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Case report

Seizure induced ventricular fibrillation: A case of near-SUDEP

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1. Introduction

Cardiac arrhythmias are a common complication of partial seizures, with ictal sinus tachycardia present in 16 out of 20 patients having undergone prolonged electrocardiographic (ECG) monitoring with implantable loop recorders.[1]

Ictal bradyarrhythmia and ictal asystole are more concerning but rare: a prevalence of 0.34–0.4% has been reported in large series.[2,3] These situations have received a lot of attention recently because they have been postulated as one of the mechanisms underlying sudden unexpected death in epilepsy (SUDEP), although the supporting evidence remains fragmented.[4]

The few documented cases of SUDEP (or near-SUDEP) mainly showed apnoea and hypoventilation to be the terminal event, although there are some cases with a primary cardiac mechanism.[5,6]

We report the case of a patient suffering from drug-resistant focal epilepsy who developed ventricular fibrillation at the end of a convulsive seizure, requiring cardiopulmonary resuscitation (CPR).

2. Case report

A 27-year-old male developed seizures characterised by staring, leftward deviation of the head, tonic contraction of all limbs, always with subsequent generalised tonic–clonic seizure. He has no family history of epilepsy, no family history of sudden unexpected cardiac or unexplained death and no history of febrile convulsions. His seizures were likely to occur both during the day and at night. Several interictal EEG showed semirhythmic theta activity in the right temporal lobe regions. A first brain MRI was unremarkable. Seizures became refractory to medical treatment and he had a brain trauma due to a seizure with subsequent extradural haemorrhage in the right temporal region. Various antiepileptic drugs were tried, but he still had 3–4 seizures per year despite polytherapy with valproate 750 mg, carbamazepine 1400 mg, lamotrigine 300 mg and levetiracetam 2000 mg.

At the age of 44 he was involved in a minor road accident: he was described as awake, able to speak, but markedly disoriented, so an ambulance was called. When the paramedics arrived he had a seizure characterised by staring and loss of awareness prior to secondary generalisation. Immediately after the seizure, while he was still unconscious, the paramedics applied automated external defibrillator electrode pads to his chest and the device revealed pulseless ventricular fibrillation. Cardiopulmonary resuscitation was started and he was defibrillated, until a rhythm was re-established after 2 min (Fig. 1).

He was admitted to the cardiology unit and he underwent a full cardiological work up: basal EKG was unremarkable; chest CT scan, transthoracic echocardiogram and heart MRI showed normal results. Blood tests at the time of admission showed no electrolyte imbalance.

An implantable cardiac defibrillator was placed for secondary prevention and he was discharged 5 days later without complications.

A few months later during the routine device follow-up, another episode of ventricular fibrillation was detected. The patient's diary reported a convulsive seizure that day. The patient did not remember the discharge.

3. Discussion

We report a case of "Near-SUDEP" (defined as a cardiorespiratory arrest after a seizure, successfully resuscitated, with no obvious cause and no other relevant preexisting conditions),[7] with a possible cardiac mechanism.

We hypothesise that the second episode of ventricular fibrillation might have been associated with the reported seizure as well: this would explain why the patient did not remember the episode, despite the discharge usually being very painful.

Seizures associated with arrhytmias usually arise from frontal, temporal, and insular cortex and even if it remains unclear if it is spreading to a certain brain region it is predominantly associated

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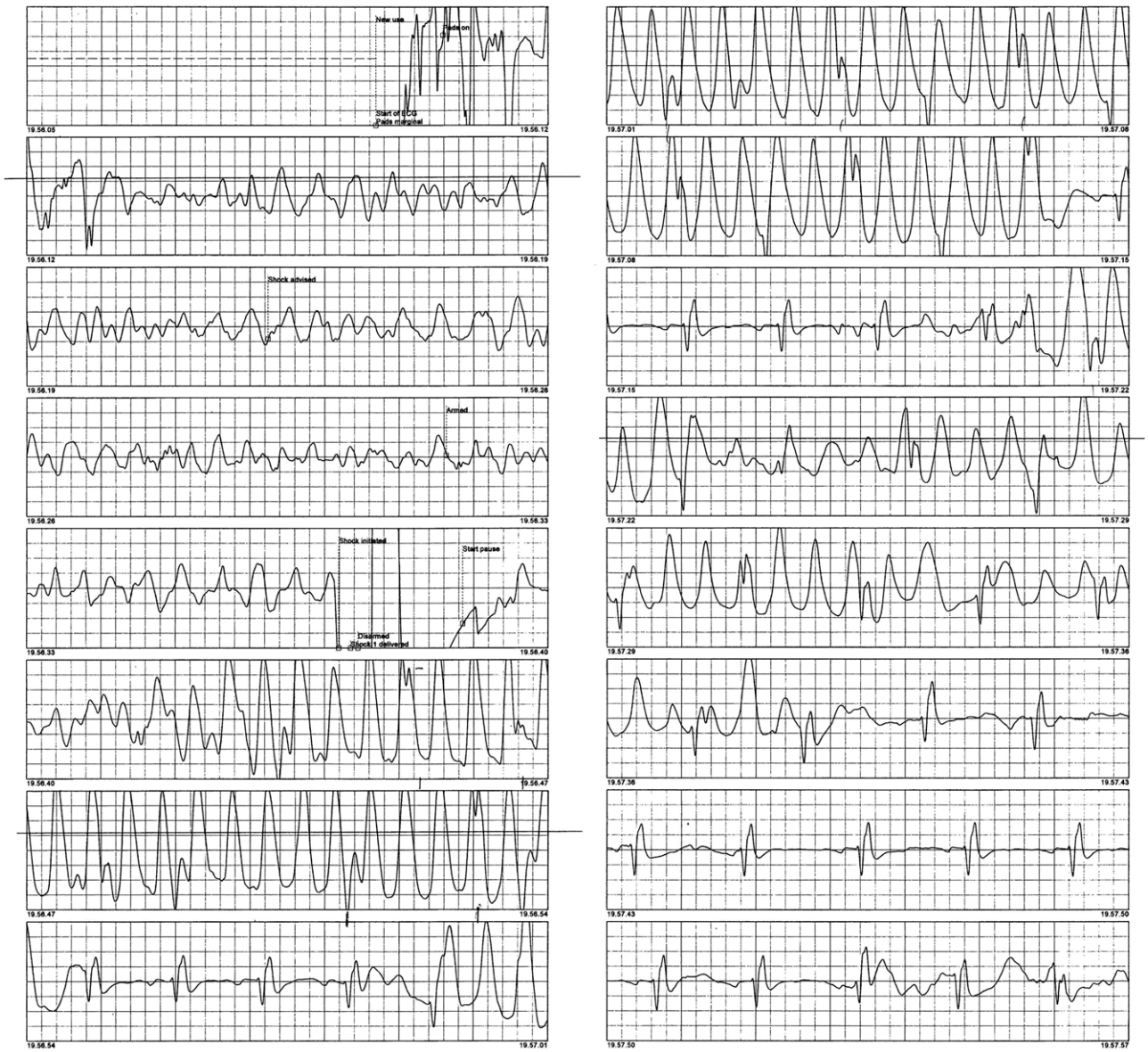

Fig. 1. ECG showing pulseless ventricular fibrillation reversed after defibrillation.

with cardiac arrhytmias, it is known that activity in the insula, cingulate cortex, amygdala, or hypothalamus, regulates cardiac function through connections to the brainstem and spinal cord nuclei.[8] In animal models, cerebral stimulation of the cingulate gyrus and temporal lobe can produce cardiac arrhytmias.[9]

Few cases of SUDEP or near-SUDEP have been recorded in EEG telemetry units, where mainly apnoea and hypoventilation were thought to be the primary cause of death. In some cases a notable finding was a terminal cessation or diffuse suppression of EEG activity before any cardiac or respiratory changes (CNS shut down), reflecting profound central inhibition and, possibly, explaining the subsequent central respiratory hypoventilation.[10] Few cases are reported with a primary cardiac mechanism.[5,6] Asystole and apnoea might promote each other through cardiorespiratory reflexes or cerebral and brainstem anoxia, but might also indicate a common dysfunction of central autonomic regulation and might be secondary to the profound central inhibition.[4]

Ictal bradycardia and ictal asystole have been documented in patients with severe focal epilepsy[1]: whether primary ictal asystole (i.e., without prior apnoea or malignant cardiac arrhythmia, such as ventricular fibrillation or torsade de pointes) can lead to SUDEP remains an open question. Nevertheless, in patients with evidence of marked cardiac arrhythmia associated with seizures, the possibility that this condition presents a risk would be sufficient in many cases to recommend on-demand cardiac pacing, which has been common practice, although in some isolated reports medical treatment alone can be a reasonable option.[11]

Despite any efforts, a satisfactory unitary explanation of the mechanism of SUDEP remains elusive. Near-SUDEP cases, especially those observed and recorded during video-EEG recording in epilepsy monitoring units, are especially helpful to document the sequence of events preceding the cardiorespiratory arrest. More cases are needed as they can contribute to our understanding of SUDEP mechanisms.

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