CASE REPORT

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Severe Traumatic Brain Injury Presenting with Wide Complex Tachycardia: a Case Report

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ABSTRACT

Background: Ventricular tachycardia (VT) is an abnormal heart rhythm that can lead to pump failure and hypoperfusion. Its causes, presentation, and treatment are well established in the literature. However, the VT treatment algorithm is based on non-traumatic patients. Due to different pathophysiology and presentation, treating VT in trauma patients should be different. **Objective**: The main purpose is to emphasize the approach to treating VT in severe head trauma patients. **Case presentation:** This case is a unique presentation of severe head trauma with a paucity of treatment approaches in the literature. In this article, we present a case of a middle-aged male patient presented to a level one trauma center with a history of falls from 2 stories height with a Glasgow Coma Scale (GCS) of 3/15. ATLS approach was followed in treating this patient, his rhythm strip showed a wide complex regular rhythm, likely representing a VT with a pulse. The patient was treated as unstable because of a decreased level of consciousness. A 100 J synchronized cardioversion was given without restoration of normal sinus rhythm, followed by Mannitol 1g/kg, treating the possibility of high intracranial pressure (ICP), after which his rhythm was restored to sinus. **Conclusion**: The restoration of sinus rhythm after treating the possibility of high ICP suggests that the cause of VT in this severe TBI patient was the high ICP.

Keywords: Ventricular Tachycardia, Head Trauma, Arrhythmia, Wide Complex.

1. BACKGROUND

Ventricular tachycardia (VT) is diagnosed when a regular and wide QRS complex (120 milliseconds or more) is present. This abnormal rhythm is originating from one of the ventricles at a heart rate greater than 120 beats per minute. VT etiology could be attributed to many causes, most commonly due to underlying ischemic heart disease. Other causes include structural heart disease, channelopathies, infiltrative cardiomyopathy, structural heart disease, electrolyte imbalances such as hypokalemia, hypomagnesemia, hypocalcemia, cocaine, and other illicit drug use, it may also be idiopathic VT (1, 2). Hypokalemia preceded by hypomagnesemia is a common trigger for VT, especially for patients with structural heart disease (3).

Approaching VT depends on the stability of the patient. VT can be divided into two main types, which are stable and unstable VT based on the perfusion status of the vital organ, with pulseless VT as an extreme form of unstable VT (2). Altered mental status in the sitting of VT is considered a sign of instability due to the hypoperfusion status of the brain tissues secondary to the serious arrhythmia. However, the cause of altered mental status in a trauma setting is multifactorial and cannot be attributed to the abnormal rhythm alone. Hence, using the level of consciousness alone as a sign of instability may lead to delivering unnecessary cardioversion shock causing increased ICP in such patients.

2. OBJECTIVE

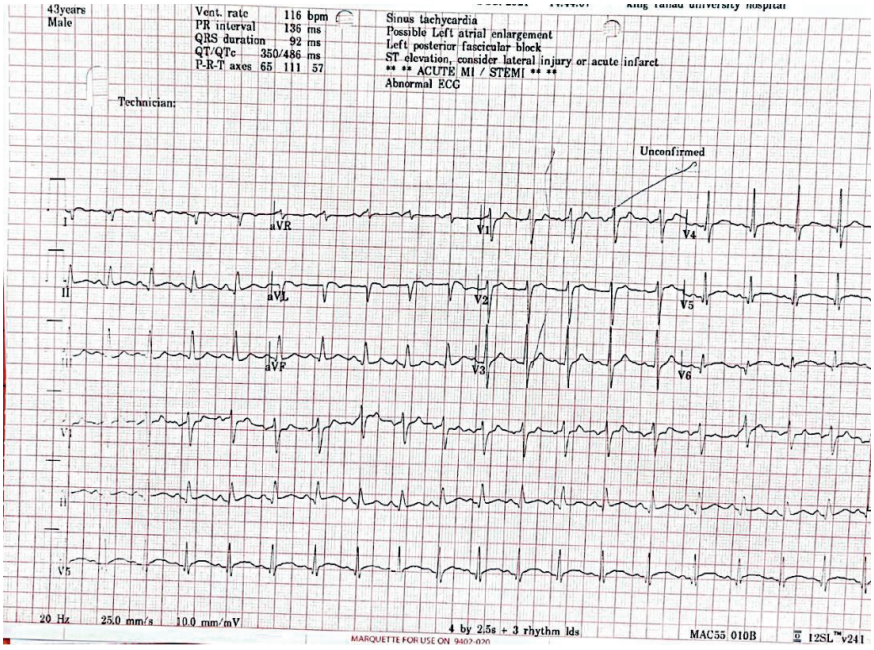
This case is a unique presentation of severe head trauma with a paucity of treatment approaches in the literature. The main purpose is to emphasize the approach to treating VT in severe head trauma patients.

Figure 1. ECG: ECG showed wide complex tachycardia.

3. CASE PRESENTATION

A 43-year-old male was brought to a trauma center emergency department as a victim of a fall down from two stories height. There were no complaints before the fall, his fall was mechanical, and as per the witnesses, he was acting normally before the fall.

Initial examination revealed a Glasgow Coma Scale (GCS) score of 3/15, his pupils were dilated and fixed, and he had no gag or corneal reflexes. His vital signs were as follows oxygen saturation was 98% on room air, his blood pressure was 128/72 mmHg, and his heart rate (HR) was 99 bpm. A trauma code was announced and ATLS Approach was followed.



Figrue 2 . Post cardioversion ECG showed normal sinus rhythm.

While on a monitor, his HR reached 174 bpm with regular wide complex waves. A rhythm strip was printed and confirmed the diagnosis of wide complex tachycardia, likely representing VT with a pulse. At that time his blood pressure was 160/110, and his GCS remained at 3/15. The rhythm was treated as unstable VT because of the level of consciousness. The patient received 100 J synchronized cardioversion without improvement. Higher joules were not delivered due to a concern of further elevation in intracranial pressure (ICP). Instead, he received the management of high ICP in the form of Mannitol 1g/kg, and rapid sequence intubation was done for airway protection using Propofol 2 mg/kg and Fentanyl 3 microgram/kg. After passing the tube, hyperventilation was initiated with a respiratory rate (RR) of 20 breaths per minute, targeting ET CO2 of 35 mmHg. After that, spontaneous restoration of sinus rhythm was achieved without further synchronized cardioversions or requiring antiarrhythmic medications.

After intubation and stabilization, extended focused assessment with sonography for trauma (eFAST), chest x-ray, and pelvic x-ray were done with no findings. The secondary survey showed scalp laceration and left ear bleed, the rest of the exam was normal.

Laboratory: Complete blood count (CBC), renal function tests (RFT), liver function test (LFT), and electrolytes all proved to be unremarkable.

Disposition: The patient was not a candidate for neurosurgery intervention and maximum medical manage-

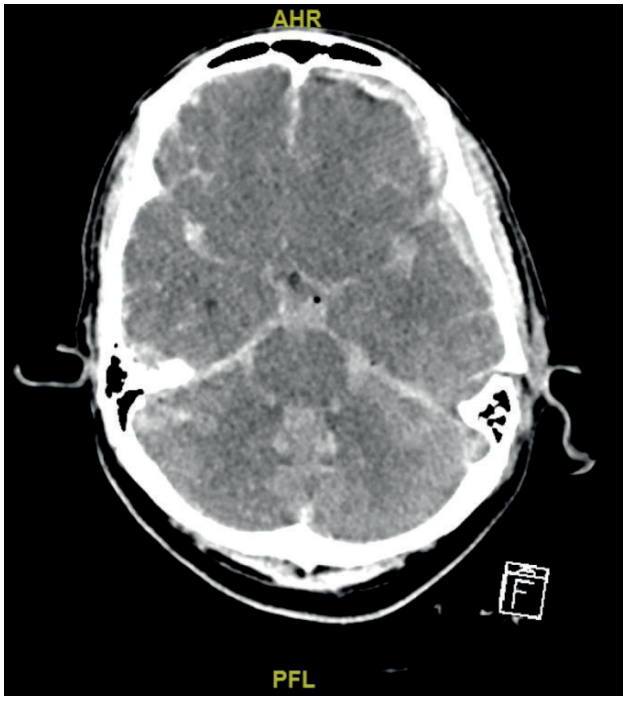


Figure 3. CT Scan: The patient was shifted to a CT scan which showed subgleal hematoma, subarachnoid and subdural hemorrhage, basal skull fracture, tonsillar brain herniation, depressed skull fracture, C6-C7 fracture, right lung contusions, T11 fracture.

ment was provided. He was admitted to the intensive care unit (ICU).

Hospital course: During the ICU length of stay, the patient was in maximum medical therapy for high ICP, and no arrhythmia was recorded. The patient underwent two brain death assessments done by a neurocritical consultant and both were positive. His brain death certificate was signed by two consultants and an organ donation protocol was initiated. By day 5 of admission, the patient was transferred to an organ transplant hospital for organ donation.

4. DISCUSSION

Ventricular tachycardia is an abnormal rhythm of the heart that can lead to pump failure and hypoperfusion. Depending on the perfusion status of the vital organs, VT can be divided into stable and unstable VT with pulseless VT as an extreme form of unstable VT. In most cases, VT presents with a wide, regular QRS complex and a rate greater than 120 beats/min.

It is commonly associated with underlying cardiac disease. On the other hand, a significant percentage of VT patients have no underlying cardiac cause (1, 2). Nevertheless, in these patients electrolyte abnormalities or a history of drug ingestion are usually found. The cardiac sequelae of TBI are important yet not well covered in the literature. However, some ECG changes related to TBI have been described in the literature and most of the ECG abnormalities described after an acute cerebral event were linked to SAH (4). The ECG abnormalities in SAH include T-wave changes, ST segment changes, U wave, QT prolongation, and pathological Q waves (5).

In the literature review, the incidence of VT following severe TBI (GCS < 9) was extremely rare (8). In 2003, Khogali, SS et al. reported a case report of a VT following a severe TBI in an alcohol-intoxicated patient in whom hypomagnesemia and hypocalcemia were found and attributed as the cause of arrhythmia and the patient was treated with magnesium sulfate with intermittent overdrive pacing which restored the sinus rhythm (7).

Another case was published in 2012 by Cronin D, et al. of a 19 years old man who fell from a horse and presented with severe TBI with a magnetic resonance imaging (MRI) study confirming the diagnosis of diffuse axonal injury (DAI) with cerebral edema. Two days after admission, the ICP monitor showed a reading of 40 mmHg with a simultaneous drop of potassium to 2.3 mmol/L. Potassium was corrected and the patient was shifted to the operation room for a decompressive craniectomy. After the operation, his potassium level reached 6.5 mmol/L which was not responsive to repeated infusions of insulin and dextrose. He then developed pulseless VT, cardiopulmonary resuscitation (CPR) was done for 40 min without return of spontaneous circulation (ROSC) and death was announced (8). Calcium, magnesium, and potassium levels are important for normal myocardium function. At abnormal levels, they can affect the cardiac cycle and myocyte action potential in different ways leading to arrhythmias and cardiac arrest (9). The possibility of electrolyte imbalance in our patient was excluded based on the normal level of the electrolytes. The toxicological cause of VT is another important cause that was excluded based on history, physical exam, and negative results of the common drugs of abuse. However, the possibility of false negative results and the presence of unmeasurable drugs remains possible and should be considered.

Another case was published in 2019 by Piastra M et al. of a 14 years old girl who presented to ED after pedestrian trauma with a GCS of 3-4/15 and bilateral mydriatic pupils patient's rhythm was sinus that suddenly evolved to pulseless VT and ventricular fibrillation (VF). 200 joules of DC shock were delivered and ROSC was achieved after one cycle of CPR with normal sinus rhythm. An early CT scan showed massive right hemisphere edema with right subdural hematoma. Decompressive craniectomy was done with hematoma evacuation and the patient was shifted to the Pediatric ICU. Later, echocardiography showed marked septal hypokinesia. She was kept in therapeutic hypothermia for 2 days, repeated head CT showed improvement in cerebral edema and coronary CT angiogram was normal (10). In 1982, McLeod AA, et al. published an observational study on the cardiac sequelae of TBI on 7 patients admitted to ICU with severe diffuse brain injury, all of them were young adults with no previous history of cardiac diseases. One of these patients was a 39 years old female who developed pulseless VT on day 5 of admission and died. An autopsy was done and a histological study confirmed myocardial necrosis (11). The urinary catecholamines of this patient were high which may support the theory of paroxysmal sympathetic hyperactivity (PSH). However, PSH was not documented in the literature at that time (12). Like in our patient, electrolytes were normal, and the possibility of cardiac disease or drug ingestion was highly unlikely. There was no clear cause of the sudden development of the arrhythmia in these two patients other than the severe TBI. In contrast to our patient, these two patients developed pulseless VT.

In literature, PSH was described in patients following severe traumatic brain injury which is a collection of symptoms and signs of sympathetic discharge leading to elevated blood pressure, heart rate, respiratory rate, deeping coma, and muscle rigidity. A cohort prospective study was conducted in 2012 by Fernandez-Ortega JF et al on 179 ICU patients admitted as severe TBI. They found that the mean time of its occurrence was 5.9 days (SD3.7 days) post-injury and no patient had PSH at the time of admission. Furthermore, it was not mentioned in the study if any patient developed VT as a consequence of the sympathetic discharge (12). The possibility of PSH leading to neurologically mediated tachycardia is less likely in our patient given the short time of the arrhythmia post-injury and the type of arrhythmia.

Another case of ECG changes after severe TBI was described in 2005 by Wittebole X, et al. In their paper, they described ECG changes mimicking the typical ischemic changes complicated by ventricular arrhythmia in a 32 years old male who was a victim of a motor vehicle accident. On the day of admission, his brain CT

and ECG were unremarkable, but MRI showed changes representing DAI. Five days after the admission, there was ST-segment elevation in the inferolateral leads and QT prolongation with multiple self-limited polymorphic VT. He was treated with Beta blocker and lidocaine infusions, troponin was elevated and cardiology was consulted as an acute coronary syndrome. Coronary angiography was done which showed normal coronary arteries, normal wall motion, and an ejection fraction of 66%. The patient improved in ICU with standard head trauma management and in 4 months follow-up, there were no ECG abnormalities (5). In this severe TBI patient with typical ischemic ECG changes, an extensive cardiac workup was done involving coronary angiography which is the gold standard test for the diagnosis of coronary artery disease and it was normal. However, the possibility of type 2 myocardial infarction must be considered which is secondary to supply and demand mismatch unrelated to acute thrombosis (6). Despite that, the ECG changes were correlated with his GCS without an obvious cardiac cause of his rhythm and ECG changes. The patient's general condition improved after treating the head injury and on his outpatient follow-up, he had a normal ECG without specific cardiac treatment.

Limitations

The major limitations were the absence of clear history about the causes of the fall, the difficulty of obtaining a history from the surroundings due to the language barrier. Osmolality level was taken after mannitol infusion which affects the result and its interpretation. Another limitation to be considered is the inability to perform extensive cardiac workups to exclude cardiac causes of VT due to the unstable general condition of the patient.

5. CONCLUSION

In our patient, there were no electrolyte abnormalities and no clear history of chest pain or cardiac disease, no history of drug ingestion, and no extensive thoracic trauma.

The restoration of sinus rhythm after treating the possibility of high ICP suggests that the cause of VT in this severe TBI patient was the high ICP.

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