Coronary Spasm Complicating Sclerotherapy of Esophageal Varices*  

Min-Ji Charrng, M.D.; Shih-Pu Wang, M.D.; Mau-Song Chang, M.D.; and Benjamin N. Chiang, M.D.

There are rare serious cardiac complications associated with endoscopic examination. An episode of coronary artery spasm developed in a 68-year-old man during endoscopic sclerotherapy for esophageal varices. The coronary artery spasm may have been triggered by a reflex increase in sympathetic discharge under stressful circumstances, and may occur most often in patients with preexisting heart disease. In patients with severe cardiac disease, ECG monitoring during the procedure seems justified.

Endoscopic sclerotherapy has gained wide acceptance for the treatment of bleeding esophageal varices. Reports of complications associated with endoscopic sclerotherapy include ulceration, stricture, esophageal perforation, fever, pleural effusion and mediastinitis.

One episode of coronary artery spasm after sclerotherapy is reported.

**Case Report**

A 68-year-old man with cirrhosis of the liver and repeated episodes of esophageal variceal bleeding received endoscopic variceal sclerotherapy at the clinic for four consecutive months, uneventfully. His other known medical problems included labile hypertension, gouty arthritis and an old anterior myocardial infarction. His previous electrocardiogram, taken on January 3, 1986, showed QS pattern in leads V1 to V6, and rS pattern in leads 2, 3 and aVF. He denied anginal pain or any other symptom traceable to the cardiovascular system. The fifth sclerotherapy was performed on August 5, 1986, using an Olympus injector (NM-1/3) and the varices were injected at three sites with a total of 10 ml of 5 percent ethanolamine oleate. Immediately after the injection, the patient suffered severe chest pain, and his systolic blood pressure fell to 40 mm Hg. The electrocardiogram showed 2 mm S-T segment elevation with disappearance of r waves in leads 2, 3 and aVF. Reciprocal S-T segment depression was present in leads 1 and aVL (Fig I). He twice received sublingual nitroglycerin 0.6 mg at 15-minute intervals. The S-T segment returned to baseline 30 minutes later. The patient was immediately transferred to the coronary care unit. Blood pressure on arrival was 90/70 mm Hg and the physical examination was unremarkable. A chest X-ray film revealed a cardiothoracic ratio of about 50 percent, without other abnormal findings. No further ST-T changes and arrhythmias were detected during ECG monitoring in the coronary care unit. However, an electrocardiogram taken one day later showed the r deflection had reappeared in leads 2, 3 and aVF. Maximal creatine kinase was 1,642 U/L (normal <120 U/L), peaking at the second day and then declining to normal level within three days. All CK isoenzymes were of MM type. A two-dimensional echocardiogram showed an enlarged left atrium and left ventricle and anteroseptal hypokinesis. The patient had an uncomplicated hospital stay thereafter. He refused to receive cardiac catheterization, and was discharged in stable condition.

*From the Division of Cardiology, Department of Medicine, Taipei Veterans General Hospital, Taipei, Taiwan.

Reprint requests: Dr. Charrng, Division of Cardiology, Veterans General Hospital, No. 201, Sec 2, Shih-Pu Road, Taipei, Taiwan, ROC 11217

---

**REFERENCES**


Gastrointestinal endoscopy is becoming increasingly important and popular, with rare complications. Serious cardiac complications are surprisingly infrequent, and minor ones are usually not appreciated unless electrocardiographic monitoring is carried out. The cardiac mortality rate associated with endoscopy was 0.002 percent in a large retrospective survey.1

Esophageal spasms can cause angina-like chest pain suggestive of coronary artery disease. Anxiety and stress are common precipitating factors, and the chest pain in both conditions may be relieved by administration of nitroglycerin and provoked by ergonovine.23 However, the typical ECG S-T segment elevation in the inferior leads with reciprocal S-T segment depression in I and aVL leads which returned to baseline 30 minutes later strongly suggests that coronary artery spasm was the important cause of the event in this patient. There was transient appearance of Q waves in the inferior leads during the attack of chest pain. Evidence from experimental and clinical reports shows that transient Q waves can be produced by myocardial ischemia alone in the absence of myocardial infarction.56 Transient Q waves have also been demonstrated in patients with Prinzmetal’s angina during episodes of chest pain.7 This patient had impaired left ventricular function from an old anterior myocardial infarction. When further impairment of blood supply to the right coronary artery put large areas of the myocardium in jeopardy, there was hemodynamic instability from which recovery occurred after the coronary spasm was relieved.

The mild elevation of creatine kinase may have originated from esophageal, rather than myocardial, damage since all the CK isoenzymes were of MM type.

Ethanolamine oleate is derived from fatty acid. Potential allergic reaction to it should be considered. Although it had been repeatedly used for five times in four months, no similar attack had occurred before. Allergic reaction is unlikely, but cannot be totally ruled out. Ethanolamine oleate itself may have some cardiotoxic effect, such as decreasing cardiac output, hypotension and bradyarrhythmia.8,9 Whether these are from similar or different underlying mechanisms remains speculative.

Coronary arteries are richly supplied with alpha-adrenergic receptors, and sympathetic stimulation results in a coronary spasm. There is evidence that stress and apprehension provoke the release of catecholamines which are responsible for ST-T changes during endoscopy.10 The incidence of ischemic ST-T changes was 23.6 percent on continuous electrocardiographic monitoring during endoscopy, and these changes occurred most often in patients with underlying heart diseases.10 Although, fortunately, these disturbances remit spontaneously in most patients, some serious complications such as acute myocardial infarction have been noted.10 Since endoscopic sclerotherapy has become a widely accepted method of treating bleeding esophageal varices, there are increasing possibilities of treating a patient with varices who may have coronary artery disease. ECG monitoring of these potential life-threatening complications during sclerotherapy seems justified. Further investigation to determine the underlying mechanism disturbing the cardiovascular system is mandatory.

REFERENCES

2 Palmer ED, Wirts CW. Survey of gastroscopic and esophagogastroduodenoscopic accidents. JAMA 1957; 164:2012-16
5 Gross H, Rubin IL, Laufer H, Bloomberg AE, Bujdosol L, Delman AJ. Transient abnormal Q waves in the dog without myocardial infarction. Am J Cardiol 1964; 14:669-74
6 Roessler H, Dressler W. Transient electrocardiographic changes identical with those of acute myocardial infarction accompanying attacks of angina pectoris. Am Heart J 1952; 47:520-26
7 Meller J, Conde CA, Donoso E, Dack S. Transient Q waves in Prinzmetal’s angina. Am J Cardiol 1975; 35: 691-95
8 Camara DS, Caruana JA. The hemodynamic effects of the sclerosant sodium morrhuate in dogs. Surg Gynecol Obstet 1985; 161:327-31
9 Perakos PG, Cirbus JJ, Camara DS. Persistent bradyarrhythmia after sclerotherapy for esophageal varices. South Med J 1984; 77:531-32