BILIARY SURGICAL PATHOLOGY
Part III

DANA VULCU, M. DEAC

„Lucian Blaga" University of Sibiu

Abstract: *„Post biliary surgery syndrome" combines the signs and symptoms which appear after the surgery, involving the gall bladder and/or the biliary ducts for biliary lithiasis. They occur after surgery and have multiple causes.*
Keywords: *residual stenosis of the sphincter of Oddi, dyskinesia of the sphincter of Oddi, non-functional biliodigestive anastomosis*
Rezumat: *„Sindromul biliarilor operaţi" reuneşte semnele şi simptomele care apar în urma intervenţiei chirurgicale pe veziculă şi/sau căile biliare pentru litiază. Acestea se manifestă la intervale variabile de timp de actul operator şi au cauze multiple.*
Cuvinte cheie*: stenoză oddiană reziduală, dischinezie oddiană, anastomoză bilio – digestivă nefuncţională*

2.4 Residual stenosis of the sphincter of Oddi

Frequently, stenosis of the sphincter of Oddi remains unknown, on the occasion of cholecystectomy, when the importance of exploring the terminal common bile duct is reduced at minimum and when the weight of the vesicular lesions is overestimated. Stenosis of the sphincter of Oddi is present in 17- 60% of the cases (4,8,12,13).

In the situations in which choledochotomy has been practiced upon the first surgical intervention, stenosis of the sphincter of Oddi, either remained unknown (calculi extraction was made and not the papilla diagnosis), or it was diagnosticated and considered reversible.

Residual stenosis of the sphincter of Oddi also includes the stenosis that occurs as a result of the sectioning the sphincter (sphincterotomy, sphincteroplasty).

The differential diagnosis between reversible stenosis of the sphincter of Oddi and the evolutive stenosis on the moment of the primary surgery is not possible, due to the fact that the lesion may seem reversible initially, brought about the edema (upon cholangiography, the oddian pass is repermeabilized after the antispastics treatment – regarding the instrumental exploration, the sounds force the passing through papilla), its further evolution is materialized in stenosis (4,9).

Clinical manifestations

Regarding the residual stenosis of the sphincter of Oddi, the clinical manifestations occur relatively early, in the first two years. Many forms have been described:

- The form with pain, typical for biliary colic, identical with the pre-surgery period of time;
- The form with persistent pain in the right hypocondrium;
- The pure angiocholitis form.

The increased precocity and intensity of the clinical manifestations are explained by the fact that, through cholecystectomy, the reservoir and the natural damper – the biliary vesicle – is suppressed, the hydrodynamic phenomena being aggravated, as well as the persistent dystonia (4).

Laboratory and paraclinical investigations

The usual laboratory investigations have a limited utility in establishing the pre-surgical diagnosis of residual stenosis of the sphincter of Oddi.

- Intravenous cholangiography and the biliary echography emphasize the indirect signs of inferior choledocian obstacle. The dilatation of the biliary duct may exist before the primary surgery and for this reason, its value is disputable in the residual oddian diagnosis. These two methods may detect the residual calculi, in exchange.
- Biliary scintigraphy, with 99 mTc, used in the residual stenosis of the sphincter of Oddi gives positive results when it emphasizes a dilated hepato-common bile duct, while the release time of the radiotracer in the duodenum is extended.
- Decisive for the diagnosis of residual stenosis of the sphincter of Oddi is the retrograded endoscopic cholangiography, accompanied by the endoscopic biliary manometry.
- Intra-surgical cholangiography emphasizes the presence of the oddian obstacle and the association of the sphincter with relaxing drugs prove the functional nature of the obstacle (spasm). If the contrast dye is not released in the duodenum, the nature of the obstacle is not organic.
- Intra-surgical diagnosis sound of papilla may establish if the lesion is permeable or not. If the

oddian passage cannot be sounded, the lesion is irreversible.

Treatment of the residual stenosis of the sphincter of Oddi

Residual stenosis of the sphincter of Oddi, associated to residual lithiasis and long cystic stump, benefit from surgical treatment, consisting in sphincterotomy or in a transduodenal or transcholedocian (rarely) sphincteroplasty.

Lately, the classic surgical procedures were replaced with the endoscopic sphincterotomy that, besides the intervention on the papilla, it also gives the possibility of extracting the calculi and accomplishing the retrograded cholangiography.

When the diameter of the main biliary duct exceeds 20 mm, biliodigestive anastomoses are practiced (choledocoduodenal, or hepatic-jejunal).

Consequences of sphincterotomy

Suppressing the function of sphincter involves three main consequences:

- The accomplishment of a continuous biliary flow in the intestine, bringing about the increase of the entero-hepatic circuit speed of the biliary acids (the contents of the bile within the biliary acids is increasing; there is a frequent and prolonged contact of the biliary acids with the intestinal mucosa, with the colonic anaerobe flora and the increase of the level of the secondary biliary acids, involved in the colon cancer occurrence). Hormonal alterations after cholecystectomy lead to perturbations of the pyloro-duodenal motricity and to the occurrence of the duodeno-gastric reflux, resulting in biliary gastritis.
- The duodeno-choledocian reflux is harmful when it is not equalled by the deflux. After sphincterotomy, the resistant fibres do not oppose the duodenal reflux; angiocholitis is more frequent.
- Common bile duct dysfunction.

Late complications of sphincterotomy

- Angiocholitis occurs when the bile reflux exceeds the deflux. Angiocholitis has three main causes:
- Angiocholitis through mechanical stasis due to an organic obstacle: residual calculi, restenosation of papilla after sphincterotomy or after sphincteroplasty (rarely), chronic pancreatitis, proximal jejunal stenosis (4,6).
- Angiocholitis through functional stasis, secondarily to the main biliary atony or to duodenal dyskinesia (dilatation of the main biliary duct is more than 20 mm or atone choledoc, when the orifice of the choledoco-duodenal communication is too small, in relation to the main biliary duct lumen, when there is a spasm of the duodenal sphincters) (4).
- Intra-hepatic angiocholitis that maintains the biliary infection in descending order.
- Restenosation of the papilla occurs after oddian sphincterotomy, a frequent situation in the cases in which the length of the incision was below 10 mm. In the case of sphincteroplasty, restenosation is excluded, the suture of the incision sides being healed per primam (4,6).
- The hepatic sufferance (hepatic abscess) occurs rarely, in the cases in which papilla restenosation is followed by angiocholitis.

The research showed that the rate of complications is much reduced after sphincteroplasty than after sphincterotomy.

2.5 Dyskinesia of the sphincter of Oddi

The sub-layer of the so-called post-cholecystectomy syndrome is first of all represented by after surgery adaptation disorders, resulting in hyper or hypotonia dyskinesia. Generally, these disorders are of small or mild intensity and ends with a food diet and drug treatment. If they persist, they receive the character of the sufferance called “post-cholecystectomy syndrome” (PRIBRAM).

- Hypertonia of the sphincter of Oddi consists in the permanent contraction of the sphincter of Oddi, brought about the organic manifestations (stenosis), or by a motor sphincterian disorder (oddian dyskinesia) (1). As a result of the increase of the shincterian tonus, the bile flow is hindered, what will bring to the mild dilatation of the main biliary duct (4). Hypertonia occurs in 1- 10% of the patients with postcholecystic syndrome. Frequently, the cause of hypertonia is represented by cholecystectomy, due to the lesions of the sphincter through calculi passage or due to the suppression of the vesicular reservoir, the oddian spasm being an adaptive reaction.

Clinically, oddian hypertonia is manifested through collective, persistent pains, localised at the level of the right hypocondrium, epigastrium, or para-umbilically with posterior irradiation in the right shoulder. Periodically, the pains are accompanied by nausea, vomiting or discrete icterus.

The laboratory diagnosis offers modest data. The cholestasis tests, increases of bilirubin and transaminases may be modified,

- Retrograded endoscopic cholangiography emphasizes the dilated main biliary duct and the prolonged filiform evacuation in the lower area (“in haemolysis tube”). The difficulty in tabulating the papilla, the dilatation of the biliary duct over 12 mm and the release delay with more than 45 minutes represent differentiation criteria of the papilla stenosis from the hypertone oddian dyskinesia.
- Endoscopic manometry is considered the golden standard in the diagnosis of the oddian dyskinesia (with the help of a micro-catheter of 1,7 mm). In oddian hypertonia, the basal pressure of the sphincter exceeds the duodenal pressure with 40 mmHg. Also, the reduction of the basal pressure after the administration of cholecystokinin, glucagons or amyl nitrite inhalation indicates hypertonia and not oddian stenosis. Manometry indicates hypertonia due to the

increase of the phase waves, retrogradely propagated with more than 50% or, due to the presence of an increased phase activity (phase contractions more than 4/ minute) (7,16).

- Morphine - prostigmine provoking tests bring about spasms of the sphincter, followed by colic and the increase of the cholestasis enzymes.
- Hepatobiliary sequential scintigraphy with 99 mTc emphasises the duodenal release delay of the radiotracer and it may be combined with a fat lunch or with morphine and prostigmin administration (1,3,7).

The treatment consists in taking dietetic and hygienic measures, allowing the consumption of fats which are not thermically processed and have relaxing effect for the sphincter of Oddi. The administration of miorelaxing drugs, anticholinergics, calcium channels blockers also has favourable effects.

Endoscopic sphincterotomy is recommended in the patients with postcholecystectomy hypertonia and long term symptoms and with increased basal pressure of the sphincter (1,10,11,15).

- Oddian hypertonia consists in the incapacity of the sphincter of Oddi to produce the intra-digestive release of the bile in the duodenum, with consequences on the intestine and stomach (previously mentioned). The incapacity of the sphincter favours the occurrence of the duodeno-billiary reflux that, through the increase of the intraductal pressure, may lead to the dilatation of the main biliary duct and may generate episodes of angiocholitis (4). Oddian hypertonia occurs after cholecystectomy or endoscopic sphincterotomy, at the same time with choledocian calculi extraction.

Clinically, the manifestations consist in food-unconditioned diarrhoea or angiocholitis, favoured by the duodeno-biliary reflux (1).

The positive diagnosis is supported by the lack of retention in the common bile duct, of the contrast dye upon cholangiography, by the presence of pneumobilia and the entry of barium in the common bile duct (Trendelenburg position) upon the barium examination, by the rapid passage of the radiotracer in the intestine, on the occasion of the hepatobiliary scintigraphy, by the retention of the contrast dye or of the tracer after the morphine-prostigmin administration (1).

The treatment consists in hygienic and dietetic diet with the consumption of cholecistokinetic food, adapted accordingly in case of diarrhoea. Cholagogic and choleretic drugs are also recommended.

Briefly, dyskinesia of the sphincter of Oddi is most frequently the consequence of cholecystectomy and in a percentage of 3- 8%, the consequence of sphincteroplasty or sphincterotomy in the biliary patients (14).

2.6 Unfunctional biliodigestive anastomoses

Biliodigestive anastomosis represents a procedure that has a variable impact on the normal physiology of the biliodigestive transit. Although, at the level of the biliodigestive derivations, the sphincter of Oddi is short circuited, the human organism adapts itself to the new anatomic and functional conditions.

Three important functional alterations are presented:

a) The continuous release of the bile in the digestive tube is common after cholecystectomy. In the case of the biliojejunal anastomoses, the bile reaches directly the intestine, avoiding the duodenum, what will bring to the decrease of the buffer capacity of the duodenal juice, regarding the gastric secretion (4).

b) The biliodigestive reflux is present at the level of the anastomoses made between the main biliary duct and the duodenum. In case of a jejunal anastomosis, the reflux is no longer produced (correct surgical technique). In the conditions in which the biliary deflux equals the biliodigestive reflux, an equilibrium is maintained. In order that the two mechanisms equalise, the anastomosis stoma should be large, permanently open and should have a diameter of at least 15 mm. Some authors consider that the repeated biliodigestive reflux brings about inflammatory lesions that lead to the degradation of anastomosis. The entry of the duodenal contents in the intra-hepatic biliary ducts produces angiocholitis, resulting in secondary biliary cirrhosis. These are the reasons for which physicians prefer the Roux-en-Y hepaticojejunostomy instead of choledocoduodenal anastomosis. The diagnosis of the biliodigestive reflux is made through the barium examination; the substance enters the biliary ducts spontaneously or after approaching the Trendelenburg position.

c) Stasis and angiocholitis

As long as the biliodigestive reflux is not accompanied by stasis, the clinical expression is missing. The factors that prevent the deflux are responsible for stasis occurrence (5,8), the infection being descendent and not ascendant. The most frequent cause of stasis is represented by the narrow or damaged anastomosis mouth.

The factors that make the biliodigestive anastomosis unfunctional are numerous and according to the moment in which they occur, they may produce:

- The immediate dysfunction occurring in the first days after the surgery; frequently, the obstruction is given by the remaining calculi (6).
- Early dysfunction occurs in the first months after the surgery. It is due to the remaining calculi and to those which migrate in the intra-hepatic biliary ducts.
- The late dysfunction is produced by a residual obstacle, by errors of surgery technique and by the degradation of the anastomoses, as a result of the irritation produced by the prolonged reflux, which lead to the stenosation of the mouth.

2.6.1. Dysfunction of the choledocoduodenal anastomose

In a percentage of 16%, choledocoduodenal anastomoses become unfunctional. The most frequent causes are:

- Obturation of the anastomosis mouth by the residual calculi from the main biliary ducts or by those which migrated from the intra-hepatic biliary ducts. The obturation of the anastomosis mouth is clinically manifested by pains, icterus and angiocholitis. Rarely, the obstruction is made by food rests (4).
- Subanastomotic pouch syndrome is produced by the lower common bile duct in the case of lateral anastomoses. Due to the siphonage phenomenon, the subanastomotic common bile duct aspirates the contents of the supraanastomotic common bile duct. In case the release is hindered (oddian stenosis, remaining calculi), biliary mud and food rests are gathered up to the level of the anastomosis mouth, leading to its obstruction. In stasis conditions, angiocholitis and recurrent calculi will occur. There is another phenomenon that occurs, that of the blind loop with the development of the microbial flora, having the same consequences. Angiocholitis also occurs in the case of a dyskinetic duodenum that does not favour the drainage of the lower common bile duct.
- The chronic inflammatory process at the level of the subanastomotic pouch brings about chronic pancreatitis, recurrent lithiasis, stenosation of the anastomosis mouth, of the papilla, repetitive angiocholitis.

  Clinically, stenosation of the anastomosis mouth is translated into repeated angiocholitis episodes.

  The diagnosis of stenosis is supported by the suggestive aspect upon the retrograded cholangiography, while the biliary scintigraphy of release indicate an extended hepatic time, the dilatation of the biliary ducts and a late passage of the tracer in the duodenum.

  In the case of the choledocoduodenal anastomoses, the reflux angiocholitis is favoured by: a narrow anastomosis mouth, the presence of the mobile calculi larger than the anastomosis stoma, by hypotonia of the main biliary duct. In all these cases, stasis brings about frequent angiocholitis episodes. The dilatation of the common bile duct more than 25 mm benefits from Roux-en-Y hepaticojejunostomy**.** Reflux angiocholitis may be the result of the subanastomotic pouch syndrome or of the duodenal dyskinesia.

  The most serious complication of angiocholitis is represented by the hepatic abscess that occurs in 6-43% of the cases.

- Gastritis and esophagitis through biliary reflux are late complications, the consequence of the continuous release of the bile in the duodenum, as a result of cholecystectomy and short-circuiting the sphincter of Oddi.

  From the endoscopic point of view, biliary gastritis may have the aspect of chronic gastritis or erosive acute gastritis and from the histologic point of view, it is characterized though foveolar hyperplasia, interstitial edema, congestion of the lamina propia, reduced number of inflammatory cells, cystic dilatation of the gastric glands.

  Clinically, dyspeptic manifestations may occur; nausea, biliary vomiting, epigastric pains after eating and refractory to the antiulcerous medication.

  The diagnosis is established by the presence of gastritis (endoscopically), by the analysis of the gastric juice (presence of the biliary acids, of lysolecithin, bilirubin), alkaline pH, gastric juice and through isotopic methods (2). The lesions dominate the antrum, but they may extend in ascendant line, producing alkaline esophagitis (non-peptic).
- Secondary biliary cirrhosis occurs in the patients with large anastomosis (over 25 mm in diameter) and who survive long after the surgery.

The treatment of the unfunctional choledocoduodenal anastomoses is the surgery with the accomplishment of another Roux-en-Y anastomosis, when the supraanastomotic segment of the main biliary duct is long, choledocolitotomy with Kehr drainage, in the case of calculi of the main biliary duct, choledocoduodenal anastomosis in the case of stenosis of the anastomosis mouth, transduodenal sphincterotomy/sphincteroplasty or endoscopic sphincterotomy in the case of the subanastomotic pouch syndrome or residual / recurrent lithiasis of the main biliary duct (4).

2.6.2. Dysfunction of the biliojejunal anastomoses

After the biliojejunal anastomoses, the complications also occur.

Anastomotic fistulae, post-surgery acute pancreatitis are rare complications and occur early after the surgery, due to technique deficiencies (anastomosis with small calibre, short jejunal loop) (6).

Late complications of the biliojejunal anastomoses:

- Painful syndrome of the right hypocondrium, unaccompanied by icterus may frequently occur, without knowing the physiopathologic sublayer (4).
- Angiocholitis occurs when the anastomotic loop is smaller than 50 cm, when the residual hepatic lithiasis is present and the migrating calculi obdurate anastomosis, when anastomosis is damaged or when the excluded loop is torsioned or compressed.

  The obstacle is emphasized upon the hepatic-transparietal cholangiography and the biliary scintigraphy of release.
- Gastroduodenal ulcers occurs as a result of the peptic aggression of the gastric juice through:
  - Reduction of the buffer capacity of the duodenal juice through the short-circuiting of the duodenum, the bile passing directly in jejunum.
  - Significant increase if the gastric acidity through the exclusion of duodenum, what brings about the decrease of the enterogastron secretion, the increase of the titre, serous gastrin or through hypoglycaemia, (due to hyperplasia of beta cells), which maintain the gastric hypersecretion on the vagal way.

- Stenosation of the anastomosis mouth occurs in percentages of 15- 23% during an average interval of 11 months. This complication occurs after the terminolateral anastomoses, when the hepatic channel is not sufficiently dilated, or when the diameter of the main biliary duct is less than 15 mm. Secondarily to stenosis, stasis takes place in the supraiacent segment, favouring the infection and the secondary lithiasis.

The diagnosis may be confirmed through scintigraphy, showing the delay of the radiotracer at the level of the liver (6).

The treatment of the unfunctional biliojejunal anastomoses is the surgical one and consists in the recovery, widening or plastic operation of anastomosis. (4).

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