Coma súbito como forma de presentación de la Endocarditis Infecciosa

Sudden coma as a presentation of Infective Endocarditis

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ABSTRACT

This case reports an uncommon presentation of Infectious endocarditis following a late hip periprosthetic infection. Patient presented with sudden coma with no abnormal head CT images. Nonconvulsive status epilepticus and central nervous system infection were excluded as well as significant metabolic, electrolyte or hormonal causes of neurological disfunction. Brain MRI revealed multiple recent small ischemic lesions suggestive of an embolic source. Serial blood cultures, under antibiotics, were negative but transoesophageal echocardiogram revealed a tricuspid valve vegetation along with a thin and mobile atrial septum where patency of the oval foramen could not be ruled out. A diagnosis of infectious endocarditis was established. A sudden coma with no CT changes is, to the best of the authors' knowledge and experience, a rare and previously undescribed presentation. This raises awareness that the diagnosis of brain septic emboli should be considered in the presence of sudden coma without other apparent causes.

Keywords: Infectious endocarditis, sudden coma, brain embolization.

RESUMEN

Este caso describe una presentación infrecuente de endocarditis infecciosa tras una infección periprotésica tardía de cadera. El paciente presentó un coma repentino sin imágenes anormales en la TC craneal. Se excluyeron el estado epiléptico no convulsivo y la infección del sistema nervioso central, así como causas metabólicas, electrolíticas u hormonales significativas de disfunción neurológica. La resonancia magnética cerebral reveló múltiples lesiones isquémicas recientes de pequeño tamaño sugestivas de un origen embólico. Los hemocultivos seriados, bajo antibióticos, fueron negativos, pero el ecocardiograma transoesofágico reveló una vegetación de la válvula tricúspide junto con un tabique auricular delgado y móvil en el que no podía descartarse la permeabilidad del foramen oval. Se estableció el diagnóstico de endocarditis infecciosa. Un coma repentino sin cambios en la TC es, según el conocimiento y la experiencia de los autores, una presentación rara y no descrita previamente. Esto hace pensar que debe considerarse el diagnóstico de émbolo séptico cerebral en presencia de coma súbito sin otras causas aparentes.

Palabras clave: Endocarditis infecciosa, coma súbito, embolización cerebral.

CLINICAL CASE

A 76-year-old, previously autonomous woman, with a history of degenerative arthritis of the hip submitted to left hip replacement surgery was admitted to an orthopaedics inpatient unit for late hip periprosthetic infection, having undergone cleaning and component exchange. *Streptococcus constellatus* was identified in the synovial fluid and ceftriaxone was started. After 23 days, however, due to poor clinical evolution, maintaining purulent drainage of the surgical wound, antibiotics were changed. Patient was started on piperacillin/tazobactam and vancomycin and underwent full removal of prosthetic material. The patient returned after surgery to the ward and was last described in her usual talkative manner, in good mood, with no neurological deficits. During that same night, a fever started with no other changes noted in her medical condition but early in the morning, less than 24 hours after surgery, the patient became unresponsive.

Neurological examination revealed a comatose state. The patient had no verbal response, no opening of eyes and no movement of the arms or legs with physical stimulation, compatible with a Glasgow Coma Score of 3 (E1V1M1), gaze deviation to the left with occasional nystagmus eye movements and bilateral extensor plantar reflex. Despite the severity of the neurological changes, the patient remained hemodynamically stable and without respiratory failure. On further evaluation, a fever of 38ºC was present with normal lung and heart auscultation, no audible murmurs and normal abdominal examination.

Immediate brain CT and CT angiography were performed with no visible signs of acute ischemic or haemorrhagic lesions or large vessel occlusion.

Nonconvulsive status epilepticus was initially considered but electroencephalogram analysis revealed a pattern of moderate to severe diffuse encephalopathy, with no epileptiform activity and no focal or regional slowing. Nevertheless, antiseizure medication was started with no change in neurological status.

Lumbar puncture was performed with no signs suggestive of central nervous system infection. Blood analysis revealed moderate anaemia (10,3 g/dL) and elevated C reactive protein (10,4 mg/dL). Leucocyte and platelet counts were normal as well as kidney and thyroid function. No acid base disorders or significant electrolyte imbalances were present. Serial blood cultures were negative.

A brain MRI was ordered that showed multiple millimetre foci of hypersignal T2 FLAIR, most with corresponding water molecules diffusion restriction, without evident enhancement after gadolinium administration, involving multiple brain areas bilaterally, supra and infratentorial, in cortico-subcortical topography but also in the deep white matter, thalamus, splenium of the corpus callosum, the cerebellous hemisphere on the left and the middle cerebellous peduncle on the right (Figure 1).

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Cómo citar este artículo: Magalhães P, Veríssimo R.
Sudden coma as a presentation of Infective Endocarditis. Galicia Clin 2024; 85-1: 25-27.
Recibido: 30/12/2022 ; Aceptado: 21/02/2023 // https://doi.org/10.22546/72/4041

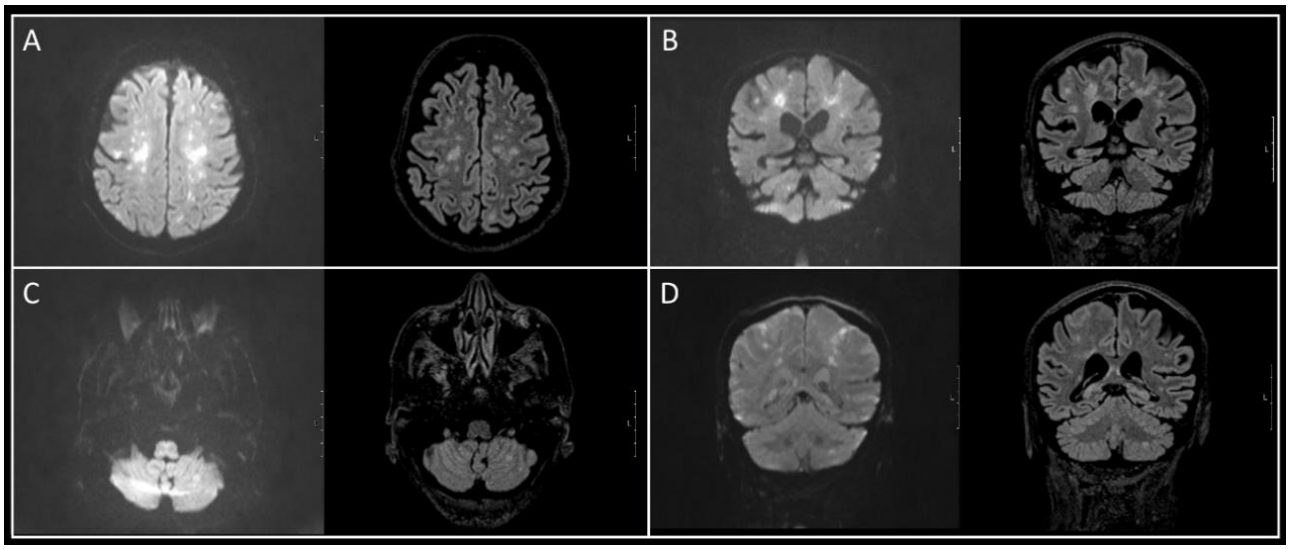

Figure 1. Brain MRI scans (in pairs, DWI to the left and T2-Flair on the right)

The presence of multiple recent small ischemic lesions in various arterial territories (anterior and posterior circulation bilaterally), suggested a proximal embolic source. Despite the absence of heart murmurs or heart failure symptoms, due to presence of fever and a known infectious source, the hypothesis of infective endocarditis (IE) as the origin of emboli was placed. The possibility of a fat embolism syndrome was also considered. This condition, frequently associated with traumatic long bone injuries, usually presents as respiratory symptoms with a typical 24-to-72-hour delay from the injury. Nevertheless, cases have been described after orthopaedic surgery, as well as rare cases of fulminant presentation and predominant cerebral embolization[1–3]. To differentiate between these two entities a transoesophageal echocardiogram was performed. This revealed a typical image of vegetation, with a mobile mass in the tricuspid valve at the level of the auricular face of the posterior leaflet, attached to the leaflet base, maximum size of 14 mm (Figure 2). The atrial septum was lipomatous, thin and mobile at the level of the oval fossa and patency of the oval foramen could not be ruled out. Thoracic CT scan showed no clear signs of vessel occlusion but areas of attenuation with a mosaic texture were present, suggesting ventilation/perfusion mismatch. No other embolic infarcts were found, including an absence of cutaneous abnormalities and no abdominal organ embolization was apparent on abdomen CT angiography. These findings alongside fever and vascular embolic phenomena, correspond to a possible diagnosis of IE (1 major and 2 minor Duke criteria).[4]

Cardiac surgery was consulted but valve repair surgery was deemed not feasible. Unfortunately, despite a slight improvement in neurological condition with spontaneous eye opening, the patient remained incapable of verbalization, understanding orders, deglutition, and motion of the legs and arms. Antibiotics were suspended, palliative care was consulted and at the time of writing this paper, patient was in end-of-life care.

DISCUSSION

Cerebral territory emboli in IE can occur in the presence of right-left heart shunts, which could not be excluded in this case.[5] Blood cultures were persistently negative, but the patient was already on antibiotics with the isolated *S. constellatus* identified only in the synovial fluid of the initial hip infection and removed material.

Neurological disease is a relatively common complication of IE manifesting in multiple forms, such as stroke, mycotic aneurysm, brain abscess or cerebral haemorrhage[6]. There is significant clinical variability, however presentation as a sudden coma with no visible changes in brain CT and CT angiography is, to the best of the authors' knowledge and experience, a rare and previously undescribed occurrence. This case report means to raise awareness to the possibility of this atypical and potentially life-threatening complication of IE, and the diagnosis of brain septic emboli should be considered in the presence of sudden coma without other apparent causes.

ETHICAL CONSIDERATIONS

The authors declare that the procedures were followed according to the regulations established by the Clinical Re-search and Ethics Committee and to the Helsinki Declaration of the World Medical Association updated in 2013.

The authors declare having followed the protocols in use at their working center regarding patients' data publication.

CONFLICTS OF INTEREST

The authors have no conflicts of interest to declare.

FINANCIAL SUPPORT

None declared.

INFORMED CONSENT

Written patient consent was obtained.

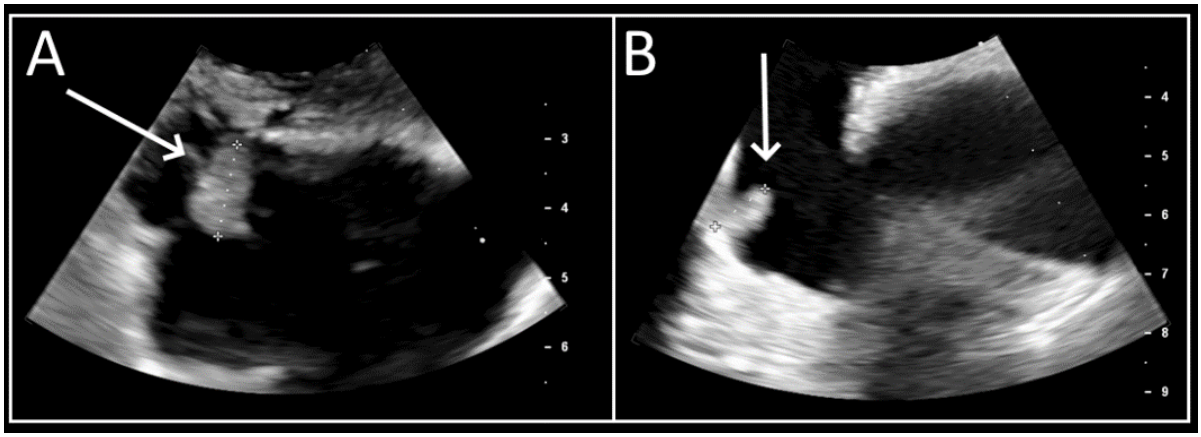

Figure 2. Transoesophageal echocardiogram showing tricuspid valve vegetation.

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