

Takotsubo cardiomyopathy – a rare, but serious, complication of epileptic seizures

Cardiomiopatia de Takotsubo – uma complicação rara, mas grave, de crises epiléticas

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Takotsubo cardiomyopathy (TKC) is a reversible, yet potentially fatal, syndrome triggered by stressful conditions, including seizures, with increasing recognition in clinical practice¹. We report a TKC in a patient with unexplained sinus tachycardia and troponin elevation after generalized seizures.

CASE REPORT

A 44 year-old woman presented with history of a subarachnoid haemorrhage five years ago, after a right posterior cerebral artery pseudoaneurysm rupture. Coil embolization was performed, complicated with a right cortico-subcortical temporo-occipital infarct with a slight left hemiparesis as a

result. She was on acetylsalicylic acid 150 mg/day, clopidogrel 75 mg/day and prophylactic phenytoin 300 mg/day.

Patient was admitted after four inaugural generalized tonic-clonic seizures (GTCS) that occurred in close succession during two hours. Consciousness was impaired between seizures, deeming it a clinical status epilepticus. She was assisted during a GTCS, sedated with midazolam perfusion and intubated, having ceased motor activity. After admission, she had no further seizures and regained conscious slowly after stopping midazolam, presenting a grade 4 left hemiparesis. Patient had infratherapeutic levels of phenytoin and started IV phenytoin. Electroencephalography (EEG) revealed pharmacological beta activity, but no epileptic activity. Brain computed

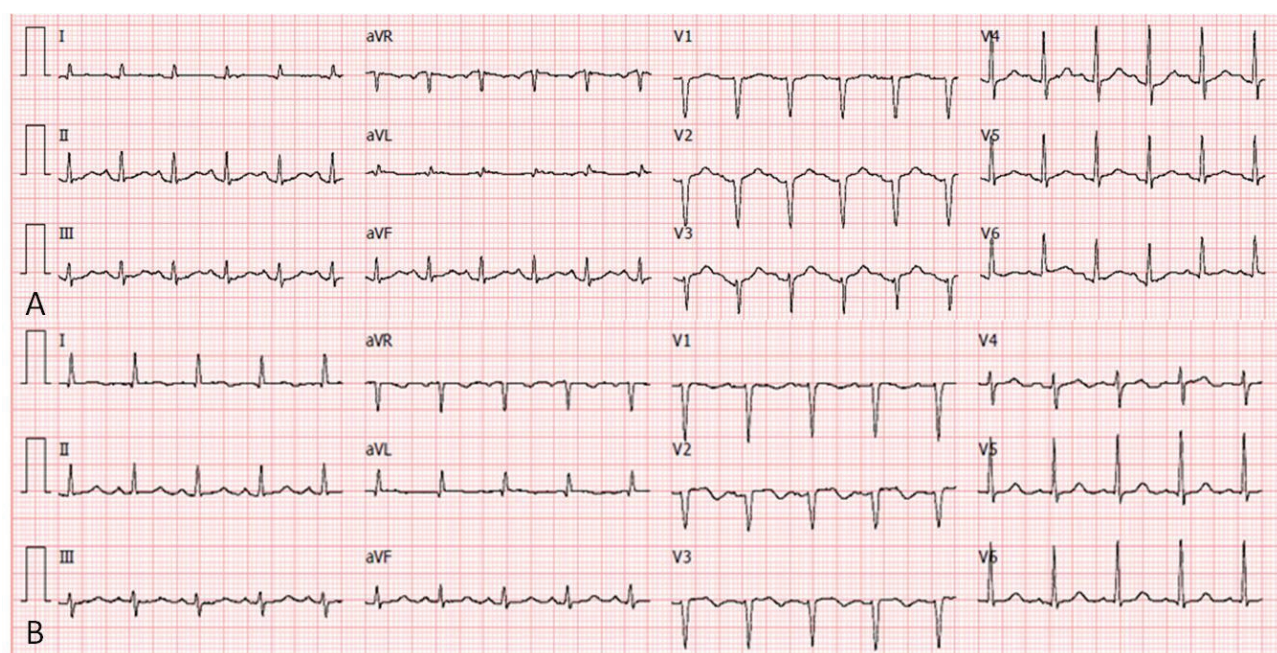


Fig 1. Electrocardiogram (ECG). (A) Admission ECG with sinus tachycardia; (B) control ECG with T wave inversion (V1-V3).

tomography (CT) identified the right temporo-occipital infarction, overlapping previous exams. Seizures were interpreted as symptomatic due to the cortical lesion and infratherapeutic levels of phenytoin.

After recovery, she was afebrile, normotensive, but tachycardic (110–130 bpm). She had no cardiac or respiratory complaints. Electrocardiogram (ECG) showed sinus tachycardia and 12 hours later T wave inversion in V1–V3 leads (Figs 1A and B). Serum troponin was 6.48 ng/mL. Transthoracic echocardiogram demonstrated mild depression of left ventricle systolic function, hypokinesia of apical segments with hypercontractility of the basal ones. These kinetic changes were confirmed by left ventriculography (Fig 2). Coronarography was normal, establishing the diagnosis of TKC. She continued treatment with ramipril and double antiplatelet therapy, previously prescribed because of her endovascular intervention, with clinical improvement.

DISCUSSION

Takotsubo cardiomyopathy is a reversible dysfunction of the left ventricle in the absence of coronary disease, probably related to excessive catecholamine release due to sympathetic hyperactivity induced by stressful factors².

Patients present signs of ventricular dysfunction or only sinus tachycardia. Chest pain is infrequent in post-epileptic TKC³. Troponin levels are elevated in the absence of coronary disease, and ECG may present with Q waves, ST segment elevation or T wave inversion². Treatment is supportive and mortality is as high as 8%³.

Association between TKC and epileptic seizures (focal or generalised) is increasingly reported, and seizures may

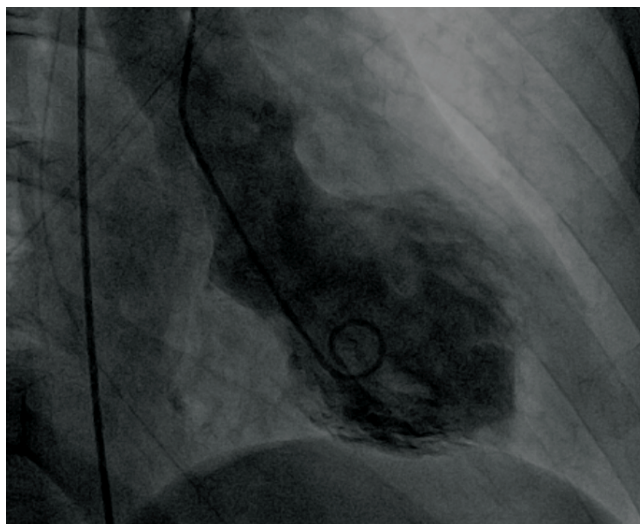


Fig 2. Ventriculography with typical left ventricular apical ballooning – hypocontractility of the apical segments and hypercontractility of the basal segments.

be the triggering factor of catecholamine release that promotes the cardiotoxic response. Focal epileptic activity of the temporal lobe may cause autonomic dysfunction that promotes cardiac damage⁴. The majority of post-epileptic TKC cases are women, mean age 62.7 years. Epilepsy etiology may be varied¹.

Cardiac complications are one of the main causes of mortality in epilepsy⁵. Incidence of TKC after epileptic seizures is unknown and could be related to cardiac death in epileptic patients¹. The authors highlight the importance of suspecting TKC in patients with seizures and signs of cardiac dysfunction as subtle as sinus tachycardia. Timely recognition of this complication is important to provide adequate supportive care.

References

1. Dupuis M, van Rijckevorsel K, Evrard F, Dubuisson N, Dupuis F, Van Robays P. Takotsubo syndrome (TKS): a possible mechanism of sudden unexplained death in epilepsy (SUDEP). *Seizure* 2012;21:51–54.
2. Chockalingam A, Mehra A, Dorairajan S, Dellsperger KC. Acute left ventricular dysfunction in the critically ill. *Chest* 2010;138:198–207.
3. Stöllberger C, Wegner C, Finsterer J. Seizure-associated Takotsubo cardiomyopathy. *Epilepsia* 2011;52:160–167.
4. Naganuma M, Isoda K, Ishizaki M, Ito K, Hirano T, Uchino M. Epilepsy and Takotsubo cardiomyopathy: a case report. *Intern Med* 2011;50:2397–2399.
5. Rodríguez de Antonio LA, Aguilar-Amat Prior MJ, Iváñez-Mora V, Díez-Tejedor E. Miocardiopatía de Takotsubo como complicación de un estado epiléptico. *Rev Neurol* 2011;53:673–676.