

# Chest Pain and ST Segment Depression Caused by Expansion of Gastric Tube Used for Esophageal Reconstruction

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## Abstract

**We describe a 76-year-old man in whom a gastric tube was used for esophageal reconstruction via the anterior mediastinum after esophagectomy for esophageal cancer. Stenosis of the tube resulted in fluid accumulation, which directly compressed the heart and caused angina-like chest pain associated with ST-segment depression in lead V<sub>2-3</sub> on the electrocardiogram (ECG). Coronary angiography revealed no stenosis. Drainage of the fluid in the gastric tube resulted in immediate relief of symptoms and normalization of ECG. Angina-like chest pain associated with ST segment changes were caused by expansion of the gastric tube and compression of the heart. (Internal Medicine 44: 217–221, 2005)**

**Key words:** esophageal cancer, complication, ST change, CAG, balloon plasty

## Introduction

During radical surgery for esophageal cancer, a gastric tube is often used as the reconstruction structure to replace the excised esophagus. In the early postoperative period, certain complications associated with surgery itself may occur, such as suture failure, infection and anastomotic stricture. Late complications may also occur, which are related to the reconstructed gastric tube, i.e., problems of oxyntic capacity, retention capacity and passage of food. In particular, it was reported that tumor recurrence and anastomotic stricture might be correlated with the major complications and decrease the quality of life (1). However, there are only a few

reports of cardiopulmonary complications (2). We report a patient who developed clinical symptoms and electrocardiographic (ECG) changes resembling angina pectoris caused by expansion of the gastric tube in the late postoperative period after reconstructive surgery for esophageal cancer.

## Case Report

A 76-year-old man underwent reconstructive surgery using a gastric tube via the anterior mediastinum after esophagectomy for esophageal cancer in 1998. No signs of recurrence of cancer or any other related symptoms were noted. In April 2001, after breakfast, the patient complained of chest pain, but the symptoms were transient and disappeared after a short period of time. After lunch on the same day, the same symptoms occurred again, and he was referred to our hospital by an ambulance. On arrival, the symptoms had already disappeared. Physical examination showed blood pressure (BP) of 135/74 mmHg with a regular heart rate of 60 beats/min. There was no jugular venous distention, heart sounds were normal and no murmur was audible.

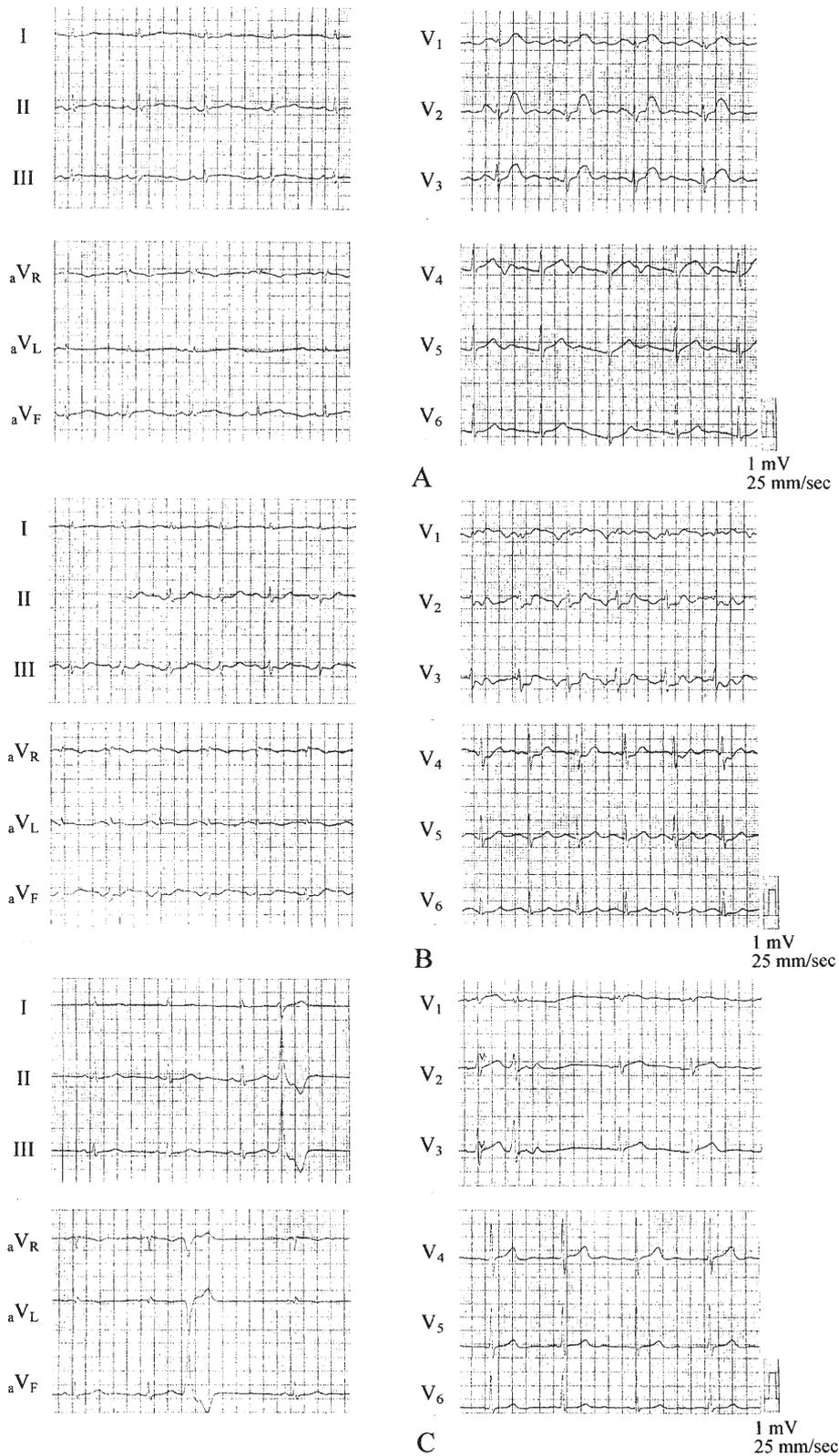
Plain X-ray films showed clear lung fields, abdomen and no specific findings in both lower extremities. The ECG showed normal sinus rhythm and T-wave mild elevation and notch of ST segment in leads V<sub>1-3</sub>, U wave and no ST segment depression (Fig. 1A). An echocardiogram showed normal motion of the left ventricular wall and no valvular disease, but a hypoechoic structure ventral to the right ventricle was noted (Fig. 2A). Laboratory test revealed slight elevation of CPK, but no elevation of troponin T. One hour after the arrival to the hospital, the chest pain reappeared, and the patient collapsed with a systolic BP of 66 mmHg with a regular heart beat at rate of 94 beats/min. ECG revealed ST segment depression in leads V<sub>2-3</sub> (Fig. 1B). No

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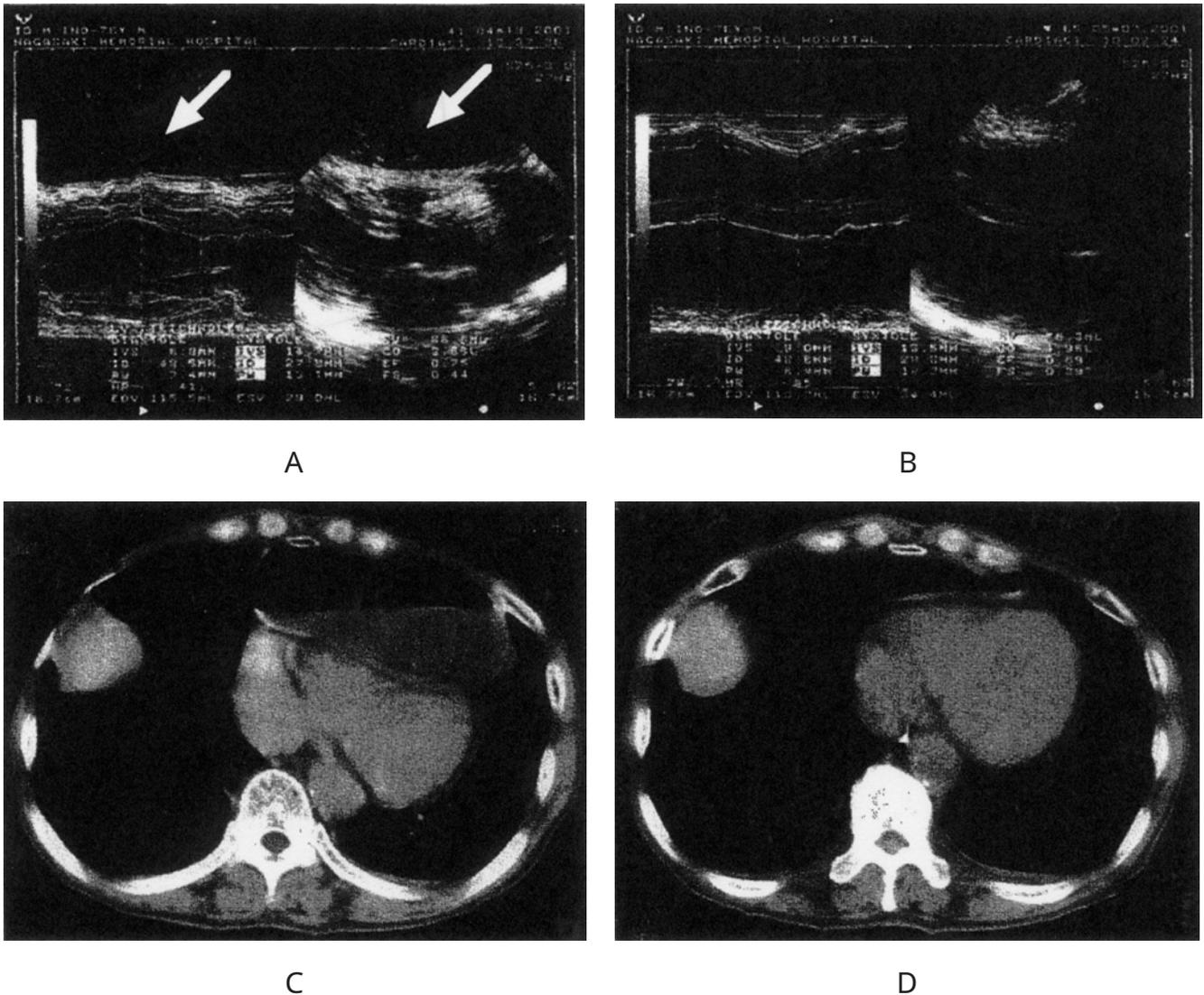
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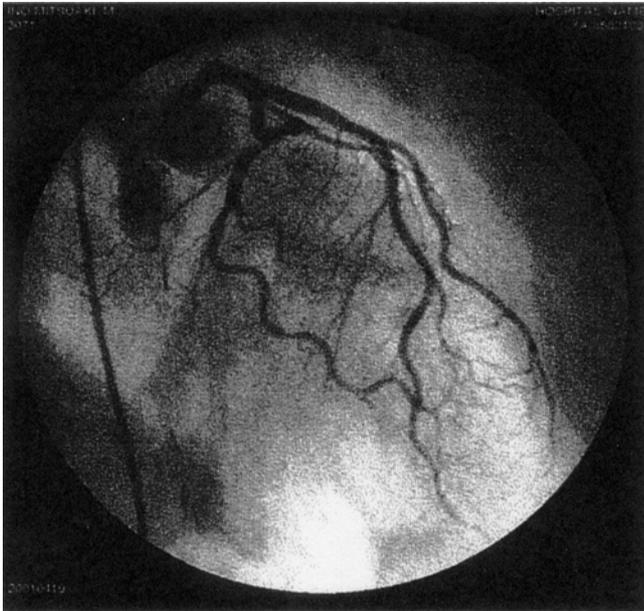
**Figure 1. Electrocardiography. A:** On admission, the ECG showed normal sinus rhythm and mild elevation of T-wave and notch of ST segment in leads V<sub>1-3</sub>, U wave and no ST segment depression. **B:** ST segment depression was revealed in leads V<sub>2-3</sub> before coronary arteriography. **C:** ST changes disappeared after drainage of excess fluid in the gastric tube.



**Figure 2. Echocardiography.** A: An echocardiogram showed hypo echoic structure (55 mm) ventral to the right ventricle on admission (arrows indicated). RVDD (right ventricular dimension end-diastole) 3 mm, LVDD (left ventricular dimension end-diastole) 49.5 mm, LVDs (left ventricular dimension end-systole) 27.8 mm, SV (stroke volume) 86.5 ml, LVEF (left ventricular ejection fraction) 75%. B: An echocardiogram showed a decrease in hypo echoic structure after balloon plasty of gastric tube. RVDD 25 mm, LVDD 48.6 mm, LVDs 29.8 mm, SV 76.3 ml, LVEF 69%. C: Chest computed tomography (CT) showed massive collection of fluid in the anterior mediastinal gastric tube, which compressed the heart posteriorly during the development of angina-like chest pain. D: Chest CT demonstrated no signs of compression of the heart and no dilatation of the gastric tube after balloon plasty.

arrhythmias occurred during the monitoring. We considered the condition as an angina pectoris, and performed coronary angiography (CAG). However, CAG showed no stenotic lesions (Fig. 3A and B). Despite these findings, BP remained at 55–84/28–48 mmHg with chest pain. After completion of the CAG, the patient suddenly vomited, and chest computed tomography (CT) revealed massive collection of fluid in the anterior mediastinal gastric tube, which compressed the heart posteriorly (Fig. 2C). A tube was inserted for drainage of the fluid accumulating in the gastric tube. It resulted in immediate relief of the chest pain, normalization of BP to 140/80

mmHg, and disappearance of ECG changes (Fig. 1C). Based on the above clinical course, we considered the symptoms and ECG changes resembling angina attack were caused by compression of the heart by the expanded gastric tube (Fig. 4). Subsequently, a balloon plasty for the stenotic area of the pylorus was performed. After balloon plasty, echocardiogram (Fig. 2B) and chest CT (Fig. 2D) demonstrated no signs of compression of the heart and no dilatation of the gastric tube.

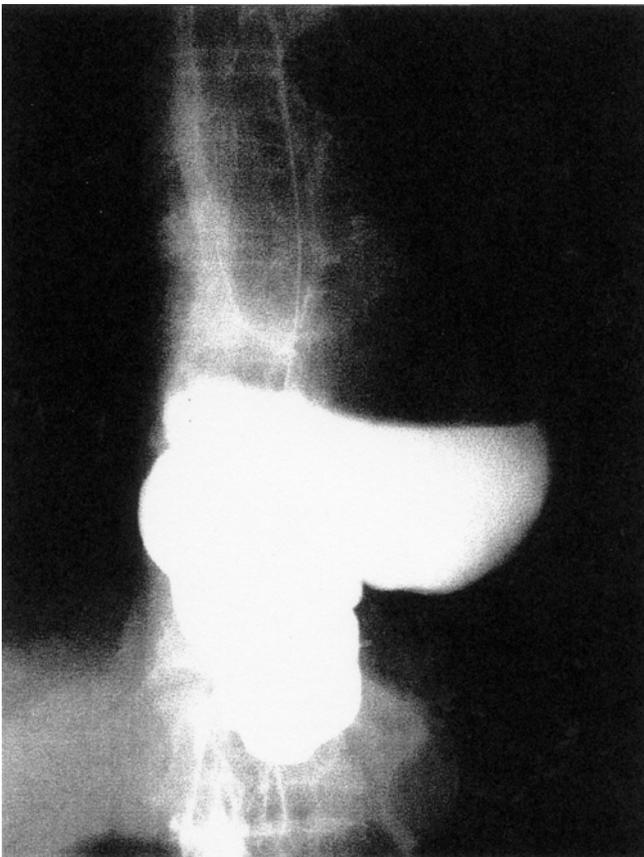


A



B

**Figure 3.** Coronary arteriography showed no stenotic lesion during development of the angina-like chest pain. A: left coronary artery, B: right coronary artery.



**Figure 4.** Esophagogram before balloon plasty showed expansion of the gastric tube by distal site stenosis.

## Discussion

Four routes are used to bypass the esophagus after esophagectomy; subcutaneous-presternal, anterior mediastinal-retrosternal, posterior mediastinal and the prepulmonary left transpleural route. The anterior mediastinal route is the most commonly used and a gastric tube is the most commonly used reconstruction structure at present (3). Previous studies have indicated that the rates of postoperative pulmonary complications do not differ between anterior mediastinal and posterior mediastinal routes (4). However, anterior mediastinal reconstruction is associated with certain cardiopulmonary complications, such as a decrease of cardiac index and atelectasis caused by mechanical compression (5). Furthermore, arrhythmia occurred in 4 to 47% of patients who underwent esophagogastrectomy for carcinoma (2, 6). However, to our knowledge, there is no report of angina-like chest pain accompanied by ECG changes in patients with a gastric tube after esophagectomy.

Pectus excavatum and mediastinal tumor could also compress the cardiac muscle. The most common ECG findings associated with pectus excavatum are negative P wave in lead V<sub>1</sub>, which is fairly normalized after sternal turnover operation, and incomplete right bundle branch block pattern related to the leftward displacement of the heart (7). Anterior mediastinal tumors could also compress the heart (8, 9), and Tarin et al (10) reported ECG findings similar to the Brugada syndrome pattern, but no angina-like ST segment changes were noted in those patients. The present patient developed angina-like chest pain and ST segment changes on the ECG.

We consider that the ECG changes resembling angina pectoris in this case were probably caused by sudden hemodynamic changes due to the rapid expansion of the gastric tube.

The mechanism of ST depression has not been satisfactorily elucidated. Li et al (11) suggested that the ST changes are due to sarcolemmal ATP-sensitive potassium channel on the myocardial cell wall. When there is epicardial ST-segment depression, it has been considered to be caused by an injury current flowing in the underlying subendocardium (12). Right precordial ST depression is occasionally observed in inferior myocardial infarction (13) and acute pulmonary thromboembolism (14). In the present case, these ECG changes might be an artifact of cardiac motion, because we observed change in the ECG baseline in each heart beat. In the meantime, ischemia by compression might be related to ECG change, although we could not prove it directly. While we could not completely exclude coronary artery spasm in our patient, this was an unlikely mechanism due to the immediate relief of symptoms and normalization of ECG following drainage of the excessive amount of fluid that accumulated in the gastric tube.

There is controversy about pyloroplasty of reconstructive gastric tube. Bemelman et al (15) reported that pyloroplasty does not prevent delayed gastric emptying after esophageal substitution. On the other hand, Fok et al (16) recommended pyloroplasty for patients in whom the whole stomach is used for reconstruction after esophagectomy, because no significant increase in complications were found in those patients who underwent pyloroplasty. In the present case, pyloroplasty was not performed after reconstructive gastric tube, but a balloon plasty of the pylorus was performed and the patient was instructed to have small but frequent meals rather than three large meals a day to prevent possible formation of an obstacle to food passage. Actually, no symptoms have appeared since then.

This is the first report of angina-like chest pain associated with ST segment changes caused by expansion of the gastric tube and compression of the heart. Gastric tube expansion should be considered as a cause of sudden angina-like chest pain in patients who have undergone reconstruction surgery of the gastric tube.

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