CASE REPORT

Epidural Steroid Injection—Procedure-related Stress (Takotsubo) Cardiomyopathy: A Rare Case

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Abstract

Stress cardiomyopathy/Takotsubo cardiomyopathy (TC) is a transient and reversible acute heart failure provoked by physical or emotional stress. This case report highlights about a patient who experienced sudden onset chest pain and breathlessness following epidural steroid injection for pain secondary to prolapsed intervertebral disc. Initial clinical features, electrocardiogram, troponin levels, and transthoracic echocardiography showed features suggestive of acute coronary syndrome but the coronary angiogram was normal. The diagnosis of stress cardiomyopathy was made and managed successfully. This case report highlights one of the rare cases of procedure-related early stress cardiomyopathy. Early diagnosis and management reduce morbidity and mortality.

Keywords: Cardiomyopathy, Epidural steroid, Stress, Takotsubo.

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Introduction

Stress cardiomyopathy represents approximately 1.2% of all troponin-positive coronary syndrome.[1] The TC following epidural steroid injection is very rare.[2] This case report highlights one such case of TC following lumbar epidural steroid injection for prolapsed intervertebral disk.

Case Description

A 45-year-old female patient who is a known case of type-2 diabetes mellitus and hypertension was referred from outside hospital with sudden onset of chest pain and breathlessness following lumbar epidural steroid injection. At the outside hospital, patient underwent epidural steroid injection for pain relief due to lower back ache caused by lumbar prolapsed intervertebral disc. Twenty minutes after procedure she experienced sudden-onset chest pain, sweating, and breathlessness. Immediately, electrocardiogram (ECG) was done which showed ST-T changes in precordial leads. Troponin T levels (>45) and pro-BNP (3570 pg/mL) both were elevated. Transthoracic echocardiography showed dilated cardiomyopathy with global hypokinesia and ejection fraction of 25%. In view of suspected acute coronary syndrome, patient was shifted to our multispecialty tertiary hospital for further management. In emergency room (ER), she was conscious, afebrile with pulse rate 112 beats/minute, blood pressure 88/47 mm Hg, and SpO_2 89% (room air). Systemic examination was normal except revealed bilateral crepitation on auscultation of lungs. Routine blood test showed the following: Hb%—12.8 g/dL, total leukocyte count (TLC)—13,300, platelet count—1.7 lakh cells/cu mm, Sr. sodium—138 mEq/L, potassium—3.9 mEq/L, chloride—103 mEq/L, Sr. creatinine—0.8 mg/dL, blood urea—28 mg/dL, total bilirubin—0.4 mg/dL, SGOT—28 U/L, SGPT—34 U/L, and S. albumin—4 g/L. Arterial blood gas analysis showed the following: pH—7.3, pCO_2—28, pO_2—105, and HCO_3—19.2. Patient underwent coronary angiogram which was normal. The diagnosis of stress cardiomyopathy was made (mayo clinical criteria), and she was shifted to cardiac intensive care unit (CICU) for further management. Initially, patient managed with furosemide infusion (10 mg/hour), noradrenalin infusion (0.05 µg/kg/minute), aspirin, heparin, noninvasive ventilation, and other supportive care. Patient condition improved gradually over 2 days with improvement in cardiac ejection fraction from 25 to 40%. Noradrenalin infusion was gradually tapered and stopped. She was shifted to ward on fourth day and later discharged home in a hemodynamically stable condition with low-dose cardio-selective beta blocker—metoprolol (to block catecholamine surge)—and aspirin on sixth day.

Discussion

Takotsubo cardiomyopathy/stress-induced cardiomyopathy or broken heart syndrome is a transient and reversible left ventricle wall motion abnormality following physical or emotional stress.[3] First described by Japanese cardiologist in 1990.[4] The name Takotsubo is derived from the Japanese word (crab pot or octopus trap) and is used for the echocardiographic appearance of left ventricle (apical ballooning and basal hypokinesia). The TC is caused by either emotional stress (positive or negative emotions) or any other organ dysfunction (secondary TC). Secondary TC is due to neurological disorders (head injury, subarachnoid

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hemorrhage), endocrine disorders, COPD exacerbation, and recent sepsis.

Multiple theories are proposed for the causes of TC. Most common is excessive release of catecholamines by overstimulation of hypothalamic–pituitary adrenal axis leading to myocardial stunning and apical ballooning.[5] Other theories include genetic cause, myocardial bridging, edema, and microvascular dysfunction. Patients usually present with chest pain (most common), dyspnea, palpitation, or syncope with physical or emotional trigger, and on examination, they will have tachycardia, hypotension, and pulmonary edema. In 30% of cases, there will be no trigger. ECG will show ST elevation (43%), ST depression (8%), T-wave inversion (50%), or prolonged QTc interval (>400 ms). NT-pro-BNP will be increased (3–5 times compared to troponins). 2D echo-large area of regional wall motion akinesia of left ventricle extending beyond the territory of single coronary artery.[6] Left ventricle ejection fraction is always compromised. No obstructive pathology is found in angiography. Cardiac MRI demonstrates subendocardial edema at affected segments (myocarditis will have subepicardial edema). Differential diagnosis includes acute coronary syndrome and myocarditis. Coronary angiogram will be performed to rule out acute coronary syndrome. The mayo clinical criteria[7] are used for the diagnosis of TC. This includes regional wall motion abnormalities on 2D echo, ECG changes with increased troponin and pro-BNP, normal coronary angiogram, and absence of pheochromocytoma and myocarditis. Complications include pulmonary edema, life-threatening arrhythmias, LV thrombus, and LV wall rupture.

Management is mainly supportive,[8] and they are managed in ICU; if the left ventricle ejection fraction is >45%, then cardio selective beta blockers (carvedilol, metoprolol), aspirin, and ACE inhibitors are used. If the left ventricle ejection fraction is between 35 and 45%, then beta blockers without ACE inhibitors/ARB are used. In case of cardiogenic shock, the noradrenalin and dopamine are avoided. IABP, LVAD, and ECMO may be considered. Intravenous levosimendan (5 µg/kg bolus over 10 minutes followed by 0.1–0.2 µg/kg/minute over 24 hours) and milrinone may be used.[9] Prognosis is usually good, and over 90% of the patients show complete recovery in 4–8 weeks. Mortality is about 4.5%,[10] and recurrence rate is 5–10% in 5-year follow-up.

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