Intra-operative Tako-tsubo cardiomyopathy during carotid body tumor excision: An indication for therapeutic use of Levosimendan

Dear Editor,

An 80 kg, 52-year-old woman, in American Society of Anesthesiologists class 2, was scheduled for excision of a chemodectoma. Pre-anesthetic examination and relevant investigations were unremarkable, and vital parameters recorded were within normal limits. In the operating room, patient was administered cefuroxime 1.5 g, ranitidine 50 mg, and metoclopramide 10 mg intravenous (IV). General anesthesia was induced with midazolam 1.5 mg, fentanyl 120 mcg, and propofol 160 mg IV. Neuromuscular blockade was achieved with vecuronium 7 mg IV. Anesthesia was maintained as per standard protocol of our institution. One hour into surgery as the tumor was manipulated, the patient developed asystole. Cardiopulmonary resuscitation was initiated immediately according to advanced cardiac life support (ACLS) protocol and after 2 cycles of cardio-pulmonary resuscitation (CPR), the heart was revived. Electrokardiogram (EKG) showed deep T-wave inversion, and central venous pressure was 26 cms of H_2O. Vasopressor and inotropic support (dopamine, dobutamine, and adrenaline) was administered to maintain blood pressure within optimum limits.

At the time of shifting to the intensive care unit (ICU), after 3 hours of surgery, the chest X-ray showed diffuse bilateral basal infiltrates, EKG showed ST-segment elevation (leads V3-V6) [Figure 1], and the cardiac biomarkers troponin-I, CK-MB, and N-terminal prohormone brain natriuretic peptide levels were raised (5.9 pg/ml, 10.4 U/l, and 8879 pg/ml, respectively). The 3D-echokardiography showed normal left ventricular size with localized myocardial thickening and apical akinesia in the mid-basal septum. Regional strain/strain rate imaging showed the presence of reduced systolic deformation with significant post-systolic deformation in the abnormally

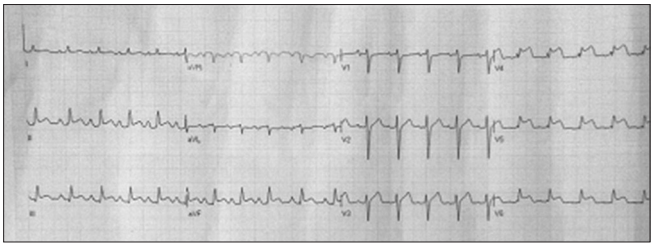

Figure 1: Immediate post-op ECG

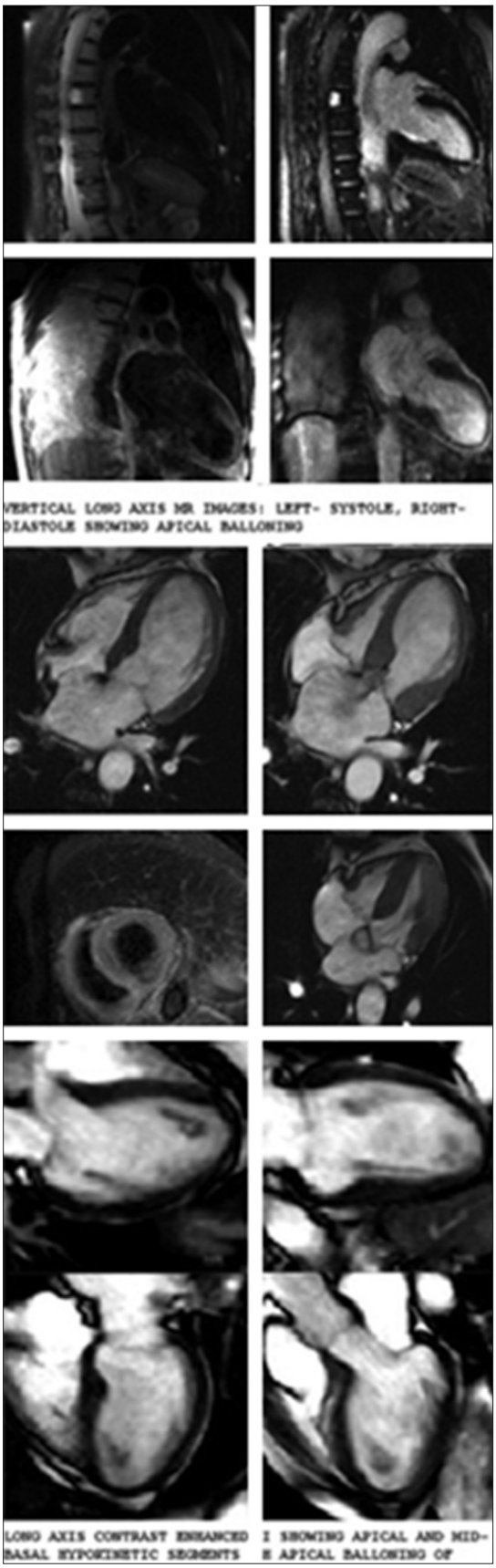

Figure 2: MRI imaging in different planes showing apical ballooning

contracting mid and apical left ventricular (LV) walls, a pattern suggestive of post-ischemic myocardium. Angiography revealed no pathology of coronary vasculature. The LV angiogram showed apical and mid-basal ballooning, and provisional diagnosis of Tako-Tsubo cardiomyopathy was made. A T2-weighted magnetic resonance imaging (MRI) showed significantly higher T2-signal intensity in the dysfunctional segments, potentially indicating the presence of myocardial edema in the affected areas that showed ballooning [Figure 2]. Viral titers were inconclusive. Intra-aortic balloon pump (IABP) support was initiated, 0.1 mcg/kg/min levosimendan infusion started, and all other vasopressors discontinued. Loop diuretics and morphine were administered for pulmonary edema.

Patient was weaned off chemical and mechanical support within 48 hrs. Beta-blockers and angiotensin-converting enzyme inhibitors were started for heart failure. The patient showed good response to treatment with complete recovery and was discharged on carvedilol, aspirin, clopidogrel, furosemide, and digoxin after 15 days.

Echokardiography, done 45 days post-discharge, showed complete normalization of the apical wall motion abnormalities and an absence of post-systolic deformation in the mid-apical and basal walls, confirming the diagnosis of intra-operative Tako-Tsubo cardiogenic shock. Cardiac MRI with gadolinium late-enhancement showed no evidence of myocardial scar. The dobutamine stress echocardiography response of apical myocardial segments was typical for stunned myocardium. At 6 months follow-up, systolic function deformation was normal in all LV segments.

Tako-tsubo syndrome or broken heart syndrome is characterized by the finding of transient LV dyssynergy, leading to apical ballooning with concomitant compensatory basal hyperkinesis. The classic presentation is usually triggered by severe emotional or physical stress and mimics acute coronary syndrome.[1] This condition probably accounts for 1% to 2% of all cases of suspected acute myocardial infarction.[2] The distinguishing features of stress cardiomyopathy according to Mayo Clinic criteria[3] include: LV dyskinesis not represented by a single epicardial artery vascular territory; absence of obstructive coronary artery disease; EKG changes such as ST elevation and/or T-wave inversion; and the absence of head trauma, intracranial hemorrhage, pheochromocytoma, myocarditis, and hypertrophic cardiomyopathy. Although there is a 2% mortality in the initial phases of this syndrome, most patients experience full recovery with rare reports of recurrence.[2] Both echocardiography and MRI-derived parameters may be indispensible in the diagnosis and follow-up of these patients as they may show spontaneous recovery of the cardiac abnormalities.[4] It is hypothesized that the reduced estrogen levels after menopause explain the predisposition of elderly women to Tako-tsubo cardiomyopathy.[5] Of the alternative causes of stress cardiomyopathy, catecholamine surge appears to be a common underlying mechanism. The LV apex contains a higher concentration of adrenoceptors, and myocardial responsiveness to adrenergic stimulation is pronounced in the apex, which explains the characteristic LV ballooning.[6] This creates a therapeutic dilemma because inotropic support using exogenous catecholamines may be counter-productive and a non-catecholamine inotrope, levosimendan may be drug of choice to tide over the phase of acute crisis. Padayachee[7] described the two cases where levosimendan was used successfully in Tako-Tsubo-related cardiogenic shock. To our knowledge, there are no reported cases of intra-operative cardiogenic shock due to Tako-Tsubo syndrome during carotid body tumor excision, in which levosimendan and IABP are employed for acute hemodynamic instability.

In conclusion, we postulate that the pathogenesis of Tako-Tsubo cardiomyopathy is multifactorial and constitutes a novel form of heart failure, which should be considered in differential diagnosis of cardiogenic shock or sudden cardiac death in individuals without obvious heart disease. High index of vigilance should be maintained during anesthesia for neuroendocrine tumors as this cardiomyopathy is precipitated by sudden, unexpected surge and abnormal catecholamine dynamics. We believe that administration of cardiotonic agents may be detrimental because of the obvious underlying mechanisms. Use of calcium sensitizer, levosimendan, along with IABP, is logical to maintain hemodynamic stability in acute phase.

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