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2173-5808/

Epileptic seizure as a trigger of acute coronary syndrome☆,☆☆

Crisis epiléptica como desencadenante del síndrome coronario

Dear Editor,

Epilepsy has an impact not only on the nervous system but also on a long list of organs and vital systems, including the cardiovascular system. Several studies have investigated, and clearly identified, effects of epilepsy on the heart. The earliest and best-known effect of epilepsy on the cardiovascular system is the increase in heart rate due to adrenaline release and increased circulatory demand; the latter is especially evident in tonic-clonic seizures. In addition to increased heart rate, other phenomena may also be present including myocardial ischaemia, stress cardiomyopathy, and conduction disorders.

We present 2 clinical cases of acute coronary syndrome triggered by epileptic seizures and provide a brief review of current literature addressing the effects of epilepsy on the heart.

Patient 1

Our first patient was 81-year-old woman with a history of arterial hypertension, ischaemic stroke, and atrial fibrillation who was partially dependent for activities of daily living. She visited the emergency department due to a generalised tonic-clonic seizure which resolved spontaneously. The electrocardiogram revealed ST segment elevation in leads II, III, and aVF. The patient also displayed elevated troponin levels (1.89 ng/mL). These findings were compatible with acute coronary syndrome, which was successfully controlled with medication. The patient experienced no associated complications. During hospitalisation, she experienced an episode of disorientation and a prolonged drop in consciousness. A brain CT scan revealed encephalomalacia in the left parietal region, which was a sequela of a previous ischaemic stroke, and no other alterations suggesting ischaemia and/or haemorrhage. An electroencephalography (EEG) conducted during a seizure revealed markedly slow background activity featuring theta-delta waves and rhythmic spike-and-wave discharges at 3 Hz in the left frontotemporal region. In view of these findings, we administered intravenous diazepam, which controlled symptoms and achieved a normal EEG tracing. Our patient was subsequently treated with levetiracetam dosed at 2000 mg/day, and experienced no further seizures. She was discharged a few days later with a diagnosis of acute coronary syndrome secondary to a partial epileptic seizure with secondary generalisation.

Patient 2

Our second patient was 86-year-old man with a good quality of life who was independent for activities of daily living, had no cardiovascular risk factors, and had a history of colonic polypectomy and parathyroid adenoma. He was attended in the emergency department due to loss of consciousness and amnesia. He was accompanied by his son, who reported that the patient had difficulty understanding instructions and spoke incoherently, although the latter symptom improved gradually. His vital signs were recorded by the emergency services and included slight increases in heart rate and arterial pressure. A cranial CT scan revealed no alterations. While undergoing the CT scan, the patient reported nonspecific pain under the right scapula. An electrocardiogram

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revealed sinus tachycardia and ST segment elevation in leads II, III, aVF, V2, V3, and V4. CPK-MB and troponin levels were 10 U/L and 0.14 ng/mL, respectively. A subsequent blood test revealed significant increases in these levels (27 U/L and 1.66 ng/mL, respectively). The patient was diagnosed with acute coronary syndrome and received pharmacological treatment, which decreased CPK-MB and troponin levels, improved EEG tracing, and minimised symptoms. When coronary symptoms had stabilised, our patient experienced 2 self-limiting episodes of aphasia, predominantly motor, and a slight increase in heart rate and arterial pressure. An EEG performed between seizure episodes revealed intermittent rhythmic delta activity in the left temporal region and periodic lateralised epileptiform discharges in the frontal region. The patient initiated treatment with levetiracetam dosed at 2000 mg/day and experienced no further seizures. He was discharged a few days later with a diagnosis of acute coronary syndrome secondary to a partial complex seizure.

The central nervous system has networks connecting the insular region, the central nucleus of the amygdala, and the hypothalamus with frontal and mesial temporal areas. These neural networks are linked to autonomic control[1]; in fact, the insular and orbitofrontal cortices in particular are considered to represent the autonomic nervous system at the cortical level.[2] The amygdala and the piriform cortex modulate hypothalamic functions; stimulating these regions may cause both sympathetic and parasympathetic autonomic visceromotor responses.[3] Likewise, stimulation of the orbitofrontal cortex and cingulate gyrus changes cardiac and respiratory frequencies.[4,5] Autonomic control tends to be lateralised: the right hemisphere may have a greater sympathetic influence, whereas the left hemisphere may be linked to greater parasympathetic control.[6] This tendency towards lateralisation depends on the type of seizure, the localisation of the epileptogenic focus, and the aetiology. In a recent study, patients with right temporal lobe epilepsy displayed an increase in heart rate before symptom onset and coinciding with onset of EEG changes, in contrast, patients with left temporal lobe epilepsy showed increased heart rate after EEG changes. The authors of this study suggested the term 'preceding tachycardia sign' to refer to this increase in heart rate in right temporal lobe epilepsy.[7] In general, patients with refractory temporal lobe epilepsy are more likely to experience alterations in autonomic cardiovascular control regardless of lateralisation of the epileptic focus.[8]

In epileptic patients, the massive catecholamine release caused by epileptic seizures may result in myocardial ischaemic events due to vasoconstriction of the coronary arteries; this effect is more marked when plaque builds up in these arteries.

Tachyarrhythmias caused by epileptic seizures may be explained by the propagation of epileptic discharges to the right insular cortex. Thus, tachycardia is more frequent and long-lasting in patients with temporal lobe epilepsy since propagation of electric discharges is also easier and more lasting than in the case of extratemporal epilepsy.

Bradycardia and asystole are less frequent. It has been hypothesised that the frontal and left insular cortices and the amygdala are involved in epileptic discharge; this is supported by studies reporting atrioventricular block after stimulation of the left basal temporal lobe.[9]

Lasting bradyarrhythmia and tachyarrhythmia may cause myocardial ischaemia. A study found myocardial fibrosis in patients who died suddenly[10] and another study reported elevated plasma levels of CK-MB and BNP in epileptic patients.[11] Determining whether a syncope is of cardiac or neurological origin (the latter represents less than 5% of the cases) is challenging.[12] This being the case, we should be mindful of the influence of the cerebral cortex, hypothalamus, amygdala, and periacueductal grey matter on baroreceptor and chemoreceptor regulation: impairment in any of these areas may affect autonomic cardiovascular control, leading to imbalances and their subsequent clinical manifestations. On the one hand, cardiovascular events triggered by epileptic seizures may cause syncope; on the other, some epileptic seizures affecting the pontine reticular formation may result in decreased consciousness[13] and consequently syncope. Therefore, syncope would be a direct manifestation of seizures in the second case, whereas in the first case it would secondary.

In summary, our clinical cases support the hypothesis that epileptic seizures, and especially generalised tonic-clonic seizures, indicate a predisposition to acute cardiovascular disease, which points to an additional vital risk in patients with epilepsy.

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Influenza A virus: A possible trigger factor for hypnic headache?☆,☆☆

¿Virus de la gripe A como factor desencadenante de una cefalea hípnica?

Dear Editor:

Hypnic headache (HH), a primary headache first described by Raskin[1] in 1988, is listed in group 4 of the third edition of the International Classification of Headache Disorders, beta version (ICHD-3 beta), which was published in 2013.[2] HH usually begins after the age of 50 and presents as recurrent oppressive headache in most cases. This type of headache presents only during sleep, waking the patient ('alarm clock' headache) and lasting for up to 4 hours, without any characteristic associated symptoms. To diagnose HH, other possible causes of nocturnal headache must first be ruled out, with special attention to sleep apnoea/hypopnoea syndrome (SAHS), nocturnal hypertension, hypoglycaemia, and medication overuse. However, presence of SAHS does not rule out a diagnosis of HH.

The ICHD-3 beta differentiates between hypnic headache (Table 1) and probable hypnic headache (Table 2).

We present the case of a 41-year-old woman with a history of essential hypertension, type 2 diabetes mellitus, class II obesity, migraine without aura, and obesity-hypoventilation syndrome (OHS). She had been hospitalised due to a viral respiratory infection with influenza A; during her hospital stay she only required nasal cannula oxygen therapy in the first few days and experienced a mild headache that resolved as symptoms improved. In the month following discharge, our patient was referred to the headache unit due to episodes of headache appearing only at night (usually around 2.00-3.00 a.m.), with a frequency of 2-4 episodes. Headache was either hemicranial (affecting both sides) or holocranial, pulsating and stabbing, and of moderate intensity. Episodes lasted a mean of 15 minutes and made her get out of bed to engage in motor tasks until symptoms subsided completely. She displayed no other

Table 1 ICHD-3 beta diagnostic criteria for hypnic headache.

A	Recurrent headache attacks fulfilling criteria B-E
B	Developing only during sleep and causing wakening
C	Occurring on ≥10 days per month for >3 months
D	Lasting ≥15 minutes and for up to 4 hours after waking
E	No cranial autonomic symptoms or restlessness
F	Not better explained by another ICHD-3 diagnosis

Table 2 ICHD-3 beta diagnostic criteria for probable hypnic headache.

A	Recurrent headache attacks fulfilling criterion B and 2 of criteria C-E
B	Developing only during sleep and causing wakening
C	Occurring on ≥10 days per month for >3 months
D	Lasting ≥15 minutes and for up to 4 hours after waking
E	No cranial autonomic symptoms or restlessness
F	Not fulfilling ICHD-3 criteria for any other headache disorder
G	Not better explained by another ICHD-3 diagnosis

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