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Original article

Cardiac injury after convulsive status epilepticus in children

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ABSTRACT

Objective: Convulsive status epilepticus (CSE) is a medical emergency with high mortality that usually occurs within 30 days following the seizure activity. One of the potential mechanisms contributing to mortality in this period following CSE is cardiac injury. The aim of the present study was to evaluate cardiac injury after CSE in children.

Patients and methods: Sixty children presented with CSE were enrolled in this study. Thirty healthy children with matched age and sex were taken as a control. Electrocardiogram (ECG), echocardiographic examinations, plasma concentration of cardiac troponin I (cTnI) and brain-type natriuretic peptide (BNP) were done 6 h after control of seizure for patients and control groups.

Results: Thirty three patients were presented with CSE for the first time. ECG changes were present in 55% of patients with CSE in the form of conduction abnormalities, ischemic changes, and arrhythmias. Echocardiographic examinations revealed a significant increase in left ventricular end-diastolic dimension (LVEDD) and left ventricular end systolic dimension (LVESD) in patients with CSE than control group. Moreover, a significant decrease in LV systolic function and RV diastolic function were detected by tissue Doppler. The mean plasma concentrations of BNP and cTnI were significantly higher in patients with CSE than the control group (p value < 0.001). The overall mortality in our study was 8.3% (5/60); four of them had ECG changes. There was significant increase in duration of CSE, length of intensive care and hospital admission in CSE patients with ECG changes than those without ECG changes with p values 0.001, 0.031 and <0.001 respectively.

Conclusion: Cardiac injury in convulsive SE is common and may be under recognized. So, cardiac assessment should be a routine step in CSE patients' management.

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1. Introduction

Convulsive status epilepticus (CSE) is a seizure or series of recurring seizures lasting for more than 30 min although there is a general consensus now that single seizure exceeding 5–10 min should be treated as SE.[1] This medical emergency has a high mortality rate ranging between 10 and 30%. Death usually occurs within 30 days of the initial convulsive activity.[2] CSE can result from various etiology including stroke, hypoxia, metabolic derangement, head trauma, fever, intracranial hemorrhage and sudden antiepileptic drugs withdrawal.[3]

CSE activates the autonomic nervous system, increasing sympathetic nervous system control of the cardiac function increasing the blood pressure and heart rate leading to cardiac arrhythmias, pulmonary edema, and hypoxia.[4] Moreover, massive catecholamine release results in the formation of cardiac contraction bands that cause cardiac contractile dysfunction and may represent the cause of death in CSE.[5] These data suggest that CSE produces tachycardiac ischemia following the activation of the sympathetic nervous system resulting in cardiac myofilament damage and arrhythmogenic alteration in cardiac electrical activity. Also, these changes along with augmented cardiac afterload from tonic muscle contraction increase myocardial oxygen demand leading to a perfusion/demand mismatch that may be severe enough to produce subendocardial ischemia in the absence of coronary flow impairment.[6]

Electrocardiogram (ECG) abnormalities have been reported in 35% of seizures and include ventricular premature contractions, atrial fibrillation, atrioventricular block and QT prolongation.[7] Although ictal bradycardia (<40 b/m) is rarely reported, occurring in only 2% of seizures, it is considered as a potential contributor to sudden cardiac death. Most changes are benign and reversible, however potentially serious changes occur in 6–13% of seizures.[8] Death following CSE may result from uncontrolled tachycardia and long-term myocardial damage.[9]

Acute neurologic insults have been associated with specific patterns of neurocardiogenic injury known as stress-induced cardiomyopathy; or Takotsubo cardiomyopathy (TC); however, not all patients meet established criteria for this syndrome. The syndrome presents with reversible akinesis or dyskinesis of the left ventricular segment, associated with new ST-segment or T-wave abnormalities mimicking acute myocardial infarction (MI).[10] There are case reports of TC occurring after generalized CSE. So, it is very important to do echocardiography after CSE for early detection and treatment of this cardiac affection to improve the outcome of the affected patients. However, relation of TC to sudden unexpected death in epilepsy (SUDEP) is still controversial as some authors stated that it is a possible mechanism of SUDEP,[11] others stated that only complicated TC with ventricular arrhythmias is the one responsible for SUDEP.[12]

Many biochemical markers of cardiac injury are used nowadays to diagnose early cardiac affection e.g. creatine kinase-MB (CK-MB), cardiac troponins, brain natriuretic peptides (BNP), and N-terminal probrain natriuretic peptide (NT pro BNP).[13]

Cardiac troponin I (cTn I) is a myofibrillar protein regulating the interaction of actin and myosin and is considered as a highly sensitive and specific marker of myocardial injury. It is released from the injured myocardium to the circulation in proportion to myocytes injury. It is present only in the heart so considered as an excellent serum marker for myocardial injury.[14]

Brain type natriuretic peptide (BNP) is a neurohormone; first discovered in the hypothalamus but mainly synthesized in the myocardium in response to wall stress. It has diuretic, vasodilator and renin angiotensin aldosterone antagonist effects. Several studies suggested that its increased can be considered as a good marker of the severity of cardiac injury and ischemia.[15] The aim of this study was to evaluate the possible cardiac injury and affection following CSE in children.

2. Patients and methods

The present study is a prospective case-control study that was carried out in Tanta University Hospital, pediatric department, neurology unit in the period from June 2013 to June 2015. Sixty children came to emergency room (ER) with CSE were enrolled in the study. Thirty healthy children with matched age and sex were taken as a control and were chosen from children attending pediatric outpatient clinic in Tanta University hospital for minor complaint such as common cold, sore throat or skin infection with no history of cardiac, neurological, hepatic, renal or endocrinal disease checked by history, clinical investigations and their recorded files. The study has been approved by the local ethics committee of the Faculty of Medicine, Tanta University. Consents were obtained from all parents of patients. After control of seizures, patients were transferred to the neurology unit of pediatric department where investigations were done.

Exclusion criteria: patient with sepsis, cardiac, hepatic, renal, endocrinal or muscle disease were excluded from the study.

All the participants were subjected to the following:

- Thorough history taking: history of previous seizures and their frequency if present, medication, fever, trauma, developmental and obstetric history.
- Complete physical examination: including blood pressure estimation, complete neurological examination for signs of central nervous system infection, and fundus examination.
- Initial routine laboratory investigations: including complete blood count, liver function test, renal function test, blood sugar, serum calcium level, arterial blood gases, and antiepileptic drug serum levels (if indicated).
- Brain magnetic resonance imaging (MRI): for diagnosis of a cause of CSE and to diagnose children with structural brain disease.

The following investigations were done 6 h after control of CSE (confirmed by EEG):

- ECG: ECGs were done using 3 channels α 1000 apparatus and were initially classified by defining specific parameters