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Endoscopic Manometry of the Sphincter of Oddi in Patients with Lemmel's Syndrome

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Abstract: Endoscopic manometry was performed to evaluate the motor activity of the sphincter of Oddi (OS) in six patients with Lemmel's syndrome, four of whom had acute cholangitis and two of whom had acute pancreatitis. As controls, 24 patients undergoing cholecystectomy without juxtapapillary duodenal diverticula (JPD) for cholelithiasis or cholesterol polyps in the gallbladder were also studied.

The OS basal pressure and contraction pressure values were 12.4 ± 5.1 mmHg and 103.4 ± 24.3 mmHg, respectively, in the patients with Lemmel's syndrome, and 19.5 ± 5.1 mmHg and 136.8 ± 28.2 mmHg, respectively, in the control patients. These differences between the groups were statistically significant; however, the wave frequency was not significantly different between the groups. The mean percentages of antegrade, simultaneous, and retrograde sequences were 37.5% ± 11.3%, 19.9% ± 8.7%, and 43.4% ± 11.7%, respectively, in the patients with Lemmel's syndrome, and 66.5% ± 11.0%, 20.2% ± 4.7%, and 14.3% ± 9.2%, respectively, in the controls. The differences between the groups were significant ($P < 0.01$) for the antegrade and retrograde sequences. These findings indicate that dysfunction of the OS in patients with Lemmel's syndrome could be important in the development of hepatocholangiopancreatic disease caused by duodenobiliary and duodenopancreatic reflux.

Key Words: Lemmel's syndrome, endoscopic manometry, sphincter of Oddi, motility

Introduction

Although juxtapapillary duodenal diverticula (JPD) have not been clearly defined, duodenal diverticula which develop within 1–2 cm from the papillae Vateri are generally regarded as JPD.[1–3] While the pathogenesis of JPD is unknown, they often accompany hepatocholangiopancreatic diseases because of their anatomical proximity to the papillae Vateri.[1–4] In this location, a JPD may cause dysfunction in the sphincter of Oddi (OS).[5] The functions of the OS are to regulate biliary and pancreatic flow into the duodenum, to prevent bile reflux into the pancreas and vice versa, and to protect against duodenoductal reflux.[6] Therefore, OS dysfunction negatively affects the flow of biliary and pancreatic secretions which may contribute to the development of hepatobiliary and pancreatic disorders. Lemmel[7] reported the presence of JPD with hepatocholangiopancreatic diseases, excluding cholelithiasis, in terms of papillen syndrome or Lemmel's syndrome. In general, patients with OS dysfunction have either decreased or increased OS pressure with common bile duct dilatation, slow drainage of the common bile duct and pancreatic duct, and an increased proportion of retrograde propagation direction of phasic contraction.[2] The purpose of the present study was to investigate the OS motor activities of patients with Lemmel's syndrome in terms of OS by endoscopic manometry.

Subjects and Methods

Subjects

The subjects of this study included six patients with Lemmel's syndrome, four of whom had acute cholangitis, and two acute pancreatitis. There were two men and four women aged between 49.0 and 72.0 years, with a mean age of 62.2 years. Diverticula were found endoscopically to be within 2 cm of the papillae Vateri, being 2.2–3.8 cm in diameter, with a mean diameter of 2.8 ± 0.8 cm, and the diameters of their common bile ducts were 1.8–3.2 cm, with a mean diameter of 2.6 ±

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0.5 cm. As controls, 24 other patients without JPD or dilatation of the common bile duct who were hospitalized to undergo cholecystectomy for cholelithiasis or cholesterol polyps in the gallbladder were also studied. The controls comprised 10 men and 14 women aged between 35 and 64 years with a mean age of 52.4 years, and none had hepatocholangiopancreatic dysfunction. Bacteriocholia was found in all of the patients with Lemmel's syndrome, but in none of the control subjects.

Methods

The patients and control subjects were fasted for at least 8 h prior to the examination. Manometry was performed without sedation or anticholinergic drugs during or just before the study, and carried out endoscopically through retrograde cannulation of the OS with a manometric catheter. OS pressure was then measured with a triple-lumen water-perfused polyethylene catheter (open-tip, Muto, Tokyo, Japan) attached via pressure transducers (carrier amplifier AP 601G, Nihon-Koden, Tokyo, Japan) to a pen recorder (polygraph system, Nihon-Koden). The triple-lumen manometric catheter had an extended diameter of 1.8 mm (5.5 Fr), a luminal diameter of 0.5 mm, and a length of 200 cm, and had three openings of 0.5 mm spaced 2 mm apart on one side. The most distal opening was 5 mm from the end of the catheter. The catheter perfusion rate was 0.25 ml/min (pneumohydraulic capillary system, Arndorfer, Greendale, WI, USA). Under this catheter perfusion rate, the pneumohydraulic capillary system yielded reproducible and reliable pressure data.[8] The OS basal pressure, the amplitude of the phasic contractions of the sphincter, and their direction of propagation were determined. The OS basal pressure was calculated by subtraction of the duodenal pressure from the OS basal pressure between phasic contractions. The amplitude of the phasic contractions was caluculated by subtraction of the OS basal pressure from the peak pressure. The direction of propagation of the phasic contractions was calculated by drawing a line between the waves recorded in the cephalad and caudad lumens, this line being positioned at the beginning of the upstroke of each wave (Fig. 1). The percentage of each type of sequence was determined after noting the direction of the line and classifying each phasic contraction as either antegrade, being toward the duodenum; simultaneous, being no propagation; or retrograde, being away from the duodenum. Obtaining prolonged tracings is often difficult due to problems encountered in maintaining catheter placement during respiration and concurrent duodenal motor activity; however, the number of phasic waves occurring simultaneously in an antegrade, simultaneous, or retrograde direction could be recorded over a period of least 10 min once the OS movements had stabilized after the first 20 min.

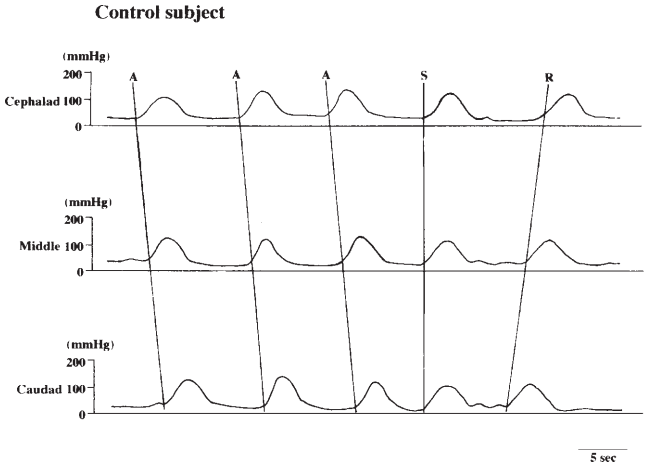

Fig. 1. Pressure profile of the sphincter of Oddi in a control subject. The control subject demonstrated a retrograde propagation direction of phasic contraction rather than an antegrade propagation direction. *A*, antegrade sequence; *S*, simultaneous sequence; *R*, retrograde sequence

Statistical Analysis

The values obtained are expressed as means ± SD. Data were analyzed by means of the chi-square test, and a *P*-value of less than 0.05 was regarded as significant.

Results

The pressure profiles of the OS in a control subject and in a patient with Lemmel's syndrome are shown in Figs. 1 and 2, respectively.

Basal Pressure of the OS

The mean basal pressure in the OS was 12.4 ± 5.1 mmHg in the patients with Lemmel's syndrome and 19.5 ± 5.1 mmHg in the controls (Table 1), being significantly lower in the former group ($P < 0.01$).

Contraction Pressure of the OS

The mean contraction pressure of the OS was 103.4 ± 24.3 mmHg in the patients with Lemmel's syndrome and 136.8 ± 28.2 mmHg in the controls (Table 1), being significantly lower in the former group ($P < 0.01$).

Table 1. Motor activities of the sphincter of Oddi assessed by endoscopic manometry

	Patients with Lemmel's syndrome	Control subjects without Lemmel's syndrome
Basal pressure	12.4 ± 5.1 mmHg	19.5 ± 5.1 mmHg*
Contraction pressure	103.4 ± 24.3 mmHg	136.8 ± 28.2 mmHg*
Frequency of contraction waves	8.7 ± 1.1/min	7.4 ± 2.3/min
Antegrade sequences	37.5% ± 11.3%	66.5% ± 11.0%*
Simultaneous sequences	19.9% ± 8.7%	20.2% ± 4.7%
Retrograde sequences	43.4% ± 11.7%	14.3% ± 9.2%*

Values are mean ± SD; * $P < 0.01$

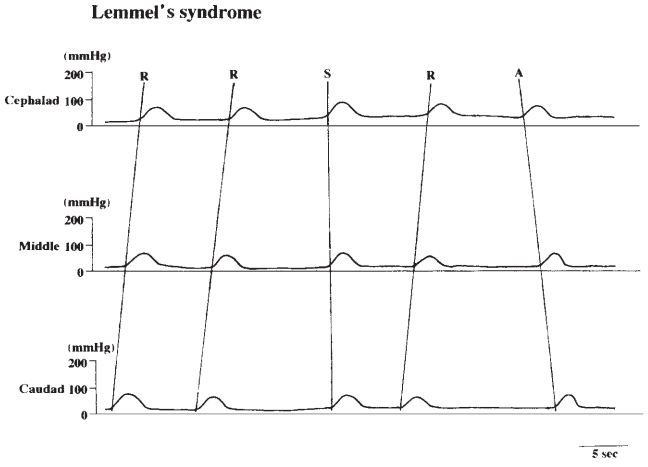

Fig. 2. Pressure profile of the sphincter of Oddi (OS) in a patient with Lemmel's syndrome. The patient with Lemmel's syndrome demonstrated a lower OS, a lower contraction pressure, and many retrograde phasic contractions compared with the control subjects. *A*, antegrade sequence; *S*, simultaneous sequence; *R*, retrograde sequence

Frequency of Contraction Waves in the OS

The mean frequency of contraction waves of the OS was 8.7 ± 1.1 cycles/min in the patients with Lemmel's syndrome and 7.4 ± 2.3 cycles/min in the controls (Table 1), without a significant difference between the two groups.

Mean Percentage of the Antegrade Sequence

The mean percentage of the antegrade sequence in the OS was 37.5% ± 11.3% in the patients with Lemmel's syndrome and 66.5% ± 11.0% in the controls (Table 1), being significantly lower in the former group ($P < 0.01$).

Mean Percentage of the Simultaneous Sequence

The mean frequency of the simultaneous sequence in the OS was 19.9% ± 8.7% in the patients with Lemmel's syndrome and 20.2% ± 4.7% in the controls (Table 1), without a significant difference between the two groups.

Mean Percentage of the Retrograde Sequence

The mean percentage of the retrograde sequence in the OS was 43.4% ± 11.7% in the patients with Lemmel's syndrome and 14.3% ± 9.2% in the controls (Table 1), being significantly higher in the former group ($P < 0.01$).

There were no serious complications caused by OS manometry. Although two of the control subjects suffered acute episodes of pancreatitis after this study, they recovered completely after 4 or 7 days of conservative therapy.

Discussion

Patients with JPD are likely to suffer regurgitation of their duodenal contents with intestinal bacteria into the bile duct and pancreatic duct, the result being that hepaticocholangiopancreatic disease may later occur. Eggert et al.[9] reported that bacteriocholia with typical intestinal bacteria was found in 38 (76%) of 50 patients with JPD. Goldman et al.[10] also reported recurrent cholangitis after biliary surgery in two patients with JPD. The pathophysiological mechanisms of JPD, including Lemmel's syndrome, have yet to be clarified, although manometric studies of the OS long ago suggested that OS dysfunction may cause hepatocholangiopancreatic disease. Miyazaki et al.[11] reported that the degree of disturbance of bile flow is proportional to the size of the JPD, and that the intermittent blockage of bile flow in patients with JPD contributes to the OS dysfunction of patients with common bile duct dilatation. On one hand, Løtveit et al.[12] reported that the muscular tone and contractile activity of the OS are lower in patients with JPD than in those without JPD, whereas other groups[13,14] stated that the muscular tone and contractile activity of the OS were significantly lower in patients without JPD than in those with JPD.

Therefore, we investigated the pathogenesis of patients with Lemmel's syndrome in terms of the aspects of OS pressure by endoscopic manometry.

In the present study we evaluated the pressure profile of the OS in patients with Lemmel's syndrome accompanied by acute cholangitis or pancreatitis. These patients demonstrated significantly lower basal pressure and phasic contraction pressure, an increased proportion of retrograde propagation direction of phasic contractions, and a decreased proportion of antegrade propagation direction of phasic contractions, compared to the control subjects. Moreover, all of the patients with Lemmel's syndrome developed both common bile duct dilatation and bacteriocholia. These findings strongly suggest that OS dysfunction contributes to the onset of Lemmel's syndrome.

The intraduodenal pressure in patients with JPD, including Lemmel's syndrome, was significantly higher than that of patients without JPD.[3] Because OS dysfunction during periods of high intraduodenal pressure severely disturbs normal bile and pancreatic flow in these patients, OS dysfunction is likely to cause or contribute to hepatocholangiopancreatic diseases with the reflux of duodenal contents into the bile duct, pancreatic duct, or both. In conclusion, the present study demonstrated that the function of the OS was impaired in patients with Lemmel's syndrome. However, such functioning is quite complicated,[5,6,14] and the modulation of OS activity is multifactorial. Further studies on patients with this disease are thus required. In the future, technical developments may increase our knowledge of the physiology and pathophysiology of biliopancreatic motility, and these advances may better define the pathogenesis of Lemmel's syndrome.

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