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Short communication

Takotsubo syndrome (TKS): A possible mechanism of sudden unexplained death in epilepsy (SUDEP)

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ABSTRACT

We report a case of Takotsubo syndrome after epilepsy, and review the literature. We identified 59 cases of Takotsubo syndrome after focal or generalised epilepsy. As in Takotsubo syndrome in general, the patients were mostly female (84%), with a mean age of 63 years, and the evolution was generally favourable. There was one death and one stroke, and 4 cases were of relapsing Takotsubo after a new seizure. Takotsubo syndrome may induce cardiac arrhythmias. A near-SUDEP (sudden unexplained death in epilepsy) was reported in one patient. Animal models of SUDEP have shown similar cardiac lesions to those seen in Takotsubo syndrome, and strengthen the hypothesis of a link between these conditions. Takotsubo syndrome after epilepsy may be relatively common; we suggest measurement of serum troponin levels in high-risk patients and cardiac follow-up.

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1. Introduction

Takotsubo syndrome (TKS) is an acute cardiomyopathy that occurs after stressful conditions, including seizures and status epilepticus.[1] SUDEP (sudden unexplained death in epilepsy) may be secondary to unexplained respiratory failure or cardiac arrest related to seizures.[2] We report one case of TKS after seizures and review the literature suggesting a link between TKS and SUDEP.

2. Case report

A 50-year-old woman with a stressful psychiatric condition, presented with a first tonic-clonic seizure after a partial seizure with eye and head deviation to the right. A second fit occurred in the emergency room (March 2009). Cardiac auscultation revealed a gallop rhythm. The serum troponin level was 7.51 ng/ml (<0.15 ml) and the ECG showed antero-septal Q waves (Fig. 1). The patient did not undergo endotracheal intubation and received no vasoactive drugs before the first ECG. Echography demonstrated transient antero-infero-apical hypokinesia with an ejection fraction estimated at 36%. coronarography was normal and left ventriculography (Fig. 2) revealed akinesia of the basal segment and mid portions of the left ventricle and hyperkinesias of the apex. A diagnosis of "inverted TKS"[3] was established. The patient was treated with acetylsalicylic acid, bisoprolol, perindopril and sodium valproate (1000 mg/day), which was later changed to lamotrigine because of weight gain. Prolonged EEG monitoring and brain MRI were normal. The patient recovered fully from cardiac and neurological points of view.

3. Discussion

TKS, first described in 1991 by Dote et al.[4] in Japanese patients, is increasingly recognized in cardiology. TKS occurs after a stressful condition, including seizures, and is considered as a possible toxic reaction to excessive and acute catecholamine release, but other factors, such as vasospasm or microvasculature failure, are also probably involved.[1] The exact mechanisms are not yet fully understood.[5]

Our patient fulfilled the criteria for TKS: reversible akinesis or dyskinesis of the left ventricular segment, associated with new ECG ST-segment or T-wave abnormalities mimicking acute myocardial infarction. The physiopathology of the reversible cardiomyopathy with apical sparing seen in our patient is similar to that of TKS cardiomyopathy, but with an inverse left ventricular pattern: "Inverted TKS". Coronary angiography should be performed as soon as possible in such cases to rule out obstructive coronary disease.[6,7] TKS predisposes patients to embolism, cardiogenic shock, heart failure, arrhythmia and sudden death, with an acute lethality of up to 8%. The differential diagnosis from acute myocardial infarction is not always easy, and treatment for myocardial infarction is often the first therapeutic option. TKS is a

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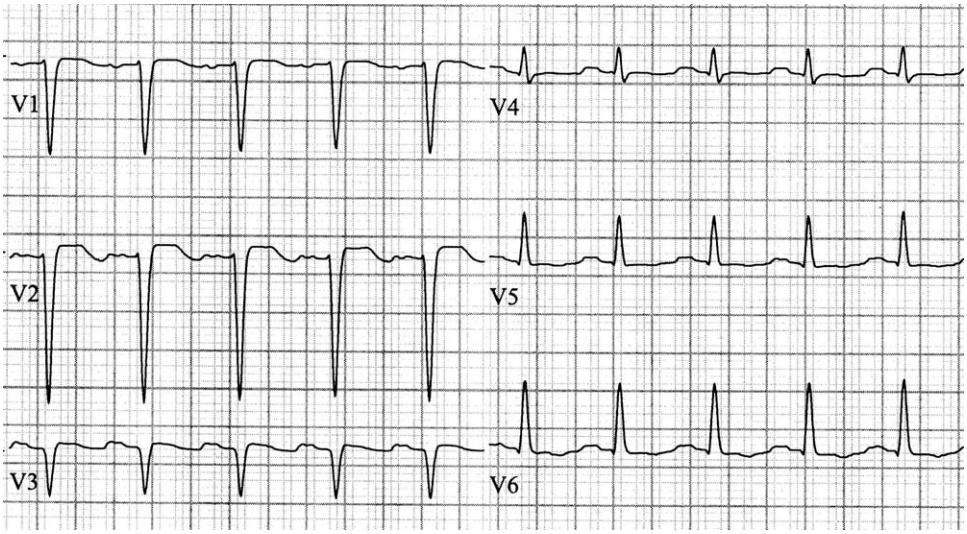

Fig. 1. ECG: antero-septal Q waves.

condition that requires specialist care and diagnosis can be difficult with possible differential diagnoses including prolonged syncope and convulsions due to acute coronary syndrome. Once the diagnosis is confirmed (high troponin, Q-wave, cardiac ballooning, normal coronarography, sometimes cardiac MRI), there is no specific treatment and management relies on supportive therapies and methods to prevent possible complications, such as giant thrombus. Unless there are acute complications, full recovery is usual.

TKS after seizures has seldom been reported. We found 59 documented cases of possible TKS secondary to seizures (Table 1), including 4 cases[8–11] of relapsing TKS after new seizures. In 5 of these 59 cases,[2,12,13] a diagnosis of TKS was not suggested even in the discussion sections of these papers, despite description of the specific cardiac characteristics of TKS. Hence, the association of TKS and epilepsy may be underreported and underestimated in clinical practice. Reports of multiple cases by different teams support this view (Table 1). Some of the cases reported were exposed to additional stress after seizure, like intubation,[14] and a small minority received vasoactive drugs.[8,10]

The four cases of relapsing TKS after seizure, two of whom received vasoactive drugs,[8,10] further strengthen the etiological link. For all the post-epileptic TKS cases, we note that the majority were female (84%), with a mean age of 62.7 years. The epilepsy is often poorly described in these TKS cases. At least 18 patients suffered from focal epilepsy related to vascular (13 cases[8,12,15–23]) or post-traumatic (3 cases[11,12,24]) lesions, mesio-temporal sclerosis (2 cases[2,25]), meningioma (1 case[12]), or delayed post-surgery (1 case[26]). Generalised seizures were related to hyponatremia (4 cases[10,14,27,28]) alcoholism (8 cases[10,14,19,23,29,30]) and electroconvulsive therapy (ECT) (3 cases[31–33]). Status epilepticus (convulsive or not) was noted in 5 cases.[8,25,34–36] In most cases, there was full cardiac recovery, as described in TKS, but one case was lethal.[16] In one case,[21] a stroke occurred on the 4th day secondary to embolism. One giant cardiac thrombus was reported in another case.[37] Many authors comment on the absence of chest pain in their patients.[14,23,38,39] Shimizu[36] documented elevated catecholamine levels in their case and performed a cardiac biopsy, which showed similar abnormalities to those previously described in TKS. Stöllberger compared 36 seizure-associated TKS episodes to a series of non-epileptic TKS patients and found that the epileptic TKS patients were younger, more frequently male, and rarely had chest pain.[10]

SUDEP is a serious complication of epilepsy. Its physiopathology is still under debate. Some animal models suggest cardiac abnormalities[5] similar to TKS. One case of seizure-associated TKS was reported as a near-SUDEP,[2] and many

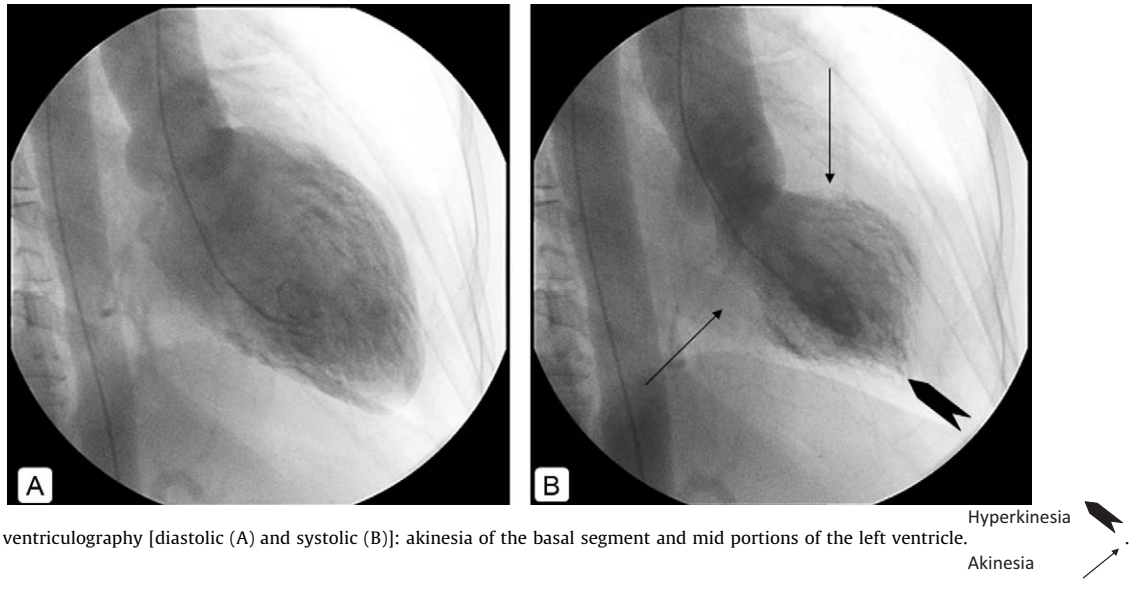

Fig. 2. Left ventriculography [diastolic (A) and systolic (B)]: akinesia of the basal segment and mid portions of the left ventricle.

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