Atypical Stress Induced Cardiomyopathy During Endoscopic Sinus Surgery With Septoplasty in an Elderly Male Patient

Hee Sook Lee, MD, Jae Ryung Yi, MD, Yu Na Jung, MD, Gi Ho Jo, MD, Jung Ju Sir, MD, Seung Min Choi, MD

Department of Internal Medicine, Cardiovascular Center, National Medical Center, Seoul, Korea

Stress-induced cardiomyopathy, also known as Takotsubo cardiomyopathy, is caused by emotional or physical stressors and mimics acute myocardial infarction. Stress-induced cardiomyopathy is characterized by acute, reversible left ventricular apical ballooning without significant coronary artery stenosis. New variants of stress-induced cardiomyopathy with localized wall motion abnormalities or an inverted pattern with a hyperdynamic apex have been reported. We present a rare case of a sudden cardiac arrest due to atypical stress-induced cardiomyopathy (nasal packing and the injection of epinephrine) in an elderly male patient during elective endoscopic sinus surgery with septoplasty under local anesthesia. In this case, only the basal and midportions of the left ventricle were affected, whereas the apex was completely spared. The patient rapidly and completely recovered without sequelae.

Key Words: Stress-induced cardiomyopathy, Takotsubo cardiomyopathy, Nasal packing, Epinephrine

INTRODUCTION

The definition of stress-induced cardiomyopathy is acute onset of hypokinesia of the apical portion of the left ventricle (LV) with compensatory hyperkinesia of the basal walls, which is accompanied by reversible, ST-T segment changes or increased cardiac enzymes; but a normal coronary artery is shown on the coronary angiography. Recently, a variant form of stress-induced cardiomyopathy has been reported, which shows an inverted pattern with a hyperdynamic apex. Here, we report a case of a man who had a sudden cardiac arrest due to a variant of stress-induced cardiomyopathy affecting only the basal and midportions of the LV during elective endoscopic sinus surgery with septoplasty under local anesthesia.

CASE REPORT

A 72-year-old man was admitted to the Department of Otorhinolaryngology for elective endoscopic sinus surgery with septoplasty under local anesthesia due to chronic rhinosinusitis. He had no history of cardiac disease or hypertension, drug allergies, previous operations or underlying disease. His initial physical status was assessed as American Society of Anesthesiologists class 1. His initial vital sign was stable. His electrocardiogram, echocardiogram, and pulmonary function test were within normal limit, and no abnormal findings were detected in blood chemistries. For prevention of nasal bleeding, anesthesia and hemostatics were administered by packing the soaked strip with 2% lidocaine plus 0.01% epinephrine and spraying 0.01% epinephrine 2 times in both nasal cavities just before the operation. When the operation started, a nasal mucosal injection of 2% lidocaine plus 0.005% epinephrine for 15 mL was done. He underwent an uncomplicated paranasal sinus surgery under local anesthesia. But sudden cardiac arrest occurred for 3 minutes just after the operation started with Pulseless electrical
activity on electrocardiography. Cardiopulmonary resuscitation was done and after successful resuscitation, the electrocardiography recovered to normal sinus rhythm with normal heart sounds and no murmurs, the blood pressure was 120/60 mmHg, heart rate was 60 to 70/min, and oxygen saturation was 98% on room air. At that time, the white blood cell count was 10,700 cells/mm³ with 61% lymphocytes, the creatine kinase (CK) level was 72 U/L, creatine kinase-myoglobin (CK-MB) was 0.5 ng/mL, N-terminal pro-brain natriuretic peptide (NT pro-BNP) was 16.1 pg/mL, and creatinine was 1.3 mg/dL. Serum urea nitrogen, serum proteins, and electrolytes were within the normal range. He was transferred to the Department of Cardiovascular Medicine for further evaluation. After 7 hours, CK-MB was elevated to 12.1 ng/mL, and Troponin I (TnI) was elevated to 9.54 ng/mL. And after 72 hours, CK-MB and TnI fell to the normal range, but NT pro-BNP was elevated to 894.1 pg/mL. An echocardiographic evaluation depicted a decreased LV ejection fraction (30%), with new regional wall motion abnormalities (i.e., hypokinesis of the basal and mid segments of the LV and hyperdynamic apex) (Figs. 1, 2). He underwent emergent coronary angiography but the coronary angiogram showed normal coronaries (Fig. 3). He had no increased inflammatory markers. No endocrine diseases or other serious concomitant disorders were present. He was treated with hydration and antibiotics for prophylaxis of the operation site infection. We repeated the echocardiogram after 72 hours and it revealed the complete recovery of his LV systolic function and regression of regional wall motion abnormalities. After 5 days, he was discharged without sequelae.

**DISCUSSION**

Typical stress-induced cardiomyopathy, or transient LV apical ballooning, has been described as a cardiac syndrome comprising transient LV dysfunction with chest symptoms and electrocardiography (ECG) changes mimicking those of an acute myocardial infarction. Transient LV apical ballooning is accompanied by minimal biomarker elevations and absence of acute occlusive coronary artery disease. The true prevalence of the apical ballooning syndrome remains uncertain. It occurs most frequently in women over 50 years of age. Although originally reported in Japan, it has been recently described in white Caucasians in Europe and North America. Almost 90% of reported patients are female. Exact mechanism is unknown, but events appear to be temporally related to stressful situations where there are high levels of adrenergic stimulation. It is known that endogenous adrenergic stimulation (e.g., pheochromocytoma) can result in manifestations of this entity. A previous case occurring after administration of epinephrine has been reported.

The following observations support the hypothesis of epinephrine-induced myocardial effects: First, individual differences in the anatomy of cardiac sympathetic innervation or the distributions of adrenoceptors might result in the involvement of a variety of left ventricular myocardial seg-
ments. In typical apical ballooning, high local concentrations of epinephrine might evoke basal hyperkinesis, increasing mechanical wall stress at the apex and thereby increasing end-diastolic pressure and brain natriuretic peptide (BNP) levels\(^9\). Secondly, elevation of circulating plasma epinephrine probably causes the myocardial histological changes because of the epinephrine cardiotoxicity. These changes, which differ from those in ischemic cardiac necrosis, include contraction band necrosis, neutrophil infiltration, and fibrosis. These findings probably reflect consequences of high intracellular concentrations of Ca\(^{2+}\), and it has been proposed that Ca\(^{2+}\) overload in myocardial cells produces the ventricular dysfunction in epinephrine cardiotoxicity\(^9\). Thirdly, the possibility that epinephrine caused hypersensitization in the body cannot be ruled out. Carter et al.\(^ {10} \) measured the discharged epinephrine and the metabolites after an epinephrine injection and reported that the detected amount was 2 times more than the expected value, which can lead to cardiovascular crisis, such as arrhythmia and cardiac arrest. They mentioned the possibility of endogenous epinephrine hypersensitization by the externally injected epinephrine.

As opposed to patients with acute myocardial infarction, those with transient LV apical ballooning syndrome have generally a benign prognosis. Only 1.1% of reported patients died during the hospitalization period and almost all surviving patients recovered fully\(^9\).

In contrast to most cases reported in the literature, we report a rare case of stress-induced cardiomyopathy with focal wall motion abnormalities affecting only the basal and mid LV segments, sparing completely the apex, but comprising all the remaining characteristics of stress-induced cardiomyopathy. Our patient has some features different from the typical presentation of stress-induced cardiomyopathies. First, we postulate that the triggers in this patient were excessive catecholamine stimulation due to emotional and physical stress associated with discomfort due to endoscopic sinus surgery or pain during the operation. Also, systemic absorption of epinephrine from nasal application of epinephrine-soaked gauze and intramuscular injection of epinephrine may be responsible for the manifestations of this syndrome. Secondly, in most reported cases, stress-induced cardiomyopathies presents as an ST-segment elevation myocardial infarction with classical ECG changes. However, the patient’s postcardiopulmonary resuscitation (post-CPR) ECG did not show the ST-segment elevations or T inversions, as is often seen in stress-induced cardiomyopathies.

Our patient experienced sudden cardiac arrest because of the mucosal injection and nasal packing of epinephrine to prevent nasal bleeding or emotional and physical stress during paranasal sinus surgery. But we did not check the patient’s serum epinephrine concentration because the post-CPR condition can also raise epinephrine concentrations. We assume that epinephrine cardiotoxicity and hypersensitivity reaction cause the cardiac arrest, which leads to post-CPR atypical stress-induced cardiomyopathy\(^{15,7} \). Our patient experienced an extremely rapid recovery of the left ventricular systolic dysfunction. This rapid recovery may involve a relatively smaller area of myocardial involvement and a less intense illness\(^{13,12} \).

Endoscopic sinus surgery with septoplasty under local anesthesia is a minimally invasive surgical procedure and usually has a good outcome, with most studies reporting an 80–90% rate of success without complications. But, in elderly male patients, in response to nasal packing and mucosal epinephrine injection that are commonly used to control bleeding in epistaxis and after paranasal sinus surgery, some patients have exhibited cardiac arrest in clinical settings, and atypical stress-induced cardiomyopathy must be included in the differential diagnosis\(^ {10} \).

REFERENCES