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Periampullary duodenal diverticulum and some rarely complications: review of literature

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Abstract:

Duodenal diverticulum was first reported by Chomell in 1710. With modern radiological techniques and widespread use of endoscope it has been found that these diverticula occur more frequently than was formely supposed. Most of these are asymptomatic, situated in second part of duodenum and are rarely associated with complication which are usually cause of presentation. Diverticula located at a distance 20 mm from the ampulla of Vater are known as periampullary duodenal diverticula (PDD). It is suspected to be a cause for biliopancreatic diseases on patients with malfunction of the sphincter of Oddi and reflux of pancreatic juice and bowel contents. Its prevalence rises with increasing age

Keywords

Periampullary duodenal diverticulum, Pancreatitis, Choledocholithiasis, Jaundice

1. Introduction

Duodenal diverticula was first reported by Chomall in 1710 and first well documented report was made by Morgagni in 1762. With lengthening of life span, diverticulosis has come to occupy a more important position in the sphere of clinical gastroenterology.

Duodenum is second most common site of diverticula in alimentary tract after colon followed by jejunum, ileum and stomach.(2,3) Duodenal diverticula are found in up to 25% of patients, but rarely cause symptoms when complications occur, early diagnosis is essential if treatment is to be successful.(3) Diverticula of duodenum are classified as primary and secondary.

These occur mainly in later decades of life with peak incidence between 50 and 60 years of age and it increases with age.(1,2,4-6) Female preponderance has also been reported.(1-5) Incidences varies with diagnostic method used.(3) In UGI barium series it is 0.016 to 6%, (2,4-8) in autopsy series 22 to 23%, (2,4,5,7) where as on ERCP studies incidence is 9 to 23%.(6,7) Over 95% of duodenal diverticula project from inner or pancreatic border of duodenal curve in second, third and fourth parts.

Second part is most common site with 85 to 90% of total DD.(1,2,4,9) Third and fourth parts of duodenum have 20 and 10% of diverticula respectively,(2) but up to 30 to 40% of these may arise from third and fourth part of duodenum.(10) Diverticulum may be single or multiple and as many as 6 or more has been reported. Incidence of multiplicity at x-ray series is 1.4 to 23.5% and in autopsy series it is 3.5 to 30%. Diverticula located at a distance 20 mm from the ampoule of Fater are known as periampullary diverticula. The reason for the high incidence of diverticulosis in this anatomical region is the presence of congenital weakness in the wall of the duodenum in the area where holedohus ductus ductus pancreaticus and enter the lumen of the gut. Furthermore, the penetration of blood vessels in the mucous membrane, additionally leads to weakness of the wall to enhance the formation of diverticula (8, 9, 11). These include 75% of duodenal diverticula (11, 12). They are of particular interest for clinical practice.

2. Etiology

These are true pouches or sacs protruding from duodenal lumen not caused by any recognizable

intrinsic or extrinsic disease. PDD are acquired and consist of a sac of mucosal or sub mucosal layers herniated through a muscular defect in bowel wall but precise manner of the development is not known.(2,6,10) However, there exists a locus minoris resistentiae which may be created by passage of the biliary, pancreatic duct and blood vessels through the wall of duodenum.

Congenital absence of an adequate muscle coat heterotrophic pancreatic tissue. Once diverticulum is formed it may increase in size through the years.(2,6,10)

The sac lie along side, extend behind or even penetrate into the pancreas and occasionally common

bily duct (CBD) and duct of Wirsung may open into the diverticulum (3,4,6) PDD or windsock diverticula are not true diverticula and occur as single saccular structure which are connected to the entire circumference or only part of the wall of the duodenum. Only less than 100 cases have been reported in the literature. During early foetal life duodenal lumen is initially occluded by proliferating epithelial cells and recanalises later on. Abnormal recanalising may lead to a duodenal diaphragm or web which may not produce symptoms in child hood however over the period of time peristaltic stretching may transform it into an PDD. PDD may project as distally as fourth part of duodenum and often a second opening is located eccentrically in the sac and both sides of the diverticulum are lined by mucosa.(3,6,10)

3. Clinical features

There are no characteristic symptom complex from which one may make a positive diagnosis of PDD. Many believe that there are three main factors in production of symptoms.

I. Mechanical causes producing
   1. Delayed empting of diverticula
   2. Pressure on the common bile or pancreatic duct
   3. Obstruction of the duodenum

II Inflammatory causes producing
   1. Symptoms simulating peptic ulcer, gall bladder or pancreatic disease
   2. Pyloro-spasm
   3. Perforation

III Neoplasia

4. Complications

4.1.Periampullary duodenal diverticulum and pancreatitis

It is suspected to be a cause for biliopancreatic diseases on patients with malfunction of the sphincter of Oddi and reflux of pancreatic juice and bowel contents. Its prevalence rises with increasing age [5]. Some studies have found an increased prevalence of acute idiopathic pancreatitis [4, 7]. Inflation of diverticulum can cause compression of the pancreatic duct, which may result in development of pancreatitis [1,3] his is the standard font and layout for the individual paragraphs.

The presence of periampullary duodenal diverticulum (PDD) is often observed during upper digestive tract barium meal studies and endoscopic retrograde cholangiopancreatography (ERCP). A few papers reported that the diverticulum had something to do with the incidence of pancreatitis. Studies have shown that there is insufficiency of the sphincter of Oddi in patients with diverticulitis, as well as a higher percentage of bacterial contamination of the duodenum and the bile ducts in them.

The research of Zoeph et al. [31] is on two groups of 350 patients, differentiated on whether the patients have or do not have PDD. The researchers did not find significant difference in the frequency of acute pancreatitis in both groups. Uomo et al. [7] examined 433 patients, on which ERCP had been made during the period 1992 - 1994. They faund acute idiopathic pancreatitis in 13.7% of the group with periampullary duodenal diverticulum compared to 1.8% of the group without it. They demonstrated an association of PDD with acute idiopathic pancreatitis and postulated that the duodenal diverticulum is a risk factor for acute idiopathic pancreatitis.

Etiological causes in patients with acute pancreatitis who are often admitted to surgical clinics are generally focused on a few frequent factors. But less frequently observed etiological causes may constitute problems for patients and doctors. [9]. As a congenital malformation, the diverticulum can be enlarged with age and lead to an onset of clinical symptoms. Data published in literature is quite contradictory. It does not point unquestionable proof for the significance of the periampullary duodenal diverticula in etiology of the pancreas pathology, especially in the acute and chronic pancreatitis.

Several authors have shown that compression of CBD, dysfunctions of ampulla or a poorly emptying diverticulum with a narrow neck can lead to pancreatico-biliary disease and acute pancreatitis. Some even suggested that PDD be included in the aetiology of acute pancreatitis especially in the elderly.

4.2.Periampullary duodenal diverticulum and choledocholithiasis

Manometric data shows that a PDD is associated with reduced pressure in the sphincter of Oddi. This atonic could allow reflux of microorganisms of the diverticulum with stasis. The bacterial overgrowth in diverticular pocket and around it is a cause of colonization of the ductus holedohus in inadequate, sphincter of Oddi by bacteria which produce B-glucuronidase. This enzyme leads to the transformation of the conjugation in unconjugated bilirubin, which in turn binds to the calcium ions from the bile and form insoluble nuclei, leading to the development of concrements. This may be a cause of primary and recurrent choledocholithiasis, which are more common in patients with PDD than in patients without PDD. The presence of bile in combination with bacteria in the lumen of the diverticulum leads to inflammation of the papilla and

affects the motility of the sphincter. Contractile malfunction of the latter leads to reflux of gastrointestinal contents in the ductus holedohus, bacterial infection and the formation of pigment gallstones. Observations of a number of authors indicate a secure connection between the PDD and the availability of primary ones in extrahepatic bile ducts.

The inflation of a diverticulum can cause compression of the ductus holedochus, which may result in an development of choledocholithiasis [2,3]. A few papers reported that the diverticulum had something to do with the incidence of choledocholithiasis. The impact of periampullary duodenal diverticulum (PDD) for the formation of gallstones in the bile duct is widely accepted [3,4,6,7]. Scientific publications on these issues began to appear in the late 70s with the introduction of endoscopic methods, in particular endoscopic retrograde cholangiopancreatography (ERCP).

The review of the literature reveals data on the incidence of choledocholithiasis in patients with PDD ranging from 12% to 68% [4,7]. These theories put PDD in the role of an independent factor in the pathogenesis of choledocholithiasis.

PDD are important causative factors in the bile duct stone formation.(14,15) Since choledocholithasis leading to biliary obstruction is a well known etiology of acute pancreatitis there is debate whether the increased incidence of pancreatitis with PDD is due primarily to the mechanical complications or associated biliary stones.

4.3.Periampullary duodenal diverticulum and jaundice

The reason for the occurrence of obstructive in patients with PDD states compression that it exerts on the biliary tract. This is confirmed by the successful relief of jaundice after surgical removal of diverticula. Obstructive jaundice in the absence of gallstones and caused by PDD is known as "Lemmel's syndrome" (4).

Hall et al. make an analysis of 641 patients undergoing endoscopic retrograde cholangiopancreatography (ERCP). Divide them into two groups - with and without PDD. They found that the first incidence of jaundice is 48.4% (47 patients out of 95) and the second -43% (41patsienti 95), p <0.01. For similar results reported other authors (1,2,5,6,10,13).

Shaymardanov in 2003. Published an analysis of 148 patients in whom the incidence of jaundice is 20.9%. Anastasios J. Karayiannakis et al in 2012 publish material painless patient with obstructive jaundice. They point out that Periampullary duodenal diverticula are not uncommon, but are usually asymptomatic. In a study Solhang observed 755 patients who have received ERCP. In the groups with and without PDD establish frequency of jaundice 27% vs. 8%, p <0.001.

The symptoms simulating primary biliary tract disease may occur in DD located near ampulla of Vater and some time CBD may terminate in the diverticulum. The “perivaterian diverticulitis” may give rise to obstructive jaundice. The presence of such diverticulum should be considered in elderly patients who present with obstructive jaundice in the absence of gallstones or a tumor mass in the pancreatic head.

5. Conclusion

The etiology of the complications of the billiopancreatic system is complex. PDD is only part of this etiology. The survey of dependence on him would serve as a possible assessment of risk of specific complications.

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