Topical aspects of the development of chronic biliary pancreatitis

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Diseases of the digestive steadily increasing around the world, which is associated with increased alcohol consumption, excessive consumption of spicy and fatty foods, smoking, psycho emotional factors, lower social standards, co-morbidities. In recent years, the prevalence of digestive diseases among the population of Ukraine increased at 24.7% morbidity — by 8.7%, mortality — in 14.0% [22].

Chronic pancreatitis (CP) is one of the most common, the most pressing problems in modern gastroenterology due to difficulties in early diagnosis and treatment of low efficiency. CP is a chronic, lasting more than 6 months, progressive disease characterized by inflammatory and degenerative and to a lesser extent, necrotic changes in the pancreas, a violation of its terrain talk, repeated exacerbations and gradual replacement of parenchymal organ connective tissue with the development of its exo- and endocrine insufficiency.

CP is a polyetiological disease. The main etiological factors: alcoholism, liver disease and bile of the excretory tract, hyperlipidemia, performance chemicals (including drugs), duodenal ulcers (DU), lack of protein in the diet, hereditary factors hyperparathyroidism, cystic fibrosis, pancreatic trauma, allergy.

Morbidity of diseases and CP across Europe is 4 to 8 cases per 100 thousand. People a year, and incidence of — 200 — 500 100 thousand patients. Noticeable certain frequency dependence of CP on the socio-economic level of the country. The highest rate for the disease is observed both in poor countries, due to malnutrition, and in developed — through increased use of alcohol and animal fats. The disease tends to increase. In developed European countries is much CP younger — the average age of patients with this diagnosis decreased significantly from 50 to 39 years and a 30%

increase in the number of women. The incidence of pathology is growing, which is associated with increased alcohol consumption and improved methods of diagnosis, including diseases of the hepatobiliary system.

The most common causes of CP related liver disease and biliary ducts. According to various sources on the global scholarly literature in 35 — 60% of biliary ducts pathology recognized factor which leads to CP [14]. About a third of the population of the planet suffers from biliary pathology. Pathology of the biliary system is characterized by wide prevalence, diversity of clinical manifestations of complications. Therefore, timely qualified diagnosis, rational treatment and prevention are important and can prevent a number of complications. In recent decades, there is steady growth biliary diseases — pancreatoduodenal zone.

Gall stone disease (GSD) — diseases of the hepatobiliary system, metabolic disorders caused by cholesterol and/or bilirubin in combination with other factors that lead to the formation of gallstones and is one of the most frequent causes of chronic biliary pancreatitis (CBP) which in recent years has tended to increase. GSD incidence increases with each decade twice. Also, statistics show the rejuvenation of this disease. In Ukraine, the prevalence GSD is 5-12%, whereas in North America and Europe, where the population is predominantly take foods rich in cholesterol, is 30%.

According to V. H. Vasilenko, "GSD is a charge for long and satisfying life" [3]. Risk Factors combine the concept of “four f”: 1) female over forty (women over 40), 2) fat (predisposition to obesity), 3) flatulent (flatulence), 4) fertile (able to birth). Women suffer from GSD to 2 times more than men. This is due to hormonal effects on cholesterol metabolism. Estrogens inhibit the synthesis of bile acids, increasing the concentration of cholesterol in bile. Also common in women during pregnancy have elevated cholesterol in the bile, which is a result of hormonal changes [19].

For the first time human gallstones have been described in the XIV century. The following types of stones: cholesterol (containing cholesterol), pigment (bilirubin and its containing polymers), lime (composed of carbonate of lime) and mixed.

Cholesterol stones are round or oval, white or yellow, smooth, radiant with the cut structure. Pigment stones containing bilirubin and clay, they are always multiple, small, black. Mixed stones vary in color, shape and size. In 80 — 90% of patients with GSD residents of Europe and North America cholesterol stones formed, and the inhabitants of Asia and Africa dominated pigment stones.

In 1901 the famous scientist E. Opie is explained the mechanism of this disease — the appearance of biliary-pancreatic reflux during obturation sphincter of Oddi (OS) by concrement (theory of common duct) [16]. This theory explains with anatomical features include common bile in the duodenum and pancreatic ducts.

As is known, the pressure in the common bile duct is 250 mm. Lower than Wirsung's (300–500 mm), which prevents maligns bile duct in pancreas. The mucous membrane ducts resistant to a mixture of bile and pancreatic enzymes at normal pressure in the ductal system. Its resistance is broken hypertension. During prolonged interaction of bile and pancreatic secretion is associated release of bile acids that damage the protective barrier straits pancreas.

During stimulation of small concretions big duodenal papilla (BDP) develops an inflammatory process in it (papillitis), and subsequently formed stenosis.

With the passage of concretions in the duodenum inflammation subsides in pancreas, and when it increases retention hypertension, difficult outflow of pancreatic secretion and therefore appeared choledoho- and duodenopancreatic reflux. Clinically it is manifested arose stenosis and increase pain [25].

In 1884 there was an operation to remove concretions, which were in the doorway BDP. This phenomenon is described by two scientists, whose names it is called s primary city of constrictive papillitis or disease of Del-Vale-Donovan, and recognized as causes of obstructive jaundice screening for stones in gall bladder.

Secondary stenosis of BDP occurs after a trauma during the passage of small concretions or due to pathological changes in the duodenal mucosa or choledochitis.

Not always choledocholithiasis leads to CBP. Often this is an ampullar lithiasis and BDP concrements. Most of the stones in the gall ampoule with BDP origin, but there is evidence pancreatogenic lithiasis with the occurrence of pancreatitis and

jaundice. The presence of calculus gall bladder up to 5 mm increases the risk of CP 4 times [1].

GSD is often associated with other diseases. Many researchers and scientists argue that the GSD is not an independent entity as always and combined with chronic cholecystitis, considered as second primary (physical and chemical) stage [13]. Chronic cholecystitis — chronic inflammatory disease gall bladder wall, accompanied by a violation of its motor function and suction capacity, changes in the structure and properties of bile (dysholiya) with frequent involvement biliary vessels (angiocholitis) and ducts (cholangitis).

An important feature of municipal economy in the early stages you are appearing biliary sludge (BS) in gall bladder. BS is the initial stage of the formation of concretions, arising from breach of biochemical sculptor down bile, leading to precipitation its main components. [9] The term «biliaris» of Latin means «yellow», «sladge» — sediment deposition on the bottom gall bladder calcium and sodium salts, bile acids, pigments, cholesterol, bilirubin, mucus.

Normally bile — a yellow or brown viscous liquid, the main components of which are bile acids, bile pigments, cholesterol, phospholipids and water. Cholrsterol is translated from the Greek language — "hard of bile". Bile acids and lecithin cholesterol support in the liquid state, that is, prevent its crystallization [11].

Occurrence of BS is contributed by: bile cholesterol saturation, value abuse in the family component availability pronuclears (mucus glycoproteins, immunoglobulins, ionized calcium, bilirubin, phospholipids) and antinuclears (apolipoprotein A1 and A2, bile acids, lecithin, acetylsalicylic acid).

In ultrasound has the form of a cloud of multiple slices echo-positive without acoustic shadows in the lumen gall bladder. However, ultrasound is not always informative, namely when large size and inclusion do not give an acoustic shadow. More reliable detection method is BS microscopy bile collected in gastroduodenal sounding.

There are 3 types of base stations: microlithiasis (small hyperechoic including without acoustic shadow), bile clots ointment and a combination of both types

[12]. BS may disappear on its own, but can be a substrate for subsequent calculus formation. Sludge containing microliths may pass freely across the ductal system, irritating the mucous membrane of the biliary tract, thus causing pain. According to new data, the cause of non-defined pain in the right upper quadrant in 83% of patients can be BS.

One of the most frequent complications is the emergence of CBP. BS found in 50% of patients with CP. In 90% of cases with sludge observed OS hypotonus, and there is reflux of bile to Wirsung's duct. According to a Japanese researchers, BS is a marker of early cancer gall bladder. Therefore, they recommend BS endoscopic catheterization with cytology for the atypical cells.

Early diagnosis and appropriate management BS treatment has important clinical implications because of the large probability of further development of various complications of biliary-pancreatic zone.

Gastroduodenal diseases of system often accompany GBD and contribute to the recurrence of CBP. Frequency of housing and communal services and related erosive — ulcerative lesions of gastroduodenal area is around 25-27% [21]. This increases the acidity of gastric juices caused hypergastrinemia that excessive stimulation for pancreas and with the additional difficulty of outflow of secretion leads to intraductal hypertension and exacerbation of pathological process.

Among biliary pathology that leads to the CP, as dyskinesia, manifested motoric-evacuation function of gall bladder and biliary tract. Dyskinesia can be as independent disease, and accompany GSD, cholecystitis. Share dyskinesias biliary system diseases system is 12–25%.

Among the causes of isolated, nutritional causes (food allergy, irregular meals, eating low-calorie, fried, fatty foods, coupled with sedentary lifestyle), disorders of the nervous regulation of gall bladder changes in hormone levels gastrointestinal and endocrine glands (menopause, lack of adrenal glands, single cyst and polycystic ovaries, hypothyroidism, hyperthyroidism, obesity, diabetes), ulcers, gastritis, duodenitis, diseases of the spine. The inflammatory process in the mucosa of the

duodenum leads to disruption of the hormone cholecystokinin-pancreozymin, which plays a central role in the regulation of motility.

Dyskinesia divided into hypokinetic (atonic) — couple with the predominance of tone of the sympathetic nervous system and hyperkinetic (spastic) — with predominance of tone sympathetic nervous system. The first type gall bladder showed an increase, slowing down its contractile function, bile stasis. In the second type of change observed opposing express gall bladder reduction, reduction of bile in it, the secretion of bile in small portions.

In both types of dyskinesia as a result of uncoordinated gall bladder bile duct and sphincter disrupted flow of bile into the lumen of the duodenum that causes CBP and other digestive disorders.

According to statistics, mainly women suffer 10 times more often than men. Often ill pregnant decreases as the tone of the uterus, intestines, biliary tract.

In the case of a long course of gradually developing receptor system damage nerve cells occurring morphological changes mucosa gall bladder the development of its inflammation. Therefore dyskinesia is seen as a preliminary step to cholecystitis.

Long hypomotoric dyskinesia of gall bladder and OS spasm leading to stagnation of bile, a violation of its colloidal stability, the development of inflammation with the formation of stones.

Also cause of CBP may be the anatomical features of bile and pancreatic ducts, place their compounds, their lengths and diameters. Statistically, long and wide choledoch several times increases the occurrence of CP than the average patient in the presence of common bile duct.

Morphologically CBP is characterized by degenerative lesions in parenchyma cancer, uniform expansion or stenosis of the main pancreatic duct in a particular area.

GSD leakage in many cases is accompanied by concomitant diseases of the digestive system, namely hepatopancreatobiliary zone. In recent years, municipal economy is growing both in Europe and in Ukraine. Correspondingly, increasing number of cholecystectomy (CE). Although CE is the main etiopathogenetic treatment,

but not always solve the problem and may even trigger exacerbation or progression of comorbidity. Among the areas of hepatopancreatoduodenal remove gall bladder often displayed on the operation of pancreas. V. A. Zorina et al. in the study patients after 85% CE marked increased blood levels α 1-antitrypsin, with a 34.7% of the rates prevailing rate of more than 2 times. Timely and technically well-executed operation, especially in the early stages GSD does not affect the functional state of the second pancreas. At full restoration of patency of the bile and pancreatic w Poles reduced the severity of pathological processes in pancreas comes pancreatocytes regeneration and increases their activity. Reparative processes begin with and are characterized by stromal and reverse the development of connective tissue in the parenchyma move that helps restore the functional activity of the gland. But long process full recovery does not occur.

Almost a third of patients have had a history of CE, continue to bother abdominal pain, and dyspepsia. Their presence is related to two main factors: how metabolic cholesterol (GSD etiological factor), and that the secretion of bile is the new anatomical and physiological conditions (in the absence gall bladder) [10].

Loss of physiological role gall bladder — namely the concentration of bile in inter-digestive period and release it into the duodenum during digestion is accompanied by the passage of bile into the intestine and digestive disorders [8]. When changing the chemical composition of bile and its chaotic flow of disturbed digestion and absorption of fat and other substances of lipid nature, leading to the reduction of bacteria and duodenal content, microbial contamination in the duodenum, weakening growth and operation of the normal intestinal flora, intestinal disorders hepatic circulation and reducing the total pool of bile acids. Under the influence of microflora bile acids to be premature deconjugation that causes duodenal mucosal lesions, the appearance of duodenitis, enteritis, colitis, cholangiogenic diarrhea [4].

Often after CE there is a violation enzymatic-forming function of the pancreas, which leads to heightened pancreatitis. The frequency of CP after CE is 15-90% [6].

All pathological manifestations that occur after the CE united under one name — postcholecystectomical syndrome (PCES). By the term "PCES" refers compensatory

dilatation of the common bile duct with reduced contractile function, dysfunction and OS spasm, chronic duodenal obstruction, secondary pancreatic insufficiency, cholangiogenic diarrhea [7]. OS is a fibro-the muscle pouch containing the end of the common bile duct and pancreatic and common channel that passes through the wall of the duodenum.

SB performs important functions — namely, the secretion of bile and pancreatic secretions into the duodenum during digestion and prevents back flow of duodenal contents into the common bile and pancreatic ducts.

Cholecystokinin-pancreozymin is a hormone produced by the duodenal mucosa and helps reduce OS gall bladder and relaxation when you receive food in the gut. Not digestion opposite changes occur — gall bladder relaxation and toning OS.

Mucosa gall bladder produced antagonist cholecystokinin-pancreozymin-anticholecystokinin that gall bladder relaxes and tones the OS in the rest period. When you remove gall bladder anticholecystokinin action ceases, and consequently formed dysfunction OS, in most cases, with a predominance of his spasm [17]. Dysfunction OS is found in 30% of patients with CP [15]. It can be primary and secondary. Primary and linked to a decrease in muscle mass and decreased sensitivity gall bladder receptor system to neurohumoral stimulation. Secondary observed structural changes (ductal stenosis and sphincter), chronic inflammation of the extrahepatic biliary system, can also occur with hormonal disorders, pregnancy, hepatitis, cirrhosis, treatment somatostatin, diabetes.

There are 2 types of violations n s tone OS hypo-and hypertonicity. The first type is reflux duodenal content and in the common bile duct pancreatic district in the development of inflammation in the pancreas and biliary tract. When another complicated type exit of bile and pancreatic secretion in the duodenum, increased pressure in the common bile and pancreatic ducts, and therefore increases the pain.

SO dysfunction is also divided into biliary (choledochal sphincter dysfunction) and pancreatic (dysfunction of the sphincter of pancreatic duct), which is important for the differential diagnosis of pathological process [5].

Duodenostasis and duodenal hypertension have considerable importance in the development of OS dysfunction, manifested duodeno-biliary-pancreatic reflux. Duodenal mucosa produces enterokinase which activates enzymes pancreas. Activated trypsin is necrosis of the parenchyma of leukocyte infiltration, phospholipase A and B damage phospholipase A layer membranes pancreatocytes, elastase affects elastic membrane of blood vessels, causing hemorrhage, kallikrein increases vascular permeability, which led forth one's to leakage of fluid, ie swelling cancer [7].

CBP also contribute to the development of chronic liver disease (hepatitis, cirrhosis, steatosis). This is because when the lesions formed abnormal liver bile, which contains large amounts of free radicals when hit in pancreatic duct causing precipitation of proteins, formation of stones, the occurrence of inflammation [24].

Nonalcoholic fatty liver disease (steatosis) and CP is interconnected, that each of these diseases may precede the other. The spread of the disease is about 30% in Europe and increases to 90% in the presence of obesity. NAFLD seen as hepatic manifestation of metabolic syndrome. There are three mechanisms of lipid accumulation in the liver: excess revenues, excessive synthesis by the body and lack of output [23].

Prolonged progressive course incretory CP develops pancreas failure (diabetes), insulin resistance. One of the mechanisms that explains the development NAFLD in CP is that when insulin resistance increases the activity of the enzyme fatty acid synthase that produces an excess of triglycerides and cholesterol. Regular consumption of fatty foods leads to increased production of HC-pancreas and lipolytic enzymes at a normal volume and bicarbonate secretion. The result is the formation of Precipitation proteins "protein plugs" disturbed flow secretion, inflammation develops pancreas. Free fatty acids produced by the hydrolysis of triglyceride lipase influenced lipocyte-toxical exert influence on pancreas that shows the influence of the oppressed increased concentration of lipids on the function of β-cells, causing endocrine insufficiency. Internal secretion failure occurs much later than the external and because insulocytes better preserved than the acinar cells due to the presence in them locking mechanism of apoptosis.

Also has proven that with the defeat of the liver virus hepatitis B and C the possibility of replication in the parenchyma Pancreas (acinar, ductal, endocrine cells) and initiating autoimmune processes, directed on β-cell pancreas that is accompanied by a decrease in insulin secretion in patients with diabetes Type 2 [20].

Often diseases such as worm infestation, cysts pancreas polyps gall bladder violate the flow of bile, contributing to stagnant processes, leading to disruption of the colloidal structure of bile. The literature describes different types of helminths (ascariasis, opisthorchiasis, ankylostomiasis, echinococcosis, strongyloidosis) with mechanical, toxic, reflex, allergic effects, both pancreatic-biliary system and the body as a whole. Parasitic pancreatitis some scholars have identified as a distinct form of the disease.

One of the most common helminths, which leads to destruction pancreas is ascariasis. Ascarids crawl into the head duct and additional pancreas that is a violation of outflow of pancreatic secretion, hypertension in the ductal system and the development of pancreatitis. Prolonged stay in the Pancreas and roundworm may develop an abscess, blockage of ducts cluster roundworm eggs.

Defeat pancreas opisthorchiasis observed in one third of patients with disturbed incretory and excretory functions. In these patients the disease is long because these parasitic worms in the body for about 20 years and implement CP.

Echinococcal cyst, located in the head of pancreas compresses the common bile duct, gives the outflow of pancreatic juice, and toxic effects of helminth lead to inflammation of the pancreas.

Another cause of CBP is the transition from gall bladder inflammation, bile duct of pancreas for venous and lymphatic vessels in the absence of biliary-pancreatic reflux. When lymphogenous lesions usually damaged pancreatic head, due to its anatomical location and proximity gall bladder. The source of destruction is a chain of enlarged lymph nodes that are lit from head to gall bladder pancreas [2].

CBP peculiarity that distinguishes it from other forms is the presence of exocrine pancreas and chronic biliary disease, as shown on the digestive process.

The syndrome of exocrine insufficiency in CBP due to a decrease in mass parenchyma due to its atrophy, fibrosis, violation of the outflow of pancreatic secretion due to blockage of ducts.

Secondary pancreatic insufficiency occurs when pancreatic enzymes are not up- or in the intestines. It is this mechanism are leading with biliary pancreatitis, since the GSD, PCES observed asynchronism entering the duodenum bile and pancreatic juice, which is a manifestation of the three pathogenic mechanisms: insufficient activation of pancreatic lipase, acidification duodenum (inactivation of lipase), violation emulsification fat and the formation of micelles [13].

Thus, the mechanism of formation of CBP is often complex and multifactorial. Keeping these patients requires consideration of these mechanisms, existing comorbid conditions and their rehabilitation is often lengthy and not always effective. Therefore, further scientific development of this problem is urgent and very modern problem.

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Topical aspects of the development of chronic biliary pancreatitis

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This paper presents an analysis of the literature on diseases of the hepatic biliary system leading to the development of chronic pancreatitis. Biliary factors provoking the development of chronic pancreatitis include the following: functional disorders of the biliary tract, chronic cholecystitis, cholelithiasis and cholecystectomy in a medical history, hepatitis, cirrhosis, steatosis, transition of inflammation from the gall bladder and bile ducts into the pancreas, anatomical features of the biliary and pancreatic ducts. Peculiarity of chronic biliary pancreatitis, which distinguishes it from other forms, is the presence of exocrine pancreatic insufficiency and chronic biliary insufficiency, which affects the quality of the digestive process. Upon biliary pancreatitis secondary pancreatic insufficiency occurs, in which the pancreatic enzymes are not activated or inactivated in the intestine.