THE MINOR PAPILLA IN THE DUODENAL DIVERTICULUM: ITS POSSIBLE ETIOLOGICAL ROLE IN THE FORMATION OF COMMON BILE DUCT DILATATION: A REVIEW

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Key Words: Choledochal cyst, common bile duct, duodenal diverticulum duodenal papilla.
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Abbreviation: ERCP = Endoscopic retrograde cholangiopancreatography, HD = hypotonic duodenography, DIC = drip infusion cholangiography, MRCP = magnetic resonance cholangiopancreatography.

Abstract
This is a review of common bile duct dilatation and duodenal diverticulosis. Special emphasis is given to the possible role of the minor papilla in the diverticulum in the formation of the dilatation.

Introduction
Since its first report by Babbit (1969), anomalous pancreaticobiliary ductal union has been discussed as playing a possible etiological role in common bile duct dilatation (Morishita, Fujii et al., 1978; Morishita, Asakura et al., 1980). Bile duct dilatation is of particular importance in that bile duct carcinoma has been reported to develop from the dilated segment-particularly in young patients (Komi, Kunitomo et al., 1984). However, it has begun to seem unlikely that an anomalous union is a cause of the dilatation. We have previously reported two cases of common bile duct dilatation in which ERCP (endoscopic retrograde cholangiopancreatography) disclosed the minor papilla in the

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duodenal diverticulum without an anomalous pancreaticobiliary ductal union (Morishita, Asakura et al., 1980). This paper discusses the significance of minor papilla in the duodenal diverticulum in terms of the formation of common bile duct dilatation, as well as the efficacy of ERCP (Morishita, Yokoyama et al., 1997) for detecting such papilla.

Clinical Profiles of the Patients

Case 1: Three weeks prior to presentation at our hospital, a 65-year-old woman began to experience episodes of vomiting (1-6 times at 2-3 hours after supper) in association with fever (38.0° C-39.5° C), abdominal pain, and lumbago. Her body weight decreased 6 kg over this period. In addition to the above symptoms, she began to experience jaundice and itching, she visited our hospital and was admitted. Laboratory examination revealed the following: ESR 96 mm/ h, WBC 6600/mm³ (N60%, L35%, M5%), prothrombin T 13.7'' (95%), Fbg 733 mg/dl, T. Bil 7.8 mg/dl, D. Bil 4.8 mg/dl, I. Bil 3.0 mg/dl, GOT 197 K.U., GPT 131 K.U., Al-P 66.3 K.A.U., γ-GTP 789 IU/l, LDH 360W.U. (V 22.4%), TC 215 mg/dl, ZTT 17.8 Kunkel U., TTT 6.7 Mac. U., TP 8.0 g/dl, γ-glob 25.0%, AFP (-), Hbs-Ag (-), Hbs-Ab (-), S-amylase 79 S. U./dl, and U-amylase 414 S.U./dl. The results of a Meltzer-Lyon test were as follows: maximal icterus index 110; sand in the gallbladder (+); enterobacter (+); morganella (+); cytodiagnosis class I. Radiographic findings of the stomach and duodenum indicated a parapapillary diverticulum with a 21 x 22 mm diameter in the duodenum. A large diverticulum was also endoscopically observed in the orifice side of the papilla of Vater, and a minor papilla was noted in the diverticulum. ERCP showed cystic dilatation of the common bile duct, which had a diameter of 25 mm at the maximum point of dilatation. A large stone was noted in the lower part of the common bile duct. Several small to midsize stones were noted in the gallbladder. No anomalous pancreaticobiliary ductal union was observed. The diameter of the main pancreatic duct was 6.0 mm at the neck, 3.0 mm at the body, and 2.5 mm at the tail. The accessory pancreatic duct was 20 mm in length, and 2.0 mm at its largest diameter. The accessory pancreatic duct entered into the minor papilla in the duodenal diverticulum. The patient underwent cholecystectomy, choledochotomy, and choledocholithotomy at the Department of Surgery. Bile duct
Case 2: Ten years prior to presentation at our hospital, a 55-year-old woman visited her doctor due to pain in the right hypochondrium and epigastrium. She was diagnosed with cholecystitis. For the following eight years she continued to have annual episodes of the same abdominal pain, particularly after eating fatty foods, followed by two years of attacks of increasing frequency, approximately one every two months. She then visited our hospital for examination and was admitted. Laboratory examination revealed the following: ESR 16 mm/1h, WBC 3800/mm³, Prothrombin T 14.1" (64%), icterus index 5, GOT 16 K.U., GPT 8 K.U., Al-P 64 K.A.U., γ-GTP 13 IU/l, LDH 291 W.U. (V 4.1%), TC 235 mg/dl, ZTT 11.5 Kunkel U., TTT 0.4 Mac.U., TP 7.1 g/dl, γ-Glob 19.4%, AFP (-), Hbs-Ag (-), Hbs-Ab (-), R15 ICG 3.0%, S-amylase 119 S.U./dl, and U-amylase 219 S.U./dl. The results of a Meltzer-Lyon test were as follows: maximal icterus index 150 and klebsiella (+). HD (hypotonic duodenography) demonstrated a parapapillary diverticulum with an 11 × 14 diameter in the duodenum. DIC (drip infusion cholangiography) showed common bile duct dilatation. A parapapillary diverticulum was also endoscopically verified. The common bile duct had a cylindrical dilatation of 24 mm in diameter. The hepatic bile duct was slightly dilated (the largest diameter was 9 mm). No anomalous pancreaticobiliary ductal union was noted. The main pancreatic duct was slightly dilated, with a diameter of 7 mm at the head, 5 mm at the body, and 3 mm at the tail. The accessory pancreatic duct was 40 mm in length and 1.5 mm at the largest diameter. It entered into the minor papilla in the duodenal diverticulum. No stone was found. Chronic pancreatitis was suspected based on the abdominal angiography findings. No malignant findings were noted in the liver, gallbladder, or pancreas. The patient was discharged from our hospital without undergoing any surgical operation because her abdominal symptoms disappeared after admission.

Anomalous Pancreaticobiliary Ductal Union and Common Bile Duct Dilatation

Babbitt (1969) proposed that an anomalous union of the peripheral common bile duct and pancreatic duct causes common bile duct dilatation. The proposed mechanism is described hereunder. Since the common bile duct and the pancreatic duct join in an
abnormally elevated position and thus a long common channel is formed, the sphincter function of the papilla cannot reach the region of union. This formation leads to reflux of pancreatic juice into the bile duct, and is also accompanied by inflammation of the mucosa, which, in turn leads to edematous narrowing and fragility of the choledochal wall. As a result, common bile duct dilatation is induced in the upper region. We have also reported on this phenomenon (Morishita, Asakura et al., 1980; Morishita, Fujii et al., 1978;), but have suggested that the anomalous pancreaticobiliary ductal union does not always cause common bile duct dilatation. In our previous report (Morishita, Asakura et al., 1980), anomalous pancreaticobiliary ductal union was observed in eight of ten patients (80%), and not observed in two (20%). In the two patients, the bile duct and the pancreatic duct entered separately into different regions or joined in the region just proximal to the orifice. Conversely, Yoshimoto, Sakon et al. (1979) studied the anomalous pancreaticobiliary ductal union itself. Common bile duct dilatation was noted in eight of ten patients (80%) with anomalous pancreaticobiliary ductal union. No dilatation was observed in one patient, and narrow dilatation was observed in one patient with extrahepatic cancer of the bile duct.

In the present report, our patients presented with common bile duct dilatation with accompanying minor papillae in the duodenal diverticula without anomalous pancreaticobiliary ductal union. One showed cylindrical dilatation and the other showed cystic dilatation and stones.

Duodenal Diverticula and Biliary Tract Diseases

Lemmell (1934) reported on the relationship between cholepathia and duodenal diverticulum in papillen syndrome. Our colleagues (Matsuzaki 1972; Suzuki, Fujino et al., 1972; Suzuki, Takagi et al., 1974; Tsuchiya, Matsuzaki, 1969a, b; Tsuchiya, σkubo, 1965) have also investigated this relationship using HD. Bile duct disorders complicated by duodenal diverticulum were present in 16 of 30 patients (53%). The majority of both types of patients showed second parapapillary diverticula (Tsuchiya and Matsuzaki 1969a,b). Suzuki, Fujino et al. (1972) reported that parapapillary diverticula were observed in 22 of 103 patients (21%) who received HD, and cholangiopathy was observed in 20 of these 22 patients (91%). Diverticula were significantly associated with
cholangiopathy (p <0.05). On the other hand, duodenal diverticula were observed in eight of 33 patients with gall stones (24%), in four of 17 with choledocholithiasis (24%), in four of four with cholangitis (100%), in one of two with cancer of the bile duct (50%) and in two of eight with cholecystopathy (25%). That is to conclude, duodenal diverticula were present in 20 of 74 patients (27%) with diseases related to the biliary tract. When the relationship between the common bile duct and diverticula was studied, in normal bile ducts, parapapillary diverticula were noted in one of 16 patients who underwent HD (6%), while in dilated bile ducts, parapapillary diverticula were noted in six of ten patients (60%). There was a significant difference between the two groups ($\chi^2 = 6.730$). Matsuzaki (1972) described biliary tract dilatation in four of twelve patients with duodenal diverticula (33%). These findings support the possibility that the duodenal diverticulum might be associated with diseases related to the biliary tract and common bile duct dilatation. Therefore, Suzuki, Takagi et al., (1974) radiologically and histopathologically studied the papillary region in six patients (two males, four females, 42-75 years old) with common bile duct dilatation without stones with an accompanying parapapillary diverticulum, from HD, DIC, and operative findings. In these patients, the degrees of pathological conditions including inflammation, stenosis of the papilla, and changes in the peripheral site of the common bile duct were not always equal. However, there were four main causes of the primary narrowing of the papilla: 1) pressure of the diverticulum on the common bile duct, 2) dysfunction of the papilla, 3) narrowing due to the chronic fibrous changes in the papilla and 4) histological and functional ataxia of the papillary region including the diverticulum. In one patient (a 62-year-old female), the common bile duct entered into the radicular region of the diverticulum.

Papilla of Vater in the Duodenal Diverticulum

Baldwin (1911) studied four autopsied cases and three cases reported in the literature, and first described the papilla of Vater in the duodenal diverticulum. However, the papilla of Vater has more recently been reported in case studies by Suzuki, Takagi et al. (1974); Wilox, Costopoulos (1969) and Takeuchi, Miyaji et al., (1975). It has been noted in 4.4% - 6.8% of autopsied cases by Ishihara (1972) and Ishikawa (1976), as well as in 3% of a cohort of patients undergoing T-tube cholangiography (Nelson, Burheene (1976). Costopoulos, Miller, (1967) studied the papilla of Vater in the duodenal
diverticulum in twelve patients, eleven (92%) of whom had common bile duct dilatation and six (50%) of whom had common bile duct stones. Nakano, Hayakawa (1975) observed the papilla of Vater in the diverticulum of 18 of 150 patients (12%) and duodenal diverticulum diagnosed by HD, and endoscopically visualized the papilla of Vater in seven of these 18 patients (39%). In four of these seven patients (57%), disorders in the liver, the biliary tract, and the pancreas were observed, while no complications were observed in the remaining three patients (43%). However, all these patients presented with common bile duct dilatation, an anomalous course of the bile duct, and abnormalities in bile secretion.

We therefore consider the relationship between the papilla of Vater region in the duodenal diverticulum and common bile duct dilatation as follows: 1) severe inflammation from the duodenum to the biliary tract causes the diverticulum and dilatation, 2) intensive swelling of the diverticulum due to entry of duodenal contents compresses the bile duct in the duodenum and causes dilatation in the bile duct's upper region, 3) diverticulum-caused or -associated chronic inflammation of the duodenum spreading over the biliary tract causes dilatation, and 4) conversely, dilatation-caused or -associated chronic inflammation of the biliary tract spreading over the duodenal wall causes the diverticulum.

Minor Papilla in the Duodenal Diverticulum

There have been many reports on the papilla of Vater in the duodenal diverticulum. However, we rarely encounter reports on minor papilla in the duodenal diverticulum, with the exception of Ishihara's (1972) report of a single minor papilla among 65 duodenal diverticula in 41 autopsied cases. In studies by Ishikawa (1976), Simkins (1931), and Kato (1972), minor papilla are observed in 96 - 100% of autopsied cases and the accessory pancreatic duct inserts into 17 - 100% of these minor papillae. Until now, minor papillae have been considered embryological rudiments and ignored because their function was thought to have regressed, only Simkins (1931); Heiss, Shea (1978); and Mairose, Wurbs et al. (1978) have studied the minor papilla as a functional valve. Simkins (1931) studied the main and accessory pancreatic ducts of 25 autopsied cases and cases reported in the literature. In approximately 10% (3-12%) of these patients, the diameter of the accessory pancreatic duct was found to be equal to or larger
than that of the main pancreatic duct, and the accessory pancreatic ducts inserted into the minor papillae. Then, he also paid attention to the role of the minor papilla as a "safety valve." Heiss, Shea, (1978) suggested the possibility that pancreatitis could be induced by an insufficient drainage of the pancreatic juice from minor papillae. Mairose, Wurbs et al. (1978) studied blood amylase in 250 patients after ERCP. An accessory pancreatic duct was present in 12 of 48 patients (25%) who demonstrated hyperamylasemia after ERCP and accessory pancreatic duct was not present in 34 (71%) of the 48 patients. On the other hand, an accessory pancreatic duct was present in 90 of 202 patients (45%) who did not demonstrate hyperamylasemia, and was not present in 100 (50%) of the 202 patients. They reported a statistically significant difference (p < 0.01) in the appearance of accessory pancreatic ducts between the two groups and discussed the minor papilla as an "overflow valve." Matsuzaki (1972); Asakura, Morishita et al., (1978); and Morishita (1974) have studied age-related changes in the duodenum, liver and biliary tract system. The above-mentioned disorders might be accidentally induced as a result of these changes. Case 1 in the present study presented with cystic dilatation of the common bile duct. The duodenum and the common bile duct were dilated in their respective local lesions. These findings also suggest the involvement of congenital factors, including abnormalities in the development of the foregut in the embryonal stage, and local fragility of the duodenal and choledochal walls. Case 2 presented with cylindrical dilatation of the common bile duct.

We consider the relationship between the minor papilla in the duodenal diverticulum and common bile duct dilatation in light of the following: 1) extensive inflammation from the minor papilla region of duodenum to the biliary tract causes diverticulum and dilatation, 2) diverticulum-caused or -associated chronic inflammation of the duodenum, spreading over the biliary tract, with or without inflammation of the pancreatic ducts, causes dilatation, 3) conversely, dilatation-caused or -associated chronic inflammation of the biliary tract, spreading to the minor papilla region of the duodenal wall, with or without inflammation of the pancreatic ducts, causes the diverticulum, and 4) disruption of the valve function of the minor papilla, followed by a rise in pressure.
from the main and accessory pancreatic ducts to the biliary tract, with or without inflammation, causes dilatation.

The study of the minor papilla could provide a clue in determining the unknown pathological conditions of the bile and pancreatic ductal system. There were reported cases of common bile duct dilatation with papilla of Vater in the duodenal diverticulum, but no cases of common bile duct dilatation with the minor papilla in the duodenal diverticulum have been reported, probably because less advanced examination procedures were used. Simultaneous observations of the duodenum, the biliary tract and the pancreas can only be made using ERCP and/or MRCP (magnetic resonance cholangiopancreatography). Therefore, we believe that a careful study using ERCP and MRCP is required.

**Conclusion**

We experienced two patients with common bile duct dilatation, who showed minor papilla in the duodenal diverticulum without an anomalous pancreaticobiliary ductal union. Additional studies on the minor papilla will be needed to further clarify the pathological conditions of the bile duct and pancreatic duct system.

**References**


