

## Diabetic Gastroparesis: Experience with Surgical Treatment\*

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### Diabetic Gastroparesis

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Diabetic gastroparesis는 희귀한 질환중에 속하며 그 정확한 빈도에 대해서는 보고된 것이 없는데 그 주 원인은 당뇨병 환자에 있어서 위장관의 병리학적인 병변은 비교적 광범위하게 나타 나지만 그 중 극소수의 환자만이 임상적 증상을 나타내어 치료요를 요하게 되기 때문이다.

주 증상은 위내온 정제에 의한 구토, 조기 포만감과 복부팽만인데 음식이 소장으로 내려가지 않음으로 해서 혈청당의 조절도 위란하지 않게된다. 이 병의 원인을 설명코저 여러 가지 의견이나와 있지만 어느 하나도 병리학적인 조직변화와 임상적 증상과의 사이에 납득할만한 상관관계를 제시하지 못하고 있다.

방사선 상에는 위가 팽대되어 있으며 연동수축운동이 감소되어 있으나 위장 폐색증은 없는 것이 특징이다. 심한 diabetic gastroparesis 환자의 내과 및 외과적인 치료중 특이한 외과적인 경험을 하였기에 문헌을 고찰하고 그 증례를 보고한다.

### Introduction

Diabetes mellitus affects all parts of the gastrointestinal tract, but frequently causes no clinical manifestations at all. Despite extensive pathologic involvement of the gastrointestinal tract, only a small percentage of patients become symptomatic and no definite data are available on the overall frequency of gastrointestinal manifestations in diabetes mellitus. Numerous mechanisms have been proposed to account for the functional and structural abnormalities associated with the GI tract in diabetes mellitus: autonomic visceral neuropathy, diabetic microangiopathy, electrolyte imbalance such as hypokalemia and hyperkalemia, altered hormone production of insulin and glucagon,

and increased susceptibility of diabetic patients to secondary infection.<sup>1-3)</sup>

None of these fully explains GI manifestations of diabetes mellitus and there is a very poor correlation between structural changes and the functional aberrations.

Gastric retention of food due to delayed emptying of the stomach in diabetics was first described by Rundles.<sup>4)</sup> This state of atony and disturbed expulsion of stomach contents is usually insidious and frequently without any definite symptoms.<sup>5)</sup>

In 1958 Kassander reported "gastroparesis diabeticorum" as atony and delayed emptying of the stomach in patients with diabetes mellitus.<sup>6)</sup> Since this report it has become recognized that gastric motor abnormalities occur in 20 to 30% of diabetics, often without

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clinical manifestations.

Radiographically the stomach is large and distended and there is marked gastric retention for hours or even days. There is no evidence of obstruction, the retention apparently resulting from the reduction of peristaltic contractions. The motor activity of the stomach is impaired, resulting in absence of the usual three contractions per minute, or reduction of the peristaltic amplitude of those waves that are present. Several entities may radiographically simulate diabetic gastric neuropathy: autovagotomy due to tumor invasion of the vagus nerve, surgical vagotomy, and drug induced motility disturbances (tranquilizers especially diazepam, anticholinergics, ganglio-blocking agents, and tricyclic antidepressants).<sup>6,7)</sup>

For treatment, strict diabetic control is important. Cholinergic agent such as Bethanechol<sup>8)</sup> and the cholinesterase inhibitor ambenonium chloride<sup>6)</sup> have met with variable success, but are often associated with disturbing side effects. Operative drainage of the atonic stomach, either with a gastroenterostomy, or pyloroplasty, has occasionally proved to be effective in diabetic gastroparesis.<sup>9)</sup> Metoclopramide<sup>8~11)</sup> also has been used with various results.

The prognosis of patients with gastroparesis diabeticorum is guarded, as these patients usually have advanced complications of diabetes mellitus.

### Case Report

The patient is a Caucasian male, born on July 12, 1907. The patient has been obese all his life but otherwise healthy until 1972 when he had an attack of acute gouty arthritis. Diagnosis of diabetes mellitus was made in April 1973 and he was initially treated with anti-diabetic diet and an oral hypoglycemic agent. A 4 cm diameter abdominal aortic aneurysm was found in May of 1973, and a left carotid endarterectomy was required in 1974 and a transurethral resection of the prostate

was performed for him in June 1978. A 5cm diameter left renal cyst was found and the size of abdominal aortic aneurysms was 5 to 6cm at the time of prostatic surgery. In March of 1979, the patient was admitted to the hospital because of obstructive jaundice and cholecystectomy and common duct exploration followed by choledochojunostomy for choledocholithiasis was performed but at the same time the possibility of carcinoma of the head of pancreas was suspected but not confirmed. Postoperatively patient developed cerebral thrombosis with right hemiparesis but this condition rapidly improved. His diabetic condition was stable but required 30 units of NPH daily. In September of 1979, the patient experienced a hypertensive crisis and repeated cerebrovascular accident accompanied by unconsciousness of several days, grand mal seizures, aphasia, and right hemiparesis. His diabetic condition became very unstable at the same time and required 75 units of insulin daily soon after the cerebrovascular accident and the insulin requirements fluctuated widely afterwards. In December of 1979, he developed insulin resistance and his insulin requirement was up to 320 units daily until it was stabilized at 40 units daily level after oral steroid therapy and the use of pure porcine insulin. His neurological status steadily improved so that no deficit was present by December of 1981 and his general condition was satisfactory except gradual weight loss to 146 pounds from 182 pounds in June of 1981 which the patient assumed to his strict 1800 cal anti-diabetic diet. In March of 1982, patient underwent exploratory laparotomy because of continuous loss of weight to 136 pounds, increasing CEA level to 63 ng/ml and abnormal liver scan which suggested possible metastatic disease. Alkaline phosphatase and gamma-glutamyl transferase levels were elevated at this time. A mass in the head of the pancreas in addition to but not separable from the abdominal aortic aneurysm was found and biopsy specimens were taken from the mass, enlarged peripancreatic lymph nodes and the liver. Tissue report of the mass

revealed fibrosis and chronic inflammation and no pathological findings were noted in the lymph nodes. Nonspecific reactive hepatitis was present on sections of the liver biopsy.

On May 28, 1982, this man was readmitted to the hospital because of severe "gas" problem in the stomach, abdominal distention, weakness, poor appetite from continuous bloated feeling, and further weight loss. Blood pressure was 118/68, temperature 68.4°F, pulse 84 and regular, respiration 20, height 5' 8", and body weight 123 pounds. Other than signs of significant weight loss and right subcostal incisional scar and 6 cm abdominal aortic aneurysm, physical examination was essentially negative. Hemoglobin was 14, hematocrit 41%, WBC 6,200 with normal differential counts, glucose 366, BUN 38, total protein 6.3, albumin 3.6, bilirubin and SGOT normal, and GGTP slightly elevated. Upper GI series suggested pyloric channel ulcer with edema but esophagogastroduodenoscopy showed diffuse gastritis with friable mucosa with large amount of gastric fluid, 900 ml and partially digested foods in the stomach after overnight fasting but no pyloric channel ulcer or narrowing of the pylorus was found. Patch erythema with mucosal erosion was also present in the distal half of the esophagus. A small papillary type mucosal lesion was also seen in the region of ampulla Vater but multiple biopsies showed only chronic inflammation and reactive changes. No duodenal narrowing was present. Initial response to medical therapy including Cimetidine, Mylanta and frequent small feedings of bland diet was tolerated first few days then he started to have increasing abdominal discomfort, "gas", and bloatedness followed by frequent vomitings. Total parenteral hyperalimentation was started. Trials of removing nasogastric tube with oral liquid administration was not successful and nasogastric tube output remained high. Repeated Upper GI series showed no mechanical obstruction at the gastric outlet or in the duodenum but only slow and weak peristaltic waves were present in the stomach. Passage

of contrast material through small bowel and colon was normal. Gastrojejunostomy was performed on June 26, 1982 because of prolonged gastric distress and vomitings, hoping that this will empty the stomach more effectively. The result was disappointing. The high gastric output continued even after the patency of the anastomosis was verified by the contrast study and endoscopic study. The stomach continued to be large with retention of fluids and no symptomatic relief was achieved.

Metoclopramide was tried without benefit, then Bethanechol was also tried but the effect was only temporary. Subsequently, gastrostomy tube for gastric drainage and jejunostomy tube manufactured by Vivonex maker for feeding purpose were inserted and parenteral hyperalimentation was substituted by jejunostomy feeding using Vivonex completely by July 27, 1982. At this time his insulin requirement was reduced to 30 units of porcine Lente insulin daily in divided doses. The gastric output ranged between 200 to 1000 ml per eight hour period. An attempt was made to reinfuse the gastric output into the jejunostomy tube but failed due to the small lumen of the Vivonex jejunostomy tube. This tube did not allow any blenderized food feeding for the same reason. Gradually patient developed feeding problems and subsequently experienced numerous hypoglycemic reactions and when this tube was accidentally dislocated and lost, a 16 French Foley catheter was inserted instead on September 1, 1982. The patient tolerated the feeding much better with this new tube and the gastric drainage could be diverted into the jejunum using a connecting tube during the interval between feedings. The patient tolerated this arrangement well until October 25, 1982 when he required another admission to the hospital. His general condition rapidly deteriorated during this admission and ultimately expired on November 25, 1982. At autopsy, a well differentiated adenocarcinoma arising in the head of pancreas with direct extension and metastasis to peripancreatic and porta hepatis lymph nodes,

inferior vena cava, right renal hilum, aorta, celiac artery and superior mesenteric artery, and adrenals. No hepatic metastasis was present. No luminal obstruction was present in the gastrointestinal tract and choledochojejunostomy and gastrojejunostomy were patent. Three liters of fibrinous ascites was also present. An 8 cm abdominal aortic aneurysm and bilateral 3 cm iliac aneurysms were also present. Severe atherosclerosis of coronary arteries and severe arterionephrosclerosis and atheromatous emboli were also present.

### Discussion

Patients with diabetic gastroparesis have vomitings, early satiety, and abdominal distention. Gastric stasis decreases the delivery of food to the small bowel and may disturb the insulin control of serum glucose. Various medications including Metoclopramide,<sup>8,10,12)</sup> Domperidone,<sup>12)</sup> and Bethanechol<sup>8)</sup> have been used to improve the condition with less than favorable responses. Surgical therapy was only occasionally utilized with questionable results. Gastrojejunostomy never functioned effectively for the patient reported here. Parenteral hyperalimentation may be the only long term method to maintain the nutrition of these patients with severe gastric motility problems and may control their diabetic condition better since the caloric intake and therefore the insulin requirements can be adjusted more easily. Still these patients require some form of drainage procedure to relieve their symptoms of bloated feedings and repeated vomitings. Since gastrojejunostomy or pyloroplasty does not function due to lack of gastric peristalsis, a tube drainage as dependent drainage has to be used. If small intestine is not involved with diabetic neuropathy, especially early in the course of disease, these patients can maintain their nutritional status and they should have much less water and electrolyte disturbances and much less subjective symptoms by diverting the gastric output into the jejunum by connecting the gastrostomy tube and the

jejunostomy tube. The jejunostomy tube should be of sufficient size so that any type of foods can be blenderized and fed and diverting the gastric juice which very often contains many particulate material can be done easily. The prognosis of severe diabetic gastroparesis is guarded, but with combination of regimen available, we can provide better care for these unfortunate patients.

### Summary

A unique surgical experience with a patient with severe diabetic gastroparesis is presented. This patient was initially treated with gastrojejunostomy without any benefit. Metoclopramide and bethanechol were also used but only brief relief of symptoms obtained.

Total parenteral hyperalimentation was also utilized and found to be most effective. This patient still required insertion of gastrostomy tube for dependent drainage of the stomach contents to relieve his symptoms and at the same time a jejunostomy tube was inserted for feeding purpose. These two tubes were also used to divert gastric drainage into the small intestine by connecting these two tubes between feedings.

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