

Takotsubo cardiomyopathy associated with nonepileptic seizure after percutaneous endoscopic lumbar discectomy under general anesthesia

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Abstract Nonepileptic seizures are rare complication after general anesthesia. Postoperative seizure-induced oxidative stress promotes acute catecholamine toxicity of the myocardium. Takotsubo cardiomyopathy may be more frequent in the perioperative setting than commonly appreciated. We report a case of nonepileptic seizure developed during emergence from general anesthesia. The patient subsequently developed takotsubo cardiomyopathy. We now clearly recognize that patients with seizure activities after general anesthesia may be a higher risk for takotsubo cardiomyopathy.

Keywords Nonepileptic seizure · Takotsubo cardiomyopathy · General anesthesia

Introduction

Takotsubo cardiomyopathy is characterized by acute onset of reversible left ventricular (LV) dysfunction, ST-segment elevation, and a minor elevation in serum levels of cardiac enzymes. The condition is usually precipitated by psychological or physical stress. Several cases have been reported showing that seizure patients are particularly

vulnerable to catecholamine toxicity of the myocardium [1]. We report a case of a nonepileptic seizure that occurred in the operating room while the patient was emerging from general anesthesia. The patient went onto develop takotsubo cardiomyopathy in the intensive care unit.

Case history

A 67-year-old woman weighing 72 kg visited the neurosurgical department with severe low back pain that radiated to her right leg. Lumbar disc (L3–L4) herniation was diagnosed, and the patient was scheduled for elective percutaneous endoscopic lumbar discectomy. She had a prior history of hypertension and hypercholesterolemia. Routine premedications (midazolam, 3 mg, IM; glycopyrrolate, 0.2 mg, IV) were given approximately 30 min before surgery. Anesthesia was induced with 100 mg propofol IV and a 2-mg/h remifentanil IV infusion followed by 50 mg rocuronium IV to facilitate endotracheal intubation. Anesthesia was maintained using sevoflurane at 1.0–1.5 minimum alveolar concentration in 50% oxygen and a remifentanil infusion IV at 1.0–2.0 mg/h. The endoscopic lumbar discectomy, which lasted about 2 h, was performed uneventfully. At the end of surgery, after reversal of the residual muscle relaxant effect, the patient resumed spontaneous ventilation with eye opening on command. After extubation, the patient began to exhibit tonic-clonic seizure activity. She became apneic, with oxygen saturations in the 80% range. Vital signs showed a blood pressure of 160–180/80–100 mmHg and a heart rate 80–100 bpm. The anesthesiologist placed an oral airway, which was promptly batted out of position by the patient, after which positive-pressure mask ventilation was performed. An arterial blood gas showed a pH of 7.328 partial

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pressure of oxygen in arterial blood (PaO₂) of 271 mmHg and a partial pressure of carbon dioxide in arterial blood (PaCO₂) of 47.4 mmHg. Serum electrolytes were within normal limits. Oxygen saturations increased to the 90% range with ventilation. Muscle rigidity was sustained for 10 min, and 250 mg of thiopental was administered IV. Seizure activity stopped after thiopental administration. Intubation was performed after administration of 40 mg rocuronium IV, and mechanical ventilation was instituted. A computed tomography (CT) brain scan showed no abnormalities. The patient was transferred to the neurosurgical intensive care unit (ICU).

One hour after arrival in the ICU, the patient developed tonic-clonic seizure activity. We consulted a neurologist. An emergent electroencephalogram (EEG) was recorded during the seizure but provided no evidence for epilepsy (Fig. 1). Her seizure was analyzed as nonepileptic. A 500-mg phenytoin infusion IV was started, and seizure activity disappeared. An electrocardiogram (ECG) showed ST depression in leads II, V1, and V2. A bedside two-dimensional echocardiogram showed hypokinesia of the basal-inferior LV and of the anterolateral wall. Subsequently, the patient underwent cardiac catheterization, which was interpreted as a normal coronary angiogram. Left ventriculography demonstrated reverse-type LV takotsubo cardiomyopathy (Fig. 2). Serum troponin I (0.09 ng/ml) and creatine kinase-MB (CK-MB) (3.4 μg/l) were slightly elevated. Several hours later, the patient awoke fully without seizure activity. She was diagnosed with a nonepileptic seizure and reverse-type takotsubo cardiomyopathy, and conservative treatment and supportive care were instituted.

ECG performed on postoperative day 5 showed normal wall motion and LV function. During the entire hospitalization, the patient had no further seizure activity. She was discharged on postoperative day 24 without seizure activity and with no need for anticonvulsant medication.

Discussion

Nonepileptic seizures may resemble generalized or partial seizures but do not involve abnormal discharges of cortical neurons. They are caused by either physiological or

Fig. 1 Electroencephalogram (EEG) was recorded during the seizure in the intensive care unit. No epileptiform discharges were seen. This EEG is suggestive of mild diffuse cerebral dysfunction

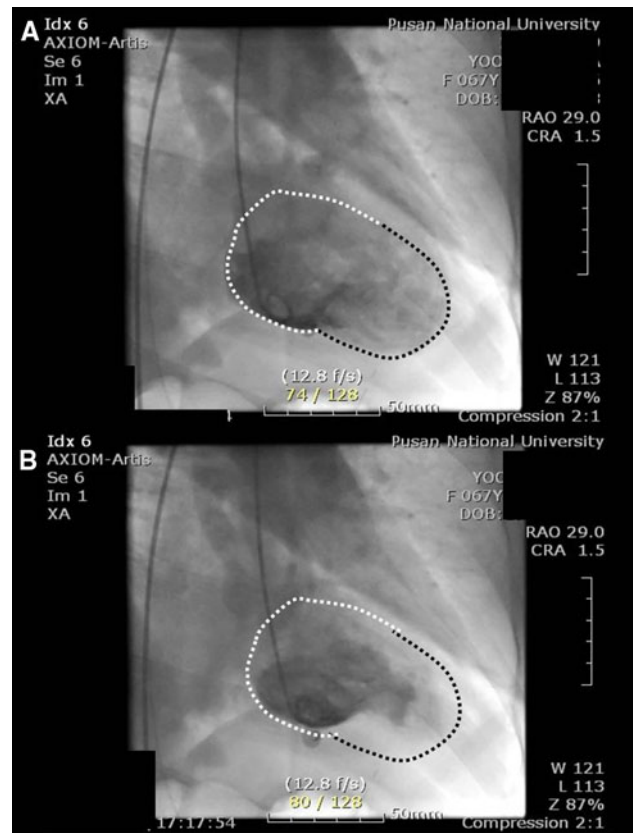
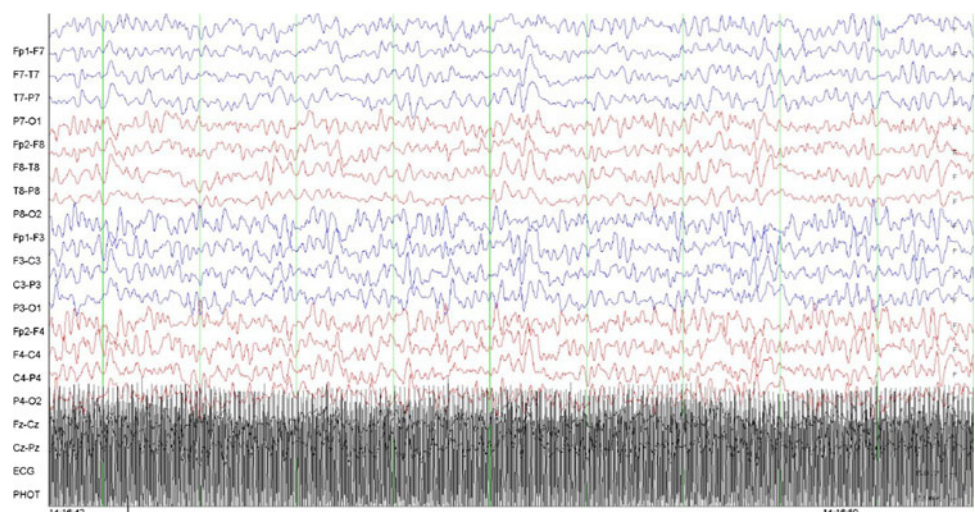


Fig. 2 Left ventriculogram during diastole (a) and systole (b). Black dotted lines (apex of left ventricle) clearly contracted during systole (b) compared with diastole (a). White dotted lines (basal wall of left ventricle) showed no contraction during systole (b) compared with diastole (a)

psychological conditions [2]. Seizures commonly develop in relation to general anesthesia [3]. Some anesthetic agents can also trigger seizures [4]. Glosser et al. [5] reported nonepileptic seizures after cranial surgery to treat intractable epilepsy. Despite these previous reports, nonepileptic seizures are rare complication after general anesthesia and are classified as either physiologic or psychogenic in origin.

In general, the postoperative physiologic environment, which can involve hypoxia, hypoglycemia, hyponatremia, and arrhythmias, lowers the seizure threshold. Our patient had no abnormal findings. Possible causes of nonepileptic seizures include anesthetics (propofol, sevoflurane, remifentanyl) and a response to the acute stress of emergence from general anesthesia. There is debate over whether propofol is a pro- or anticonvulsant. Walder et al. [6] reported that propofol administration led to seizures during induction or emergence, with generalized tonic-clonic seizures being the most frequently observed. Our patient was administered an induction dose of propofol, as the mean half-life is in the range of 30–70 min [7]. We suggest the operative time (2 h) was enough to reduce propofol concentration in brain tissue. It is thus difficult to ascribe the development of the nonepileptic seizure to propofol. The incidence of abnormal movements during sevoflurane anesthesia has been reported to be about 5%. Movement is usually reported during anesthesia induction or maintenance [8]. Emergence agitation from sevoflurane anesthesia is frequent in children but rare in adults. Nielsen et al. [9] reported generalized tonic-clonic seizures in a patient given a bolus dose of remifentanyl. Remifentanyl can cause myoclonus or muscle rigidity after an IV bolus, and it is related to remifentanyl-induced seizures rather than true seizures. Although large doses of remifentanyl can cause the development of subsequent acute opioid tolerance, our patient was administered small doses of remifentanyl by IV infusion. We suggest that there is no relationship between the remifentanyl infusion and acute opioid tolerance. There is currently not enough evidence to suggest that anesthetics induced the seizure in this patient.

Nonepileptic seizures usually develop predominantly in females and are related to previous psychiatric disorders [2]. We could not detect any psychogenic problem in our patient's history. A dysfunctional family relationship is a predisposing factor for nonepileptic seizures [10]. Her family recommended conservative treatment with physical therapy rather than the operation. It would seem likely that the causative factor for the nonepileptic seizure was the operation and the added acute stress of emergence from general anesthesia.

Takotsubo cardiomyopathy, also commonly referred to as stress-induced cardiomyopathy, is a type of nonischemic cardiomyopathy. Typical takotsubo cardiomyopathy shows

bulging out of the apex with preserved function of the basal wall. Takotsubo cardiomyopathy is well recognized and reported in the perioperative period, and a recent seizure is another predisposing factor for stress-induced cardiomyopathy [11]. The exact mechanism of takotsubo cardiomyopathy is unknown. Seizures could increase the cardiac oxygen demand, myocardial contractility, heart rate, and serum catecholamine level [12]. Coronary vasospasm tends to develop and cause nonischemic cardiomyopathy induced by the seizure. Our patient's clinical and laboratory findings were in accordance with the typical characteristics of takotsubo cardiomyopathy: postmenopausal women, ST-depression on ECG, minimal abnormality in serum troponin I, normal coronary angiography, and severe emotional or physical stress. Our patient had basal-wall hypokinesia with preservation of the other wall motions, confirmed by left ventriculography. Basal-wall hypokinesia-type takotsubo cardiomyopathy is observed in 40% of stress-induced cardiomyopathy [13].

In conclusion, when seizure-like activity develops during emergence from general anesthesia, nonepileptic seizures must be excluded. During seizure-like activity, cardiac damage, including takotsubo cardiomyopathy, can occur. Patients undergoing noncardiac surgery experience cardiovascular complications during the postoperative period. We must clearly keep in mind that takotsubo cardiomyopathy should be considered in the differential diagnosis of acute coronary syndrome in the postoperative period.

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