AUCTORES
Globalize your Research

Journal of Clinical Cardiology and Cardiovascular Interventions

Belal Sultanzai *

Open Access

Case Report

ST-Segment Elevation in a Young Woman with a Brain Bleed

Belal Sultanzai *, Edward Chau, Ezra A. Amsterdam, Sandhya Venugopal

From the Division of Cardiovascular Medicine, Department of Internal Medicine, University of California (Davis) Medical Center, Sacramento, CA.

***Corresponding Author:** Belal Sultanzai MD, University of California (Davis) Medical Center, Division of Cardiovascular Medicine, 4150 V Street, Suite 2400, Sacramento, CA 95817.

Received Date: April 24, 2023; Accepted Date: May 19, 2023; Published Date: May 26, 2023

Citation: Belal Sultanzai, Edward Chau, Ezra A. Amsterdam, Sandhya Venugopal (2023), ST-Segment Elevation in a Young Woman with a Brain Bleed, *J. Clinical Cardiology and Cardiovascular Interventions*, 6(5); **DOI:**10.31579/2641-0419/317

Abstract

A 23 yo woman with a history of traumatic brain injury was re-admitted to the hospital two months after initial hospitalization for a traumatic head injury. Readmission was prompted by follow-up computed tomography head scan that revealed hydrocephalus and a shrunken brain for which she underwent cranioplasty and a ventriculoperitoneal shunt. On post-operative day one, she developed "tombstone" ST-segment elevations (STE) which cleared spontaneously without therapeutic intervention. Echocardiography was normal and troponin was minimally elevated. Head CT revealed an epidural hematoma with midline shift, to which the striking ECG changes were attributed. The hematoma was evacuated and the patient was discharged to her care facility. She was readmitted several times during the ensuing eight-months for recurrent hydrocephalus with external ventricular drain placement. During these episodes, there was no evidence of recurrent STE. Although multiple ECG alterations have been described in association with cerebral injury, tombstone STE is rare in this setting.

Keywords: traumatic brain injury; epidural hematoma; tombstone ST-segment elevations; electrocardiogram; case report

Introduction

Cerebrovascular events, such as intracerebral hemorrhage, subarachnoid hemorrhage, and intracranial hypertension, can cause cardiac dysfunction and associated electrocardiographic abnormalities.[1] The most frequent electrocardiogram (ECG) alterations reported are nonspecific ST-T changes, inverted or broad T waves, QT interval prolongation, and ST segment depression; ST elevation is unusual in these circumstances. [1] Moreover, tombstone STE has rarely been described in relation to cerebrovascular events.

Case Presentation:

A 23-year-old female sustained a severe traumatic brain injury with intraventricular hemorrhage from a motor vehicle accident seven months before this presentation. During the prior hospitalization, she underwent a decompressive hemicraniectomy for intractable intracranial pressure and was discharged to a nursing facility after eight weeks with ventilator and gastrostomy tube dependence. Two months later, follow-up computed tomography head scan (CTH) demonstrated interval development of hydrocephalus and a shrunken brain. She was readmitted and underwent a left-sided cranioplasty with ventriculoperitoneal shunt placement.

On the first post-operative day, the patient developed sinus tachycardia of 150 beats/min. An ECG showed profound ST-segment elevations (commonly referred to as "tombstone" STE) in leads V2-V4 and lesser STE in leads I and aVL with reciprocal ST-segment depressions in the inferior leads (Figure 1).

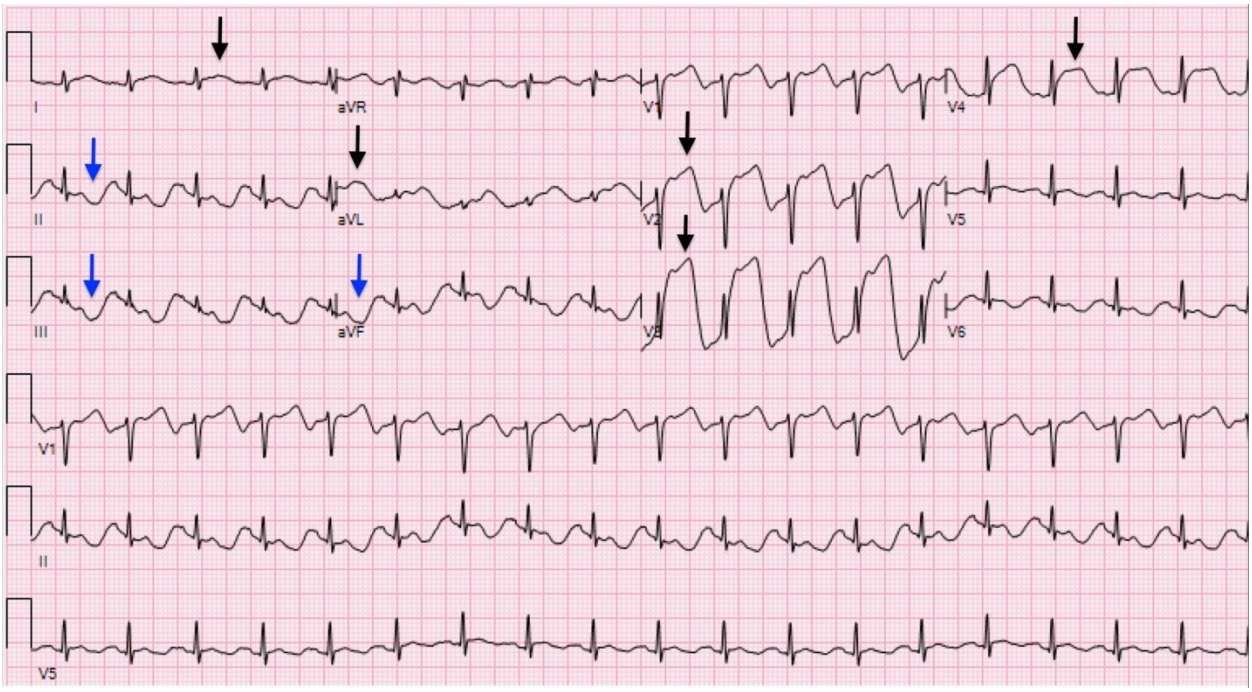

Figure 1: *"Tombstone" ST-segment elevations (STE) in leads V2-V4 and lesser STE in leads I and aVL (black arrows) with reciprocal ST-segment depressions in the inferior leads (blue arrows).*

Assessment:

On examination, the patient was afebrile with blood pressure of 105/55 mmHg, pulse now 120 beats/min, respiratory rate 14/min with arterial oxygen saturation 99% through her tracheostomy on minimal ventilator settings. She was at her baseline nonverbal status, did not follow commands, but opened her eyes spontaneously and tracked some movement. Her left pupil was 5 mm and nonreactive, while her right pupil was 3 mm and briskly responsive. She withdrew to pain in her upper extremities but not her lower extremities. The latter neurological findings were present prior to admission, prior to STE and post-STE. Cardiac examination revealed no jugular venous distension, a non-displaced point of maximal impulse, regular tachycardia, and absence of murmurs or gallops. Chest examination revealed ventilator sounds. Serum high sensitivity troponin T was 28 ng/L (initial) and 32 ng/L four hours later (reference <19 ng/L). A bedside transthoracic echocardiogram revealed normal left and right ventricular function without segmental wall motion abnormalities and no valvular dysfunction. A few hours later, she appeared more encephalopathic, with her eyes closed at rest and no longer opening spontaneously. Her right pupil reacted sluggishly. Repeat ECG at this time showed resolution of the tombstone STE in the precordial leads without any intervention but persistent tachycardia (Figure 2).

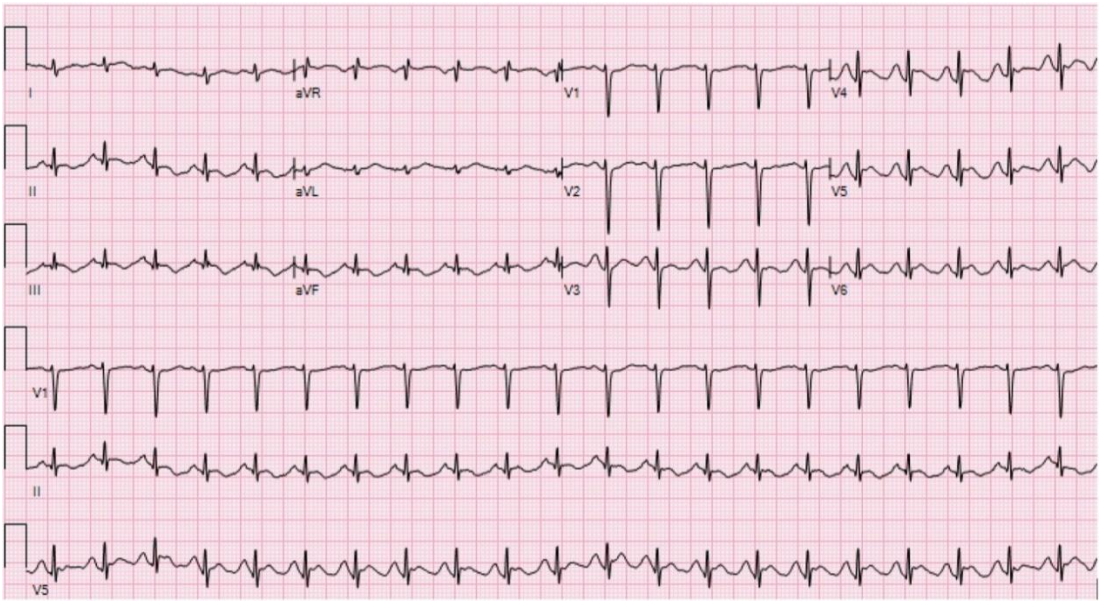

Figure 2: *Resolution of the ST elevations in the precordial leads three hours after the ECG in figure 1 was obtained without any intervention.*

She appeared more uncomfortable and had a two-minute episode of right arm extension and turning towards her right side. An urgent CTH revealed the likely etiology of the STE: an acute epidural hematoma with midline shift (Figure 3).

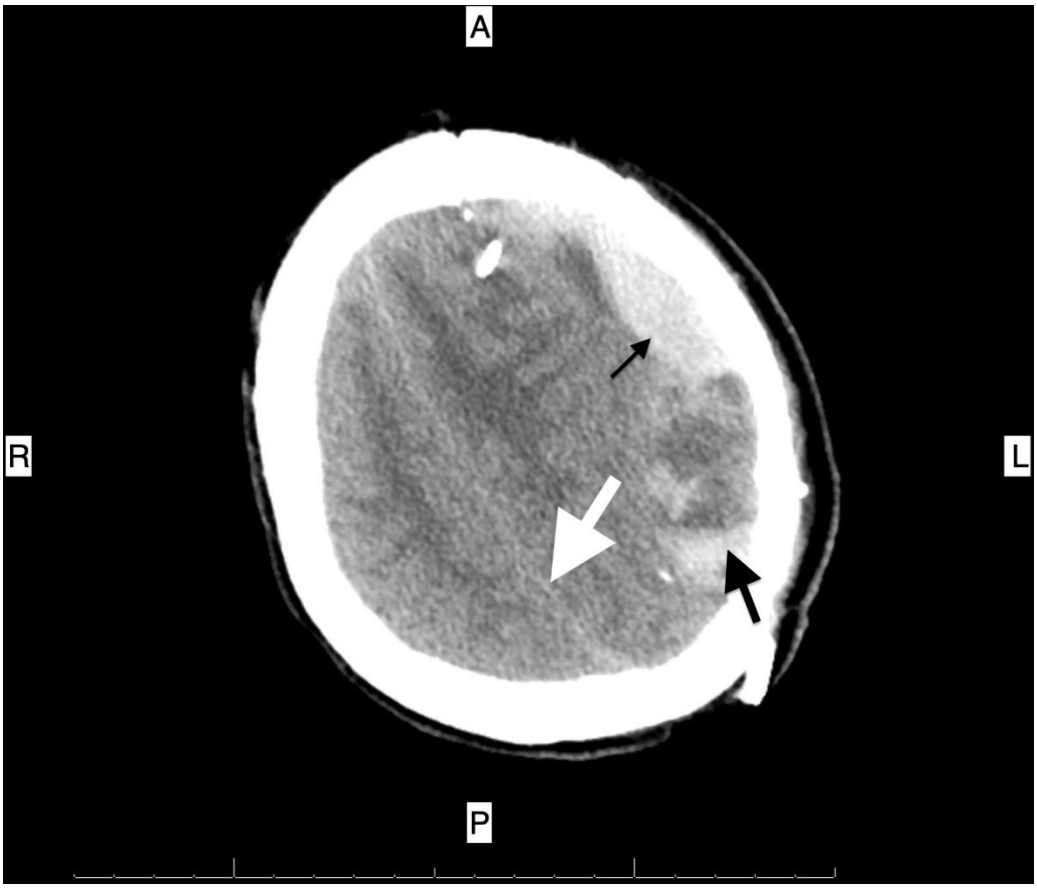

Figure 3: *Computed tomography of the head showing a large acute epidural hematoma (thick black arrow) with a separate subdural component along the left convexity adjacent to the cranioplasty site (thin black arrow), resulting in a rightward midline shift (thick white arrow). P = posterior, A = anterior, R = right, L = left*

She underwent immediate hematoma evacuation after which she exhibited resolution of her neurologic findings, including return of spontaneous eye opening and tracking without any further episodes of right arm extension or favoring of her right side.

Discussion:

The patient developed transient but pronounced ST elevation, which was attributed to elevated intracranial pressure (ICP) from the epidural hematoma and coincided with neurological status changes. Although elevations in ICP or other brain injuries may be reflected by ECG evidence of giant T wave inversions, ST elevation, ST depression, or QT prolongation, tombstone STE has rarely been described in this setting. [1,2] Regional tombstone STE in acute myocardial infarction is associated with increased adverse outcomes; however, our patient's troponin blood levels were only borderline elevated and her echocardiogram the following day remained normal. She had not been taken to the cardiac catheterization laboratory because her recent neurological procedure precluded the obligatory anticoagulation associated with coronary reperfusion procedures, should they be indicated.

Other causes of STE in a young woman, including spontaneous coronary artery dissection, coronary vasospasm, cocaine use, and stress cardiomyopathy, should be considered [3-5]. These diagnoses were unlikely because of the evidence of epidural hematoma, only minimal rise in troponin, absence of segmental left ventricular wall motion abnormalities on echocardiogram, and most importantly, the finding of cerebral hematoma.

Cardiovascular complications are common after intracerebral catastrophes. A phenomenon referred to as neurogenic stunned myocardium has been associated with ECG alterations such as STE, which may help explain our patient's striking ECG alterations in the setting of her neurological process [6]. Neurologic insults can provoke a sharp increase in plasma catecholamines with subsequent ECG and functional cardiac changes, resulting from myofibrillar degeneration, most notably in subendocardial regions of the heart, left ventricular dysfunction, or arrhythmias. However, our patient had no evidence of these latter findings [6].

Although it is recognized that elevated intracranial pressure can cause a variety of ECG changes, our case highlights the possibility that the STE was related to the cerebral insult detected by CTH, as ECG changes after neurological injury are often transient because repolarization normalizes after neurological insult resolves [6]. STE in a young woman requires consideration of non-atherosclerotic causes, including coronary vasospasm, spontaneous coronary artery dissection, cocaine use, and Takotsubo cardiomyopathy [5]. Evaluation comprises a thorough clinical history, physical examination, serial ECGs, measurement of troponin, and echocardiography to enhance diagnosis and risk stratification. Management of these ECG findings is treatment of the underlying intracranial process and follow serial ECGs for resolution of the ECG alterations.

There are several limitations to our analysis. It is a case report which represents observations from a single patient, precluding generalizability.[7] Another limitation is that the relation between the central nervous system lesions and the dramatic ST elevations is conterminous with no proof of cause and effect. Finally, the patient was noncommunicative, precluding

communication of symptoms such as chest pain which may have suggested coronary spasm as the etiology of the ST elevation.

Conclusions

Our patient's serial ECGs revealed rapid resolution of the marked ECG changes. Serial head CT in the outpatient setting revealed expanding hematoma with no acute change in the patient's clinical status since she was discharged following her initial hospitalization after the motor vehicle accident six months previously. After hematoma evacuation and ventriculoperitoneal shunt revision, the patient was discharged to her nursing facility on postoperative day four, at her previous baseline status. She was readmitted several times during the eight-month interval following discharge due to pneumonia and recurrent hydrocephalus with external ventricular drain placement. of note, throughout these hospitalizations, there was no evidence of recurrent STE.

Funding: None.

Declarations of Interest: None.

Authorship: All authors had access to the data and a role in writing the manuscript.

Publication history: Manuscript is original research that has not been published and is not under consideration elsewhere.

Type of Manuscript: Clinical Vignettes

References:

1. Yogendranathan N, Herath HM, Pahalagamage SP, Kulatunga A. Electrocardiographic changes mimicking acute coronary syndrome in a large intracranial tumor: A diagnostic dilemma. BMC Cardiovasc Disord. 2017;17:91.
2. Takeuchi S, Nagatani K, Otani N, Wada K, Mori K. Electrocardiograph abnormalities in intracerebral hemorrhage. J Clin Neurosci. 2015;12:1959-1962.
3. Chandrasekhar J, Gill A, Mehran R. Acute myocardial infarction in young women: current perspectives. Int J Women's Health. 2018;10:267-284.
4. Stern S, Bayes de Luna A. Coronary artery spasm: a 2009 update. Circulation. 2009;119:2531- 2534.
5. Wang K, Asinger RW, Marriott HJ. ST-segment elevation in conditions other than acute myocardial infarction. N Engl J Med. 2003;349:2128-2135.
6. Gregory T. Cardiovascular complications of brain injury, Continuing Education in Anaesthesia Critical Care & Pain. 2012;12:67-71.
7. Nissen T, Wynn R. The clinical case report: a review of its merits and limitations. BMC Res Notes. 2014;7:264.

DOI:10.31579/2641-0419/317