Post traumatic recurrent ventricular tachycardia in intensive care unit: It's time not to give up

To the Editor,

Cardiac dysfunction with electrocardiographic (ECG), echocardiographic (ECHO) changes and sudden cardiac death following acute neurological injury has been well reported. Here, we describe the occurrence of recurrent ventricular tachycardia (VT) following traumatic brain injury (TBI) which reverted following surgical evacuation of extradural haematoma (EDH).

A 58-year-old hypertensive, diabetic male presented in triage after road side accident with score on Glasgow Coma Scale of E3V2M5. During examination, bilateral pupils were 2 mm in size and were reacting to light. A non-contrast computed tomographic scan of head revealed right sided frontotemporal EDH with midline shift of 5.5 mm. In the triage, he had a cardiac arrest following VT for which

cardiopulmonary resuscitation (CPR) was done. His trachea was intubated and 200 joules of direct biphasic synchronous shock was delivered thrice for subsequent ventricular tachycardia without pulse. Thereafter, the sinus rhythm was restored and the patient was shifted to intensive care unit (ICU). Surgical evacuation of haematoma was planned immediately. However, the relatives refused to give consent for surgery. Supportive care with aim to prevent raised intracranial pressure (ICP) was initiated i.e. head up, sedation, mechanical ventilation, constant monitoring of GCS, pupils and vitals. Twelve lead-ECG, ECHO and baseline investigations were performed. ECHO revealed left ventricular hypertrophy with no regional wall motion abnormality and a normal systolic function. Troponin levels were elevated (0.84 ng/ml). Relatives were again counselled for need of emergency surgery but they still refused. Over the next 12 hours, the patient developed 6 episodes of VT and defibrillation reverted VT to sinus rhythm every time. After 12 hours, relatives gave consent for surgical evacuation of EDH. Intraoperatively, defibrillator pads were attached to patient and defibrillator was kept ready. Surgery went uneventful and patient was shifted back to ICU. Following surgical evacuation of haematoma, episodes of VT disappeared. Further, postoperative course was uneventful. At discharge, patient had GCS score-E4VTM5. One year follow up, patient had score of one on modified Rankin scale.

The spontaneous disappearance of life-threatening ventricular arrhythmias following haematoma evacuation suggests a neurogenic cause rather than a cardiac cause in our case. Repeated VT was due to the presence of non-evacuated extradural haematoma. As per Monro-Kellie doctrine, once the compensatory reserve is exhausted, ICP increases and brain shifts may occur. Raised ICP leads to impairment of "Neuro-autonomic cardiovascular regulation (NCR)" system. The NCR has 3 parts. The first being neuronal component - cerebral cortex (insular and medial prefrontal region, amygdala, terminal stria), hypothalamus, brainstem (periaqueductal gray matter, parabrachial bridge, nucleus tractus solitarus, medial reticular zone of the medulla). Sympathetic fibres originating from cervical and thoracic ganglia of autonomic system (second component) which supplies atria, ventricles, coronaries and peripheral vasculature (last component). Rise in ICP often resulted in sympathetic surge, wave of central catecholamine outflow and alteration in hypothalamus, insular region, which leads to cardiac arrythmias, blood pressure alterations and myocardial damage. Excessive prolongation of the QTc interval driven by abnormalities in the insula and hypothalamus have been seen in acute neurological injuries.[1] In a prospective observational study, abnormal ECG changes (62%), elevated troponin I levels (54%), and echocardiographic abnormalities (42%) were seen in severe TBI.[2] Prathep *et al*. reported in a retrospective study, elevated serum troponin levels (24%), abnormal echocardiographic changes (22.3%), without documentation of ECG changes.[3]

Autonomic dysfunction in patients with extradural, subdural or intracerebral haematoma has been postulated as a risk factor for neurogenic cardiomyopathy.[1] Autonomic control of heart rate is disrupted proportional to the degree of neurologic insult after acute brain injury. ICP >30 mmHg or a cerebral perfusion pressure (CPP) <40 mmHg may be associated with marked autonomic dysfunction and poor outcome. Ventricular arrhythmia leading to sudden cardiac deaths are well reported. We believe episodes of ventricular arrhythmias were timely intervened during each episode in our patient. Hence, recurrent ventricular arrhythmias were witnessed; otherwise a sudden cardiac death would have been seen in our case too. Sudden rise in ICP intermittently would have precipitated autonomic dysfunction resulting in recurrent episodes of VT. This would have been conclusive had ICP been monitored (not done in our case). After surgical evacuation of haematoma, ICP normalized and further ventricular arrythmias disappeared. Our case is similar to that reported by Krishnamoorthy *et al*., where within 5 minutes of subdural haematoma decompression, complete cardiac dysfunction reversibility was seen (ejection fraction improved to 55% from 35% with resolution of hypokinesia).[4]

Repeated episodes of CPR and defibrillation are considered to be a poorer marker of outcome and efforts are generally considered less fruitful. However, constant vigilance and awareness of brain heart interaction with optimal management avoided an unfavorable neurological outcome despite multiple life-threatening episodes. At one-year follow-up, the patient is able to perform his daily activities.

To conclude, recurrent ventricular arrhythmias requiring CPR following TBI may not always be a marker of poor outcome. Definitive treatment of brain injury-induced cardiac arrhythmias includes surgical

removal of intracranial pathology at earliest if possible along with intensive care to decrease in the ICP. It is worth exploring the long-term outcome of head injury patients with cardiac dysfunction.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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