Two Cases of Stress Cardiomyopathy During Esophagogastroduodenoscopy

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Abstract

Esophagogastroduodenoscopy (EGD) is considered as a relatively safe procedure. But EGD course and materials used in conscious sedation EGD, which can act as a stress on patient. Really adverse events were reported during EGD and representative adverse events are cardiopulmonary complications. Up to now, globally five cases were reported associated with gastrointestinal scopy. SCMP is a reversible cardiomyopathy that typically occurs in postmenopausal women by stress and may be recovered within a few weeks. SCMP resemble acute myocardial infarction but differ in terms of treatment and prognosis. Here, we describe 2 cases of SCMP with shock during EGD in conscious sedation.
Introduction

In general, esophagogastroduodenoscopy (EGD) is considered as a relatively safe procedure.\(^1\) However, during EGD, there may be some adverse events, such as heart failure and acute myocardial infarction (AMI).\(^1\) As far as we know, just five stress cardiomyopathy (SCMP) cases have been reported associated with gastrointestinal endoscopy.\(^2\) SCMP is a reversible cardiomyopathy that typically occurs in postmenopausal elderly women by stress and may be recovered within a few weeks.\(^3\) Here, we describe 2 cases of SCMP with shock during EGD in conscious sedation.

Case presentation

Case 1.

A 44-year-old woman with no past history underwent EGD for screening late July 2014. She had previously undergone two EGDs without any complication. The vital signs before the EGD were normal (blood pressure (BP) 110/70 mmHg, pulse rate (PR) 84/min). Electrocardiography (ECG) patterns and saturation of peripheral Oxygen (SpO2) were normal. No signs of pain or discomfort were observed before insertion of EGD scope. For the EGD, lidocaine hydrochloride (20mg of spray) was administered to the throat, and as a sedative midazolam 5mg of intravenous (IV) was used. After EGD scope was inserted through pyriform sinus into esophagus, however, she suddenly complained of chest discomfort and dyspnea. BP and PR rose up to 180/160 mmHg, and 164/min, respectively. For wakening up the sedation state, reversal agents of sedatives; flumazenil 0.25mg was immediately administered of IV and EGD was stopped. She was moved to the emergency room (ER) for the evaluation about chest discomfort and dyspnea. The patient’s 12-lead ECG showed ST elevation in the I and aVL leads, and II, III, aVF, V4~6 ST depression at ER (Fig. 1a). After 20 minutes, the BP was 62/24 mmHg, the PR 67/min, the SpO2 99% under nasal O2 2liter (L), and the respiration rate (RR) 20/min. Since BP was below 90/60 mmHg, normal saline 2L was administered of IV and the vasoactive agent; norepinephrine; was started. Initial level of cardiac enzyme was normal. After several hours cardiac enzymes level were rose up; creatine phosphokinase MB (CK-MB) 19.1 ng/ml
(normal reference range, 0.6~6.3 ng/ml) and troponin I (Tn-I) 5.39 ng/ml (normal reference range, 0~0.5 ng/ml). Transthoracic echocardiogram (TTE) showed only hypokinetic mid left ventricle with ejection fraction (EF) 58% (Fig. 2a, b).

The first impression was estimated ST elevation MI. Cardiac catheterization was undergone but the result was normal. Also left ventriculogram shows hypokinetic mid left ventricle (Fig. 3a, b)

In conclusion, she was diagnosed as SCMP and then underwent conservative treatment. After 2 days from EGD, cardiac enzyme levels returned to normal. After 3 days from EGD, SpO2 was above 95% with room air and ECG returned to normal. (Fig 1b). Chest discomfort and dyspnea were resolved. In the next day low BP recovered without vasoactive agent. She was fully recovered without other special event, and then was discharged.

Case 2.

A 45-year-old-woman underwent EGD for follow up of gastric GIST late April 2014. Last year she underwent conscious sedation EGD and endoscopic ultrasonography (EUS) without any complication. She has no past history except for gastric GIST. The vital signs, ECG pattern, and SpO2 before EGD were normal. For the EGD, lidocaine hydrochloride (20 mg of spray) was administered to the throat, and as a sedatives midazolam 5mg of IV was used. After 1 minute from EGD insertion, she suddenly complained of chest discomfort. BP and SpO2 fell to 75/50mmHg, 80% and HR was rose up 120/min. Reversal agents of sedatives; flumazenil 0.25mg was administered of IV and EGD was stopped. After withdrawal of the EGD scope, she had spit out fresh blood with cough. After supplying O2 10 L by reservoir mask, SpO2 was 90~91%. Normal saline 500 ml was dropped by IV, and then systolic BP was 90 mmHg. She was moved to the emergency room for the evaluation about chest discomfort, dyspnea, hemoptysis and shock. The patient’s ECG showed normal, but cardiac enzyme levels were rose up; CK-MB 11.3ng/ml, Tn-I 3.79ng/ml. Chest computed tomography (CT) showed multiple ground glass opacity pattern, interlobular thickening and impression was pulmonary edema. TTE showed only hypokinetic mid left ventricle, with ejection flow 45%. To rule out MI, coronary artery
CT was undergone and the result was normal, also the evaluation for pheochromocytoma; measuring metanephrine and catecholamine in plasma and through a 24-hour urine collection were performed and the results were in normal range.

In conclusion, she was estimated as SCMP and underwent conservative treatment. After 1 day from EGD, SpO2 was above 95% with room air. After 2 days from EGD, chest discomfort, dyspnea and hemoptysis were resolved. After 3 days from EGD, elevated cardiac enzymes returned to normal. After 6 days from EGD, follow up TTE showed no abnormal contraction with EF 66%. After 7 days from EGD, she was fully recovered without special event and then discharged.

Discussion

EGD is relatively safe procedure which has been widely performed. However, patients may usually suffer from anxiety and feeling of discomfort due to EGD. Conscious sedation is associated with patient tolerance and satisfaction with EGD. Thus EGD is mainly performed under conscious sedation. During practice of EGD, undesirable complication could occur. In a prospective study of 14,149 EGD, prompt cardiopulmonary incidents was 2 per 1000 cases. The 30-day mortality rate was 1 per 2000 cases. In other retrospective study of 21,011 EGD, cardiopulmonary complication was 5.4 per 1000 cases.

AMI is similar in clinical features with SCMP reported in our cases. SCMP is a cardiomyopathy which is reversible left ventricular contraction failure caused by acute stress, without coronary artery disease and mainly occurred in postmenopausal elderly woman. In most cases, SCMP could be improved in a few weeks but rarely could cause pulmonary edema, cardiogenic shock, arrhythmias, heart failure or death. Therefore, we should to distinguish SCMP with MI due to differences in treatment and prognosis. Current diagnostic criteria for SCMP have recently been published (table 1). There are a few hypotheses for SCMP; Emotional stress, certain pharmacologic agent, exogenous catecholamine, unstable condition of autonomic nervous system. Stress such as EGD and sedatives may affect an unstable autonomic nervous condition. Insertion of EGD from pharynx into esophagus
and from lower esophagus to cardia of stomach, which induces tachycardia as a result sympathetic nervous system hyperactivity. Unstable condition of autonomic nervous system caused by EGD may be a critical role of SCMP in our cases. In our case, during EGD insertion from pharynx into esophagus HR rose up to 164/mm and patient suffer from chest pain, dyspnea. Ever globally five SCMP cases were reported associated with gastrointestinal endoscopy. Previous reports had 1 case of EGD occurred in post-menopausal woman with sedation, 3 cases of colonoscopy and 1 case of simultaneously EGD and colonoscopy occurred in post-menopausal woman. Compared to previous cases, our patients were pre-menopausal women without drug allergy, medication history, and cardiopulmonary disease who before underwent several times conscious sedation EGD. SCMP in our cases is a rare cardiomyopathy but could be occurred in medical environments such as EGD. Therefore, clinicians should know that during EGD SCMP could be occurred.

For prevention and dealing with SCMP assessment of cardiopulmonary status during the EGD, as well as review of current drug and drug allergies and physical examination had to be performed. Supplemental oxygen supply reduced magnitude of size oxygen desaturation. Continuous ECG monitoring is appropriate in high-risk patients; significant cardiopulmonary disease, elderly patients, and procedure are expected to be long. Capnography can be used to monitor the patient’s respiratory activity. Also proper selection of sedatives and dosage is needed. Usually used sedatives may be benzodiazepines. Most endoscopists favor midazolam for fast onset, short duration of action, and high amnestic properties. The usual total dose is 2.5 to 5 mg. Less doses may be used in the elderly or other central nervous system depressants use. In conclusion, physicians should be aware that during EGD SCMP could be occurred. For minimization and management about SCMP during conscious sedation EGD, patient may be assessed of the risk of cardiopulmonary status, review of current drug, physical examination before EGD and should be undergone continuous ECG monitoring and pulse oximetry with or without capnography in high-risk. Also appropriate titration dose of sedative should be performed depending on patient’s risk.
References.


Table 1) Proposed Mayo Clinic criteria for the diagnosis of Tako Tsubo cardiomyopathy and long term prognosis

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<th>Mayo-clinic diagnostic criteria</th>
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<td>1. Transient hypokinesis, akinesis, or dyskinesis of the left ventricular mid segments with or without apical involvement; the regional wall motion abnormalities extend beyond a single epicardial vascular distribution; a stressful trigger is often, but not always, present*</td>
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<td>2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture**</td>
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<td>3. New electrocardiographic abnormalities (either ST-segment elevation and/or T-wave inversion) or modest elevation in cardiac troponin</td>
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<td>4. Absence of: Pheochromocytoma, myocarditis</td>
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*There are rare exceptions to these criteria such as those patients in whom the regional wall motion abnormality is limited to a single coronary territory.

**It is possible that a patient with obstructive coronary atherosclerosis may also develop Tako-Tsubo cardiomyopathy. However, this is very rare in our experience as well as in the published literature, perhaps because such cases are misdiagnosed as an acute coronary syndrome.
Fig. 2 Transthoracic echocardiography (TTE)  
TTE shows hypokinetic mid left ventricle in systole (a) diastole (b)
Fig. 3 Left ventriculogram
Left ventriculogram shows hypokinetic mid left ventricle in diastole (a) systole (b)
Fig 1. Electrocardiography (ECG)
ECG shows ST elevation in the I and aVL leads, and ST depression in the II, III, aVF, V4~6 (a), ST elevation and ST depression improvement (b)