Acute Myocardial Infarction after Upper Gastrointestinal Gastroscopy

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We describe a patient with gastric ulcer confirmed by upper gastrointestinal endoscopy who developed an acute Q wave myocardial infarction involving both the anterior and inferior wall shortly after the procedure. This life-threatening complication of gastroendoscopy was clearly demonstrated through studies of electrocardiogram, cardiac enzymes, echocardiogram and cardiac catherterization. We suggest that preventive measures should be implemented so that endoscopy can be performed under optimal conditions, especially for elderly patients with history of heart disease. [*Chin Med J (Taipei) 2001;64:581-585*]

The safety of endoscopic procedures has been a major issue in recent years. It is generally believed that upper gastrointestinal endoscopy is a safe procedure, with about one complication occurring in every 1000 procedures and the mortality rate estimated to be 0.5 - 3 per 10000 cases.[1]

Over 50% of the complications and 60% of the deaths associated with upper gastrointersinal endoscopy are cardiopulmonary in type.[2] Clinical events including hypoxemia, bradycardia, tachycardia, cardiac arrhythmias, hemodynamic changes and myocardial ischemia may occur during and following endoscopic procedures.[3-6] However, these periprocedural manifestations are usually transient, benign and uncommon, and hence the procedure has long been regarded as safe.

We describe an otherwise healthy male patient with complaint of epigastric pain undergoing upper gastrointestinalendoscopy. Gastric ulcer was found. However, several hours after the procedure, he developed chest pain and electrocardiographic ST eleva-tion. Acute myocardial infarction involving both the anterior and inferior walls was confirmed by coronary arteriograms and on the basis of subsequent serum cardiac enzymes. Percutaneous transluminal coronary angioplasty was performed.

Case Report

An 81-year-old man was admitted to the hospital because of epigastric pain of a few days' duration.

The patient was not obese. He denied history of hypertension, diabetes mellitus, hyperlipidemia, systemic or heart diseases. He did not smoke or drink alcohol. However, he had had duodenal ulcer, chronic subdural hematoma and gall bladder stone in the past 8 years, now in stable condition.

On physical examination, the patient appeared well. He was conscious and afebrile. The temperature was 37 °C, the pulse was 80/min and respirations were 20/min. Blood pressure was 142/80 mmHg. The head

and neck were normal. The lungs were clear bilaterally. Heart rhythm was regular and a grade 2 to 3/6 systolic murmur was heard over the left sternal border. The abdomen was soft, and tenderness was elicited over the epigastric region with no rebounding pain. The liver and spleen were not felt, and no masses were detected. The ranges of motion of the extremities were normal. The peripheral pulses were intact. There was no peripheral edema. Neurological examination was unrevealing. The complete blood count and blood chemistry profile were normal. The urine was normal. Radiographs of the chest showed cardiomegaly. The electrocardiogram showed sinus rhythm with ST depression over II, aVF and V4 to 6, revealing myocardial ischemia.

The patient was thought to have peptic ulcer and after premedication with buscopan and xylocaine 10% topical spray, an upper gastrointestinal endoscopy was performed which revealed a gastric ulcer. The course of the procedure was smooth and uneventful. However, the patient felt chest discomfort soon after the endoscopy, which became severe and persistent approximately three hours later, associated with cold sweating. The electrocardiogram revealed signs of an evolving Q wave myocardial infarction involving both the anterior and inferior walls (Fig. 1). An

Fig. 1. Electrocardiograms performed on admission (**A**) and after endoscopy (**B**) showing acute anterior and inferior myocardial infarction.

echocardiogram demonstrated both anterior and inferior wall hypokinesis with impaired ventricular systolic function. Creatine kinase and CKMB were 127 and 9 IU/liter on admission, respectively. After the onset of acute myocardial infarction, however, the creatine kinase and CKMB were 518 and 37 IU/liter; at 6 hours, 266 and 16 IU/liter; at 12 hours, 181 and 11 IU/liter; at 18 hours, 101 and 6 IU/liter, and at 36 hours, 80 and 6 IU/liter, respectively. LDH isoenzyme was 712 U/liter at 48 hours. Acute myocardial infarction was confirmed. Because of persistent anginal pain, the patient underwent coronary angiography, and percutaneous angioplasty of a 95% left anterior descending artery stenosis and a 99% right coronary artery stenosis were performed (Fig. 2). He got well.

Discussion

Our patient suffered a Q-wave anterior and inferior wall myocardial infarction after upper gastrointestinal endoscopy. It has been shown that upper gastrointestinal endoscopy can induce hypoxemia, bradycardia, tachycardia, autonomic nervous abnormality, cardiac arrhythmias and myocardial ischemia, which may contribute to the occurrence of cardiac events during or after the endoscopic procedures.[3-6] The pathogenesis of periprocedural ischemia is likely to be multifactorial and may include interruption of intrinsic circadian activity, anxiety about endoscopy, hypoxia, and use of sedative medications. Anxiety may trigger catecholamine release, and increased vagal activity as a result of gastrointestinal manipulation may induce vasospasm, reduce coronary blood flow or increase cardiac work. Although our patient denied previous systemic nor coronary artery disease, the baseline electrocardiogram and the clinical picture suggest that he might have had preexisting coronary artery disease presenting with epigastric pain. Indeed, a cardiologist consultation before the endoscopy might have been useful to reduce the procedure-related acute cardiovascular complications. Anyhow, the patient's acute myocardial infarction was probably provoked by the stress of endoscopy, which might have influenced the dynamic interaction between atheroscle ro-
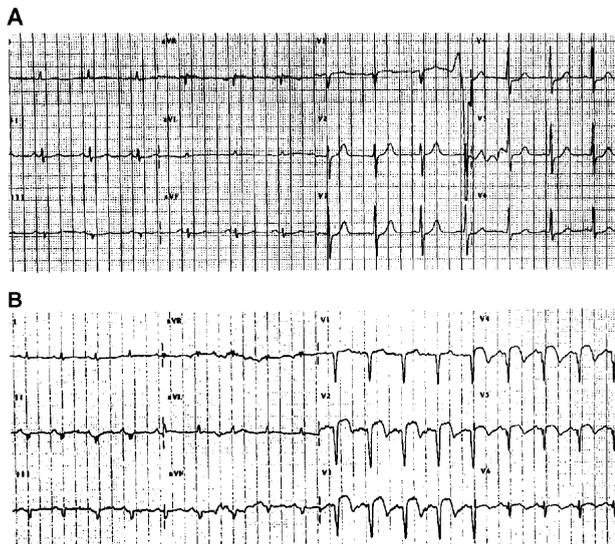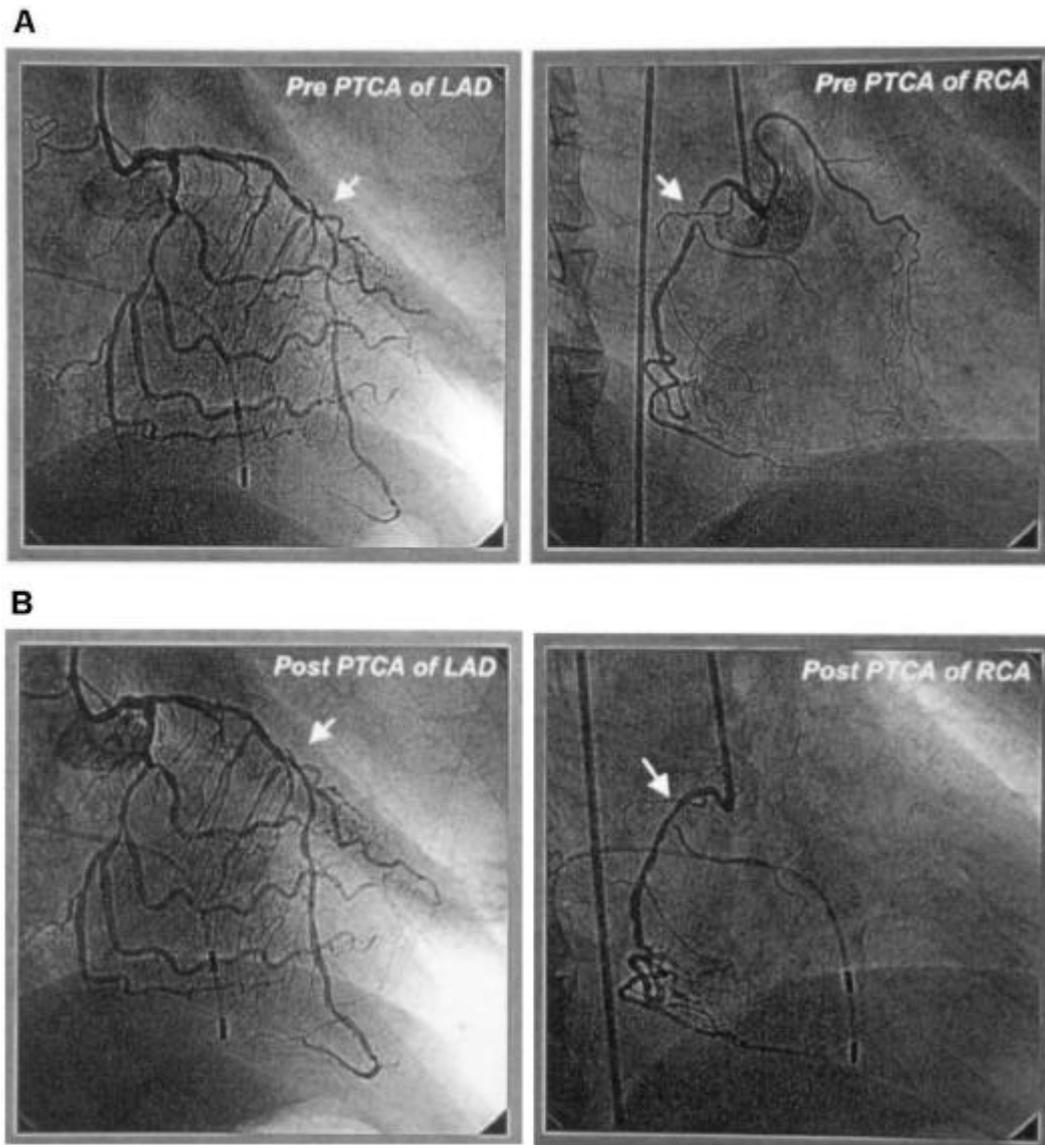
Fig. 2. Coronary arteriograms before (**A**) and after (**B**) percutaneous transluminal coronary angioplasty of the left anterior descending and right coronary arteries after the patient developed acute myocardial infarction following endoscopy.

sis, platelet aggregation and coronary spasm superimposed on the atherosclerotic stenoses, resulting in the evolution of myocardial infarction.

At catheterization, the coronary arteriogram showed atherosclerotic changes of all coronaries, with discrete stenoses in the infarct-related left anterior descending and right coronary arteries. The acute myocardial infarction was subsequently resolved by percutaneous transluminal coronary angioplasty.

Complications are a part of the practice of medicine. This is amply true for gastrointestinal endos-

copy. It has been reported that in clinical practice, endoscopy does not appear to induce overt myocardial ischemia in stable patients with recent myocardial infarction.[7] It has also been declared that since cardiac complications of endoscopy are uncommon, peri-procedural ischemia may not be clinically relevant.[4,7-9] However, our case clearly indicates the contrary. Acute myocardial infarction was clearly demonstrated in our patient through studies of electrocardiogram, cardiac enzymes, echocardiogram and cardiac cathe-terization. We would therefore suggest instead that

there is a need for fur ther stud ies on the patho genic mechanism in myocardial ischemia during en dos copy. The overall patient status must be correctly eval u ated before the en do scopic ex am i na tion. Pre ven tive ap proaches such as mon i tor ing of blood pres sure, elec tro car dio gram and pulse oximetry should also be im ple mented so that the pro ce dure can be per formed un der op ti mal con di tions and re sults in max i mal pa tient safety, es pe cially for el derly pa tients with his tory of heart dis ease.

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