
◎原 著

Studies on whole gut transit in chronic pancreatitis patients

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Abstract : Abnormalities of whole gut transit could contribute to the maldigestion and digestive symptoms of chronic pancreatitis patients. Whole gut transit was measured by radiopaque markers method. Fifteen chronic pancreatitis patients (2 females, 13 males ; age range 40–78 years) and 17 controls (4 females, 13 males, 32–73 years) were studied. Additionally, we also looked for evidence of autonomic neuropathy in the chronic pancreatitis patients by using cardiovascular tests. In chronic pancreatitis, whole gut transit was shorter than controls. These abnormalities were not influenced by the degree of autonomic neuropathy. We conclude that whole gut transit is shorter in chronic pancreatitis patients.

Key words : Chronic pancreatitis, Whole gut transit.

Introduction

Chronic pancreatitis (CP) patients frequently complain of gastrointestinal symptoms such as abdominal fullness, diarrhea, dyspepsia, and abdominal pain. These symptoms are often related to irritable bowel syndrome^(1–5). However, relatively little is known about postprandial disturbances of colonic motility in CP patients^(6–10). Failure to recognize and address impaired whole gut transit, and its sequel in these populations has clinically significant implications. We think that the noninvasive method permits an

assessment of whole gut transit. We, therefore, investigated the whole gut transit using radiopaque markers. Whereas a recent study demonstrated that autonomic nerve function influences the gastrointestinal motility⁽¹¹⁾. Thus, in the present study we have evaluated whole gut transit in two groups of CP patients who differed as to whether they had or did not have signs of autonomic neuropathy affecting the cardiovascular system.

In the present study, we have used radiopaque markers to : 1) assess colonic transit time in CP patients 2) investigate the influence of autonomic neuropathy on whole gut

transit in these patients.

Materials and methods

Subjects. Fifteen CP patients (2 women and 13 men; age \pm SD = 54 ± 7 years old) and 17 controls (4 women and 13 men; 57 ± 8 years old) were studied. Nine of 17 controls were moderate alcoholic (more than 20 g ethanol every day drinker). The height and body weight of patients and controls were almost similar. Diagnosis of CP was based on radiological findings of pancreatic calcification and irregular dilatation of the pancreatic duct by endoscopic retrograde pancreatography. In all patients, routine liver function tests were within the normal range. Abnormalities in the biliary tract were ruled out by ultrasonographic examination, at the preexperimental stage. Endoscopic studies of the upper gastrointestinal tract and the total colon showed no focal lesions that would suggest the presence of gastroesophageal reflux, peptic ulceration, ulcer scarring, malignant lesions, diverticulum, or polyps in these patients. None of the patients had ever undergone pancreatic, gastric, or biliary tract surgery. The cause of the CP was alcohol abuse in 12 patients and was unknown in three. Seven of the patients had diabetes mellitus, and two required daily insulin. None of the patients had steatorrhoea or weight loss. Patients receiving drugs that would influence their gastric or gallbladder emptying and colonic motility were also excluded from study. Informed consent was obtained prior to the study from all subjects.

Study design. The study was divided into two parts. In the first part, whole gut transit was evaluated after subjects had ingested radiopaque markers. In the second part, autonomic functions were investigated using

the cardiovascular reflex test.

Whole gut transit was obtained by the following formula as discussed in detail⁽¹²⁾. Subject ingested 20 markers at 12, 24 and 48 hours before the radiograph of abdomen was taken, the three types of markers were always ingested in the same sequence. Abdominal x-rays were obtained using a high-kilovoltage fast-film technique to reduce the amount of radiation exposure. The different markers were easily distinguishable on abdominal films. The markers were counted by the same person, who was unaware of the personal profile. All subjects were asked to maintain their usual diet for the duration of the study.

The subjects were additionally investigated by two noninvasive cardiovascular reflex tests for the assessment of autonomic cardiac neuropathy: 1) variance of heart frequency at rest (150 heart actions), 2) behavior of heart frequency in orthostasis.

Statistics. Statistical comparisons were performed by Student's *t*-test. $P < 0.05$ were considered significant. Results were expressed as mean \pm SD.

Results

Whole gut Transit. Whole gut transit was faster in CP patients than controls, but the difference was not statistically significant (Table 1).

Autonomic Cardiovascular Neuropathy. Five of the 15 CP patients had autonomic neuropathy as diagnosed by cardiovascular reflex tests. There were no evidence of an association between impaired whole gut transit and this finding (Table 2).

Table 1. Number of each marker

	48 hours before x-ray	24 hours before x-ray	12 hours before x-ray
Chronic pancreatitis patients (n=17)	4.6 ± 3.2	14.9 ± 4.7	15.2 ± 3.7
Controls (n=17)	7.0 ± 4.8	16.2 ± 3.1	19.0 ± 1.0

Table 2. Relationship of autonomic nerve function with whole gut transit markers among chronic pancreatitis patients

	48hours	24hours	48hours
patients with autonomic neuropathy (n=5)	4.3±2.3	15.4±4.3	15.6±4.3
patients without autonomic neuropathy (n=12)	4.7±3.3	14.5±4.8	15.0±3.5

Discussion

In this study, we have documented for the first time the motility of whole gut transit in CP patients. Our result indicated that in patients with CP, whole gut transit was faster than in controls.

Recent reports showed that gastrointestinal motility was affected by autonomic neuropathy in the patients of diabetes mellitus⁽¹³⁾, chronic renal failure⁽¹⁴⁾ and other several disease⁽¹¹⁾. In this study, whole gut transit in the patients of CP appeared to be unrelated to the severity of the underlying autonomic nerve function, as measured by cardiovascular reflex test so that autonomic nerve function did not play major factor of whole gut transit in CP patients.

The methods employed to assess whole gut transit time have been well described and validated⁽¹⁵⁾. Early diagnosis and management of gastrointestinal motility disorder was an important factor in the care of patients with CP.

Whole gut transit was shorter in CP patients, although the difference was not significant. There were several reports⁽¹⁵⁾ on the effect chronic ethanol on colonic motility, and the results were similar to our data. They suggested that rapid transit time in alco-

holics could contribute to transient diarrhea. In our subjects, none of them had experienced diarrhea during the experimental period. In accordance with previous reports⁽¹⁶⁾ patients with severe exocrine pancreatic insufficiency emptied the test meal more rapidly from the stomach and transit the test meal more slowly in the small intestine than did controls. However, another report⁽¹⁷⁾ indicated that patients of CP had a shortened mouth-to-cecum transit time. The mechanism of slightly faster colonic transit in CP patients was not clear.

References

1. Camilleri M, Thompson DG, Malagelada J-R : Functional dyspepsia. Symptoms and underlying mechanism. *J Clin Gastroenterol* 8 : 424-429, 1986.
2. Talley NJ, Phillips Sf : Non-ulcer dyspepsia. Potential causes and pathophysiology. *Ann Intern Med* 108 : 865-879, 1988.
3. Dodds WJ : Biliary tract motility and its relationship to clinical disorders. *AJR* 155 : 247-258, 1990.
4. Thompson WG : Gastrointestinal symptoms in the irritable bowel compared with peptic ulcer and inflammatory bowel disease. *GUT* 25 : 1089-1092, 1984.
5. Marcus SN, Heaton KW : Irritable bowel-

- type symptoms in spontaneous and induced constipation. *GUT* 28 : 156–159, 1987.
6. Regan PT, Malagelada JR, Dimagno EP, Go VLW : Postprandial gastric function in pancreatic insufficiency. *Gut* 20 : 249–254, 1979.
 7. William BL, Jordan BW : Rapid gastric emptying of fatty meals in pancreatic insufficiency. *Gastroenterology* 67 : 920–925. 1974.
 8. Takayoshi M, Tooru S, Junya K, Yoshifumi K, Masaru K, Takayoshi T : Gallbladder emptying to endogenous and exogenous stimulation in chronic pancreatitis patient. *Am J Gastroenterology* 89 : 225–231, 1994.
 9. Masclee AAM, Jansen JBMJ, Corstens FHM, and Lamers CBHW : Reversible gall bladder dysfunction in severe pancreatic insufficiency. *Gut* 30 : 866–872, 1989.
 10. Glasbrenner B, Malferteiner P, Pieramico O, Klatt S, Riepl R, Friess H, and Ditschuneit H : Gallbladder dynamics in chronic pancreatitis. *Dig Dis Sci* 38 : 482–489, 1993.
 11. Bharucha AE, Camilleri M, Low PA, Zinsmeister AR : Autonomic dysfunction in gastrointestinal motility disorder. *Gut* 34 : 397–401, 1993.
 12. Amanda MM, Sidney FP, Alan RZ, Robert LM, Robert WB, Bruce GW : Simplified Assessment of Segmental colonic Transit. *Gastroenterology* 92 : 40–47, 1987.
 13. Werth B, Meyer-Wyss B, Spinass GA, Drewe J, Beglinger C : Non-invasive assessment of gastrointestinal motility disorder in diabetic patients with and without cardiovascular signs of autonomic neuropathy. *Gut* 33 : 1199–1203, 1992.
 14. Dumitrascu DL, Barnert J, Kirschner T, Wienbeck M : Antral emptying of semisolid meal measured by real-time ultrasonography in chronic renal failure. *Dig Dis Sci* 40 : 636–644, 1995.
 15. Ali Keshavarzian, Jeremy Z. Fields : Gastrointestinal motility disorders induced by ethanol. *In Alcohol and the gastrointestinal tract*, Victor R. Preedy, Ronald R. Watson (eds). Florida, CRC Press. Inc., 1996, pp 235–253.
 16. C Johansson, DN Schmidt, PM Hellström : Changed integrated gastrointestinal response to a mixed meal in exocrine pancreatic insufficiency. *Pancreas* 7 : 205–211, 1992.
 17. B Lembcke, B Kraus, PG Lankisch : Small intestinal function in chronic relapsing pancreatitis. *Hepato-gastroenterol* 32 : 149–151, 1985.

慢性膵炎患者の全消化管通過に関する研究

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慢性膵炎の患者の自覚症状や消化不良には消化管
の通過異常が関与している可能性がある。慢性膵

炎患者15例と対象群17例で全消化管通過を測定した。さらに慢性膵炎患者は自律神経機能について評価した。全消化管通過は慢性膵炎患者は対象群より早いことが示された。しかしその機序として推定された自律神経機能異常の有無では全消化管通過に差は認められず、慢性膵炎の消化管機能異常の原因は自律神経異常ではないと推定された。

キーワード；慢性膵炎, 全消化管通過