Electrocardiogram (ECG) is an important tool to diagnose and manage different cardiac conditions. At times, ECG changes are reflective of intracranial pathology causing myocardial stress. In such instances, relying and treating cardiac condition alone can be misleading. Intracranial pathologies such as hemorrhage, aneurysm, stroke, injury, or a space-occupying lesion, have been reported to cause ECG changes mimicking acute coronary syndrome.1-3 These changes are poorly understood and thought to occur due to altered sympathetic tone and associated catecholamine surge.2 Though there are reports mentioning the appearance of ECG changes during acute neurological stress, this is an interesting case wherein an American Society of Anesthesiologists (ASA) grade 1 patient with mass effect came for elective surgery developed ECG abnormalities despite her normal physical status and no suggestive history. The common reported ECG findings mimicked acute coronary syndrome with ST elevation and T inversion with signs and symptoms of neurogenic stunned myocardium such as chest pain, shortness of breath, and hemodynamic instability.

A 42-year-old female patient, ASA1 with right sphenoid wing meningioma was admitted for elective craniotomy and tumor excision. There was no history of past illness, orthopnea or dyspnea on exertion, or any other chronic illness. She was on oral phenytoin 300 mg from past 1 week. Other systemic and physical examination was unremarkable. Serum electrolytes and hematological and biochemical investigations were within normal limits. MRI showed dura base enhancing lesion with significant mass effect (►Fig. 1). Preoperative ECG showed ST depressions with T wave inversions in lead II, V1-V6 (►Fig. 2A). In view of significant ECG changes, preoperative echocardiography was done which did not reveal any significant pathology. Troponin T (Trop T) was negative.

Since the tumor was big, patient was planned for an elective surgery and induction was done with intravenous fentanyl 150 µg, propofol 120 mg, rocuronium 50 mg, and a 7.5-mm cuffed polyvinyl chloride tube was used for tracheal intubation. Surgery was done in supine position with vigilant intraoperative vital monitoring including 5-lead ECG, heart rate, pulse oximetry, arterial blood pressure, central venous pressure, urine output, and temperature. Significant ST depression persisted in chest leads before induction and continued to be present throughout the intraoperative period. Total intravenous anesthesia was used in view of large tumor with mass effect. Bispectral index-titrated propofol, fentanyl, and rocuronium infusion were used to maintain adequate depth of anesthesia and muscle relaxation respectively. Surgery was eventful and duration was around 5 hours with a blood loss of around 2 L which was replaced with five units packed red blood cells, five units fresh frozen plasma, and five units platelets concentrate. No inotropes or vasopressors were required in the intraoperative period and patient was hemodynamically stable throughout the surgery except for the ECG changes. Postoperative elective ventilation was planned in view of massive blood loss, prolonged surgery,
and cerebral edema. Patient was weaned and extubated on first postoperative day (POD). Patient was followed up for 7 days. Serial 12-lead ECG was recorded on POD 1–7, which revealed gradual normalization of ST-T changes and T wave inversions. — Fig. 2B shows the ECG on the 7th day, which depicted complete resolution of abnormal ECG changes. The commonly reported findings are ST elevation with T inversion; however, in our case, we found ST depression and T inversion. The ECG findings were not associated with any signs and symptoms but as a part of screening, Trop T and 2D echo were done. Trop T was negative and echocardiography was normal. As the patient was stable hemodynamically and had no complains of chest pain and stable condition postoperatively, she was not subjected to serial Trop T, serial 2D echo, or invasive cardiac angiogram. Invasive cardiac angiogram should be reserved for cases of abnormal echocardiography showing regional motion abnormalities. Currently, there are no guidelines particularly for neurosurgical patients; however, some studies have impressed upon the use of ECG, serum biomarkers, and echocardiography for preoperative evaluations.  

The American College of Cardiology (ACC)/American Heart Association (AHA) task force has reserved invasive tests such as cardiac angiogram for grossly abnormal echocardiogram like regional wall motion abnormalities or abnormal septal motions which were absent in our case. In absence of abnormalities in echocardiography, no further testing is recommended.  

From this report we want to emphasize that neurological insult has a bearing on ECG which can have manifestations or may remain subtle. In our case, the ECG changes started resolving postoperatively and complete normalization was seen by the end of first week. ECG changes in neurosurgical patient can be due to organic heart disease or secondary to neurological insult. The later tend to resolve with treatment of neurological insult. Cardiac status of the patient should be evaluated from relevant history, clinical examination, biomarkers, and echocardiography. Early treatment of neurological insult should help in resolution of ECG changes. Documentation of persistence/resolution of such ECG changes may be helpful while evaluating the patient in future.

**Conflict of Interest**

None declared.

**Reference**