. For this purpose, the author used a tube from a blood transfusion system, one end of which was drawn into the left lobar duct, and the other through the distal choledoch into the lumen of the duodenum after transduodenal papillosphincterotomy. The tube was fixed and hermetically sealed at the level of the merger of the lobar ducts. Moreover, it was covered with patches of scar-altered tissues of the hepatoduodenal ligament.

Halperin [5] in case of hepatic choledochal fistula (CMIV type), he carried out the drain of the common bile duct with a T-shaped drainage brought out below the defect and suturing of the bile duct defect with the remains of the gallbladder wall with a nodal suture above the drainage.

Wolf [23] in the case of CMIV type offer to perform a hepatopathyunoanastomosis with a jejunum loop disconnected along the Roux limb drains in both lobar ducts with the second ends of these drains to the anterior

abdominal wall through the jejunum sutured to the parietal peritoneum.

With complete destruction of the wall of the hapatic choledochus, the formation of hepaticoyunoanastamosis is not always possible due to inflammatory infiltrate, which does not make it possible to technically isolate tissues without damaging the surrounding tubular structures. The closure of the hepaticoholedoch defect with gallbladder tissue residues on the T-shaped drainage leads to the formation of strictures and the need for repeated interventions. Hepaticojejunostomiaon "frame" drainages carried out into the lobar ducts, with expressed infiltrative tissue changes, presents a great risk of damage to the branches of the hepatic artery and portal vein during the formation of anastomosis. The frame drainage must be changed every 3 months with frequent washing and keep it drained for a long time.

Therefore, we offer the following method of cholecystohepatic choledochoplasty for type IV MS and the presence of pronounced inflammatory changes in the tissues of the operating area. The proposed method of cholecystohepatic choledochoplasty allows to restore the outflow of bile into the duodenum, to maintain blood supply to the cystic artery, to exclude the use of hepaticojejunostomy on the frame drainage, to prevent the development of suture and stricture hepatic choledochitis and hepatic choledochastomyosis, which improves the long-term results of treatment of patients with cholecystohepatic choledochheal fistula type IV. The developed technique leads to the recovery of patients, returning them to a full life and work, and can be recommended in the practice of biliary surgery. This method of surgical intervention was applied to 10 patients with type IV MS with good results.

CONLCUSION

The method of cholecystohepatic choledochoplasty is technically simple to implement; it prevents the development of insolvency of sutures, corrosive strictures, the duration of the use of frame drainages characteristic of traditional surgical interventions, while preserving the natural passage of bile into the duodenum.

AKNOWLEDGEMENTS

The authors gratitude the Director of the Public State Enterprise at the REM “Pavlodar Regional Hospital named after Sultanova” Syzdykov Serym for his help with collecting material when writing an article.

CONFLICT OF INTEREST

The authors confirm that there are no conflicts of interest.

REFERENCES

1. Amin A, Zhurov Y, Ibrahim G, Maffei A, Giannone J, Cerabona T, Kaul A. Combined Endoscopic and Laparoscopic Management of Postcholecystectomy Mirizzi Syndrome from a Remnant Cystic Duct Stone. Case reports in surgery. 2016.

2. Piccinni G, Sciusco A, de Luca GM, Gurrado A, Pasculli A, Testini M. Minimally invasive treatment of Mirizzi's syndrome: is there a safe way? Report of a case series. Annals of Hepatology. 2014; 13(5): 558-564.

3. Alyanov AL, Mamoshin AV, Borsukov AV, Muradyan VF. Efficacy of miniimally invasive technologies in the treatment of patients with the syndrome of obstructive jaundice. Scholarly notes of Orlov State University. Series: Natural, technical and medical sciences. 2015; 4(67): 280-284.

4. Kohanenko NYu, Pavelets KV, Glebova AB, Koryakina TV. The Selection of Surgical Tactics in Patients with Mirizzi Syndrome Using MRI Diagnostics. Bulletin of St. Petersburg University. Series: 11. Medicine. 2014; 2: 130-137.

5. Halperin EI. The Severity Classification of Mechanical Jaundice. Surgical Hepatology Analysis. 2012; 2: 26-33.

6. Styazhkina SN, Isteyeva AR, Korotkova KA, Sakhabutdinova DR, Khasanova GF. The Actual Problems of Mechanical Jaundice in Surgery. International Journal applied and basic research. 2016; 7-3: 427-430.

7. Dadhwal US, Kumar V. Benign bile duct strictures. Medical journal armed forces India. 2012; 68: 299-303.

8. Shishkin AA, Postrelov NA, Plotnikov YuV. Clinical Issues of The Extreme Course of Mirizzi Syndrome. Experimental and Clinical Gastroenterology. 2014; 8(108).

9. Beltrán MA. Mirizzi syndrome: history, current knowledge and proposal of a simplified classification. World Journal of Gastroenterology: WJG. 2012; 18(34): 4639–4650.

10. Unadkat P, Varma R, Gaikwad K, Halgaonkar P, Vaja C. Mirizzi Syndrome: A Case Report. IJSS. 2016; 2(10): 42.

11. Kurbaniyazov ZB, Makhmudov TB, Sulaymonov SU, Davlatov SS. The Surgical Treatment in Patients with Mirizzi Syndrome. Graduate Doctor. 2012; 51(2.1): 135-138.

12. Ratchik VM, Prolom NV, Orlovsky DV, Burenko AN. The Tactics and Surgical Treatment of Obstructive Jaundice in Various Etiologies. Gastroenterology. 2014; 54(4): 81-87.

13. Tamm TI, Mamontov IN, Kramarenko KA. The Features of Diagnosis and Treatment of Mirizzi Syndrome. Kharkivska hirucy shkola. 2016; 3 (78): 44-47.

14. Wani N, Khan N, Shah A., Khan A. Post-cholecystectomy Mirizzi’s syndrome: magnetic resonance cholangiopancreatography demonstration. Saudi Journal of Gastroenterology. 2010; 16 (4): 295-298.

15. Lee KF, Chong CN, Ma KW. Cheung E, Wong J, Cheung S, Lai P. A minimally invasive strategy for Mirizzi syndrome: the combined endoscopic and robotic approach. Surgical Endoscopy and Other Interventional Techniques. 2014; 28(9): 2690-2694.

16. Shiryaev YN, Glebova AV, Koryakina TV, Kokhanenko NY. Acute acalculous cholecystitis complicated by MRCP-confirmed Mirizzi syndrome: a case report.

International Journal of Surgery Case Reports. 2012; 3(5): 193-195. DOI: 10.1016/j.ijscr.2011.11.006.

17. Lacerda PS, Ruiz MR, Melo A, Guimaraes LS, Silva-Junior RA, Nakajama GS. Mirizzi syndrome: a surgical challenge, Arquivos Brasileiros de Cirurgia Digestiva. 2014; 27(3): 226.

18. Siplivy VA, Evtushenko DV, Petrenko GD, Andreschev SA, Evtushenko AV. Diagnosis and Surgical Treatment of Mirizzi Syndrome. ClinicalSurgery. 2016; 8: 8-11.

19. Batvinkov NI, Kuchta A, Rusin IV, Chapelle IA, Vasilevsky VP. The diagnosis and surgical treatment of the syndrome Mirizi. Surgery: Eastern Europe, 2012; 1: 21-27.

20. Isayeva GS. The state of coronary arteries in perimenopausal women with chest pain. J Clin Med Res. J Clin Med Res. 2014; 6(6): 451-455.

21. Hrechanina O, Isayeva G, Kolesnikova O, Isakova Y. Relations between familial hypercholesterolemia and early ichemic heart disease: an analysis of medical documentation data. Serbian Journal of Experimental and Clinical Research. 2019. DOI: 10.2478/sjecr-2019-0056

22. Koneva ES, Lyadov K.V, Shapovalenko TV, Zhukova EV, Polushkin VG. The hardware techniques for the restoration of the gait stereotype in the patients following total hip replacement: the personalized approach. Problems of balneology, physiotherapy, and exercise therapy. 2018; 1: 26-34. DOI: 10.17116/kurort201895126-34

23. Volk LL, Kirdan MV, Solonets KV. Mirizzy's syndrome: a case from practice. Smolensk Medical Almanac. 2017; 1: 74-77.