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Case report

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When seizures are not trivial!

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Abstract

We report the case of a 29 year old woman with post traumatic epilepsy which began at 20 year old. She presented multi-daily absence seizures, motor automatisms and headaches. Brain CT-scan performed, highlighted several punctiform hyperdense images like calcifications leading to the conclusion of intracerebral and subcutaneous bullet fragments. Her personal history reported head injuries at the age of 2 years in a context of unexplored and little followed armed conflict. Awake and sleep EEG found a well-organized EEG diagram with diffuse inter-ictal spikes and spike-waves discharges mainly in bi-fronto-temporal area compatible with focal epilepsy with secondary generalization. Valproic acid 1500 mg/day had been used and evolution at 3 months (April 2020) found no seizures increasing, sometimes headaches relieved by drugs.

Introduction

Traumatic Brain Injury (TBI) is a growing concern in the occurrence of late and repetitive seizures named Posttraumatic Epilepsy (PTE). Patients traumatized by blunt or penetrating military brain injury carry a high risk of intractable PTE which is a major health problem and account for 5% of all epilepsy patients [1]. We report an interesting clinical case of a young woman who developed PTE 18 years after penetrating military brain injury in the context of armed conflict.

Patient and observation

We report a medical observation of a 29 year old female patient, who presented in neurological consultation of Fann Teaching Hospital of Dakar, in January 2020 with history of seizures. We have obtained all appropriate patient consent forms. In the form the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understands that their name and initial will not be published and due efforts will be made to conceal their identity.

She is a young woman with first seizures occurred when she was 20 year old (2011) in a context of socio-family stress (her divorce). These seizures were like behavior disorders with agitation, shouting and flight. The patient took traditional drugs without success. No history of particular diseases or family seizures. Given the occurrence in 2013 of generalized seizures with tongue bite, sometimes loss of urine and post critical amnesia with a blank out event where she became unaware of what is going on around her, she consulted for the first time in a medical center where the first hypothesis of epileptic seizures is made. An EEG was requested but not performed. However, a treatment with Carbamazepine (CBZ) LP 400 mg, twice a day was prescribed without success, to which was added in 2014 phenobarbital (PHB) without stopping seizures. In 2017 the persistence of seizures led the patient to consult again and a brain MRI was requested. At the time of performing brain MRI, technicians interrupted the exam when they saw suspicious images of intracerebral metallic foreign matters. After discussion with the patient and her family, her mother reported that at the age of 2 year old (1993), she sustained head injuries in a context of armed conflict. She had been taken care of, but no major exams were done at that time.

A brain CT-scan was finally performed and highlighted several punctiform hyperdense images like calcifications leading to the conclusion of intracerebral and subcutaneous bullet fragments (Figure 1). Another EEG was asked but not performed again and CBZ have been stopped and replaced by valproic acid (VPA) chrono 500 mg per day with PHB 100mg per day, which would have allowed seizures spacing. A neurosurgical advice was asked and surgical abstention was in order. In January 2020, she came to neurological consultation where we saw her for the first time, because of a therapeutic break since 2019 in a context of lack of money, responsible for worsening breakthrough seizures with multi-daily absence seizures, motor automatisms and intense headaches. On examination patient was calm, conscious, alert, and afebrile with no evidence of

sensitivo-motor disturbances. No cranial nerves abnormality, no sign of meningeal irritation. Systemic examination was also normal. Awake and sleep EEG found a well-organized EEG diagram with diffuse inter ictal spikes and spike-waves discharges mostly over bi-fronto-temporal area compatible with focal epilepsy with secondary generalization (Figure 2). We began reducing and spacing the dosages of PHB in order to stop taking it. We informed the patient about risks associated with taking VPA and with her consent we kept it at the dose of 1500 mg/day for a weight of ≈ 132 lbs. For headaches, we prescribed a paracetamol-ibuprofen combination. A psychological follow-up has been started. Evolution at 3 months (April 2020) reported no seizures increasing. She has sometimes headaches relieved by medication.

Discussion

Seizures can occur or not after a traumatic brain injury (TBI), whatever the cause. These causes vary considerably between motor accidents and gunshot wounds or explosions [2]. Seizures after TBI can be classified as early post-traumatic seizures occurring in the first week after a brain injury and as late post-traumatic seizures occurring more than seven days after a brain injury [3]. PTE is diagnosed in the presence of recurrent late seizures [4]. According to literature, among survivors of head injuries, those with penetrating brain injuries have the highest risk of developing PTE, and foreign fragments in the brain including metal, are associated with the highest risk [5]. Our patient developed late post-traumatic epilepsy, 18 years after head injuries, the mean time since injury to develop PTE in penetrating trauma in the study of KAZEMI concerning 163 war veterans with post-traumatic intractable epilepsy being 20 years (SD 5.4) [6]. Many studies found that predictive factors associated with epilepsy risk after TBI are missile wound with dural penetration, parietal lobe trauma, single temporal or frontal lesions revealed in brain CT, occurrence of early seizure, presence of an intracerebral hematoma, post-traumatic amnesia, brain tissue loss and retained metal fragments [6, 7]. Also, it has been highlighted in case of retained fragments of bullets which typically consist of copper coverings over a lead core, that copper in the brain induces a significant inflammatory reaction which participate to the development of chronic seizures [5,8].

However, it is not known if the retain fragments have the single role in epileptogenesis or whether the presence of others markers constitute or increase the risk factors to develop PTE [2,5]. Devyn Cotter and collaborators in a systematic review concluded that beyond the process of TBI, there is a genetic element to the development of PTE but understudied [2]. Headaches seem to be a frequent complaint for patients with TBI, which varies from mixed vascular, muscle tension type or typical migraine [9]. The real problem lies in the management of these post-traumatic seizures. The use of MRI is contra-indicated in the case of intracranial bullet fragments, therefore increases the risk to miss out on additional encephalic lesions. Literatures did not find differences in seizure frequency between patients with no surgical intervention versus patients who underwent an operation and no association between location of the fragments and ictal semiology of the patients [6]. These well corroborate the non-concordance between the frontal and temporal left cortical localization of bullet fragments for our patient and the bi fronto-temporal inter ictal EEG abnormalities. According to American Association of Neurological Surgeons (AANS) standard of care, prophylactic use of conventional Antiepileptic Drugs (AEDs) (phenytoin, CBZ or PHB) is not recommended for preventing late posttraumatic seizure [3]. The prophylactic use of AEDs after TBI should be brief and limited to the prevention of immediate and early seizures [10]. Many studies, using new generation AEDs (Topiramate, Gabapentin and Levetiracetam), found their neuroprotective effect and suggest their prophylactic use for PTE in TBI. As specified by these studies, PTE patients treated with these new generation AEDs achieved remission considerably earlier than those we received conventional AEDs [10]. Unfortunately,

the availability and/or cost of these new generation AEDs do not allow better therapeutic compliance in our medical context. We are, therefore, led to use more accessible and efficient AEDs like VPA for better management of these late chronic seizures.

Conclusion

Certain type of TBI put individuals at higher risk for late posttraumatic seizure. Retained metal fragments increase seizure risk. PTE may occur in TBI, more when associated with other risk factors of epilepsy, mainly genetics. New generation AEDs give hope in the prevention and treatment of these more disabling post-traumatic epilepsies.

Competing interests

The authors declare no competing interests.

Authors' contributions

Prisca-Rolande Bassole and Mendinatou Agbetou worked for acquisition, analysis and interpretation of data and drafted the article. Maouly Fall and Marième Soda Diop-Sene revisited the article. Anna Modji Basse-Faye, Adjaratou Dieynabou Sow and Lala Bouna Seck analyzed and interpreted electrophysiological data. Moustapha Ndiaye, Amadou Gallo Diop and Mouhamadou Mansour Ndiaye gave final approval of the version to be published. All the authors have read and agreed to the final manuscript.

Figures

Figure 1: brain CT-scan: left frontal, temporal, cerebellum tent and subcutaneous punctiform hyperdensity

Figure 2: high filter: 0,53Hz, low filter: 70Hz, longitudinal EEG editing diffuse inter ictal spikes and spike-waves discharges mostly over bi-fronto-temporal area

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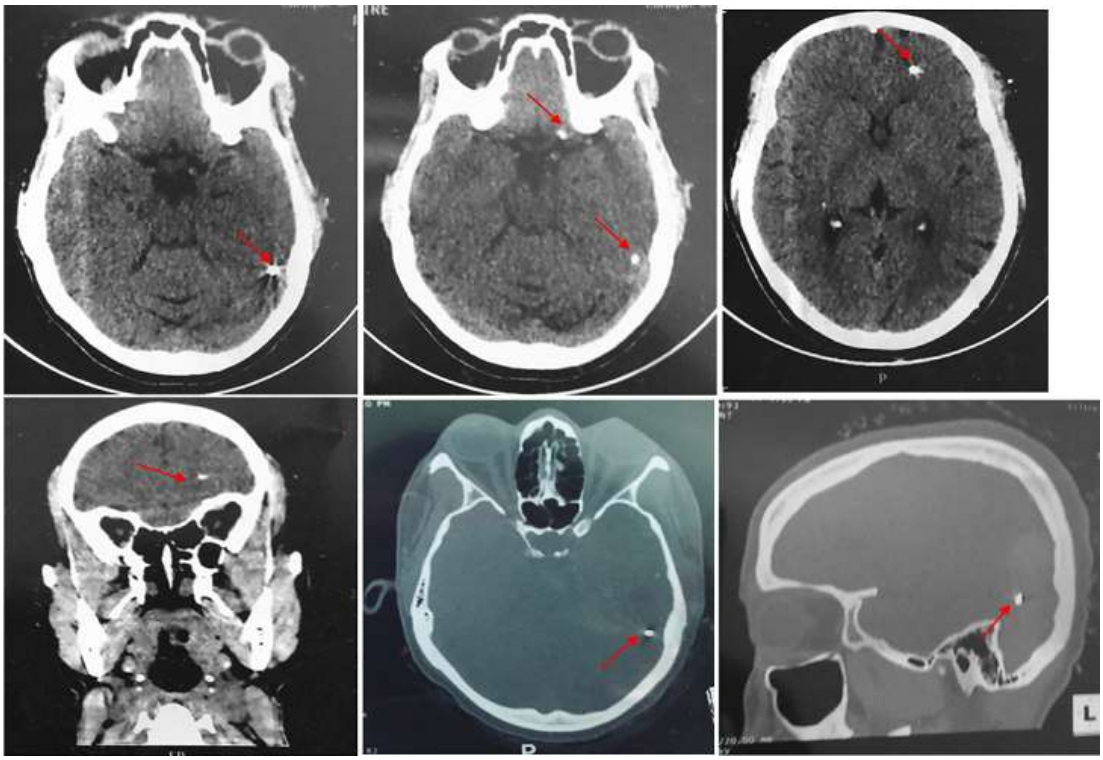

Figure 1: brain CT-Scan: left frontal, temporal, cerebellum tent and subcutaneous punctiform hyperdensity

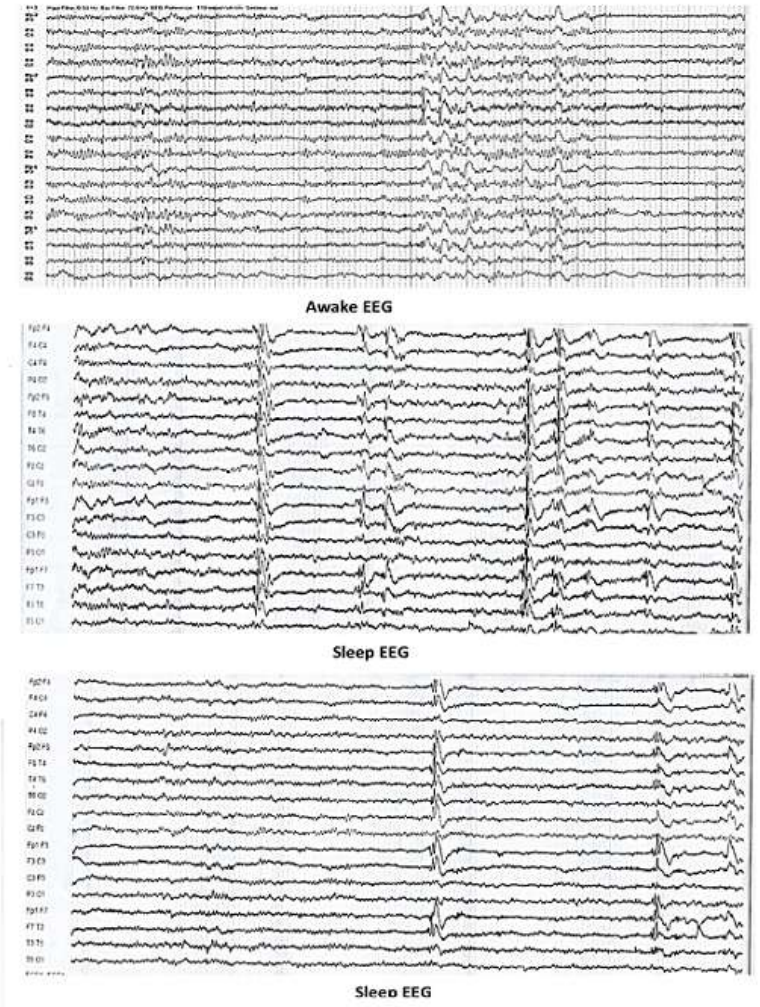

Figure 2: high filter: 0,53Hz, low filter: 70Hz, longitudinal EEG editing diffuse inter ictal spikes and spike-waves discharges mostly over bi-fronto-temporal area