TAKO-TSUBO SYNDROME: A RELAPSING CASE DURING PARTIAL CONVULSIVE STATUS EPILEPTICUS

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Introduction

Tako-Tsubo syndrome (TTS) is a transient, mostly female gender-related, cardiomyopathy characterized by apex akinesia/hypokinesia, systolic dysfunction, ECG ischaemic abnormalities, cardiac enzymes elevation, with or without anginal chest pain, and normal coronary angiography. Increased sympathetic activity triggered by severe emotional or physical stress has a pathogenic role. Among CNS disorders TTS is most frequently associated with subarachnoid hemorrhage, followed by epilepsy (1). We report a case of TTS occurring during partial convulsive status epilepticus (PCSE) and relapsing 4 years later.

Case report

A 74-year-old woman, with a recent history of sepsis, was admitted to our department with left-sided hemiparesis and continous clonic jerks. Previous neurological history was unremarkable. EEG documented right PCSE, which was unresponsive to first and second line intravenous anti-epileptics (e.v. diazepam, and valproate)(Figure 1A). Brain MRI showed volume loss and hyperintensity of the right hippocampus, suggesting mesial temporal sclerosis, diffuse cerebral atrophy and vasculopathy (Figure 2, A-B-C-D). Routine ECG showed diffuse ST-elevation and negative T-waves (Figure 3A); troponin levels were elevated. Cardiac US revealed transitory contractile abnormalities of apex and reduced systolic function (Figure 4). Coronary angiography was unremarkable (Figure 5). PCSE was successfully treated with general anaesthetics (Figure 1B), later gradually switched to oral diphenylhydantoin and oxcarbamazepine. Sepsis solved with e.v. cephazolin. Repeated ECG, cardiac US and enzymes normalised within a month-time (Figures 3 and 4, B). A diagnosis of TTS was finally made.

After four years, during a long-lasting gastroenteritis causing hypomagnesemia (0.50 mMol/l, n.v.>0.70), the patient presented a cluster of left partial convulsive seizures evolving in right PCSE. At cardiac us severe apical hypokinesia was demonstrated during PCSE, with subsequent normalization in the next month. ECG, troponin levels showed the same transitory modifications suggestive of TTS, as previously observed. Hypomagnesemia was corrected with seizures resolution. Brain MRI revealed progressive cerebral atrophy more pronounced on the right and mostly characterized by right ventricular enlargement associated with periventricular gliosis, and progression of the cerebral vasculopathy (Figure 2, E-F-G-H).

Conclusions

TTS is a rare complication of epilepsy, being only three patients with partial complex seizures reported so far (2) Furthermore, this is the first case of relapsing TTS during PCSE. A structural abnormality, i.e. mesial temporal sclerosis, together with triggering factors, as sepsis/hypomagnesemia, cause clusters of partial complex seizures evolving in status epilepticus. Asymmetric progression of the cerebral atrophy is probably related to right temporal epilectic activity (3). It is noticeable that recovery of PCSE was associated with TTS resolution. This case report underlines the close link between central nervous system disease and cardiac abnormalities and confirms the need for monitoring cardiac function during seizures (2)

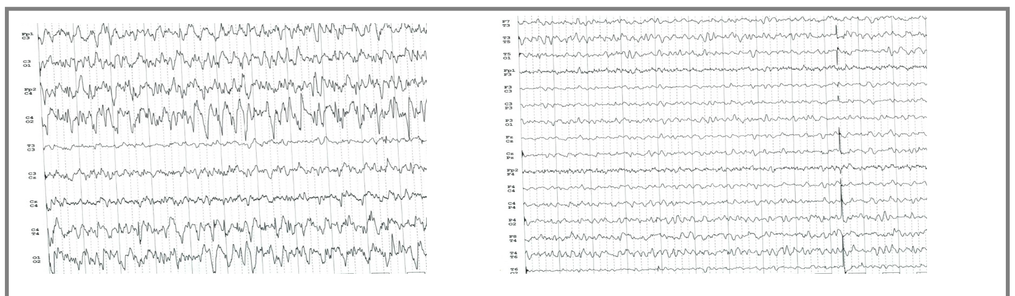


Figure 1- A Right centro-temporal partial convulsive status epilepticus spreading to the ipsilateral hemisphere and more rarely to the controlateral one. **B** Resolution of PCSE. Widespread sharp-waves.

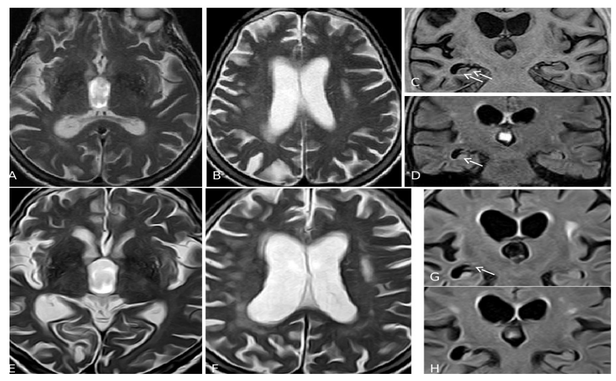
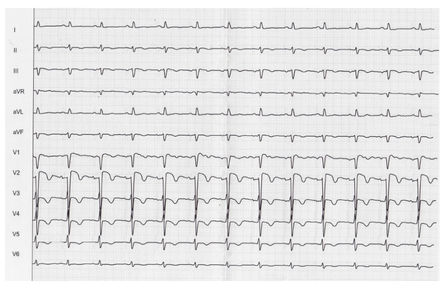


Figure 2 – TOP (Brain MRI performed over the first admission). Axial T2-weighted images, A and B, demonstrate diffuse cerebral atrophy with a slight asimmetry for R>L and focal hyperintensities in the subcortical and periventricular white matter probably related to ischaemic damage. Coronal IR, C, and FLAIR images, D, show atrophy and mild hyperintesity in the right hippocampus (arrows).
BOTTOM (Brain MRI performed over the second admission, 4 years later). Axial T2-weighted images, E and F, and coronal FLAIR images, G and H, demonstrate worsening of the cerebral atrophy with marked ventricular and sulci enlargement more on the right and a further atrophy of the hippocampus.



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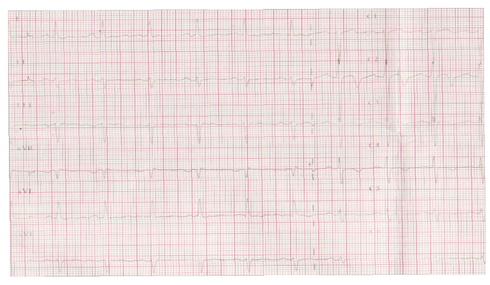
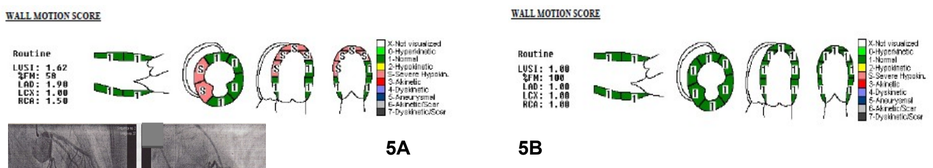


3B

Figure 3- A Diffuse ST-elevation and negative T-waves over PCSE
B Resolution of ECG abnormalities



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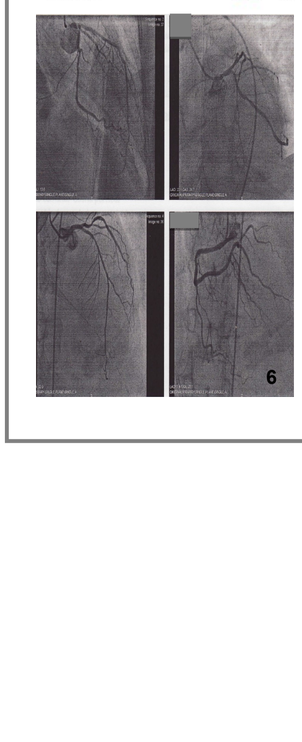

Figure 5 Wall motion score index revealing apex hypokinesia (A) over PCSE, with full resolution after 1 month-time (B). At cardiac US fraction of ejection was 30% over PCSE, which became normal after one month (FE 60%).

Figure 6 At coranorography no significant stenosis could be demonstrated.

References

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