



Advanced interatrial block as a predictor of cardioembolic stroke: is it time to change our clinical practice?*

Bloqueo interauricular avanzado como predictor de ictus cardioembólico: ¿es hora de cambiar nuestra práctica clínica?

Dear Editor:

Atrial fibrillation (AF), the most common sustained arrhythmia, is estimated to affect 5% of individuals older than

65 years of age.¹ AF is frequently asymptomatic, and 15% of strokes are associated with untreated AF.² This has led researchers to search for potential predictors of AF and of the risk of embolism. We present a case of ischaemic stroke in the context of paroxysmal AF.

The patient was a 73-year-old woman with arterial hypertension and type 2 diabetes mellitus, who was independent for daily living activities and was under follow-up for dyspnoea. Electrocardiography (ECG) revealed sinus rhythm and P-wave duration > 120 ms with biphasic morphology (+/−) in the inferior leads. Echocardiography revealed normal left ventricular ejection fraction, moderate left atrial enlargement (43 mL/m^2), and a significantly decreased peak atrial longitudinal strain (13.5%) (Fig. 1). The patient was diagnosed with heart failure with preserved ejection fraction, and her symptoms improved with diuretic treatment. Sev-



Figure 1 Baseline electrocardiography study. (A) Signs compatible with advanced interatrial block. (B) Left atrial enlargement and decreased peak atrial longitudinal strain.

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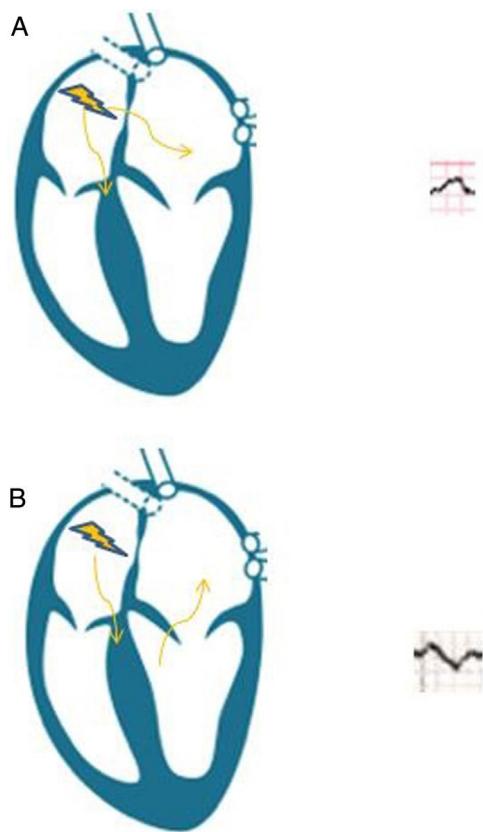


Figure 2 Atrial activation in healthy individuals (A) and in patients with interatrial block (B).

eral months later, she visited the emergency department due to predominantly sensory mixed aphasia and right central facial paresis of 6 hours' duration. She scored 6 on the NIHSS at arrival. A CT scan revealed established ischaemic stroke in the territory of the middle cerebral artery, and ECG detected AF. A Doppler ultrasound of the supra-aortic trunks revealed atherosomatous plaque, with no haemodynamic involvement. The patient was admitted to our department, where we started heart rate monitoring and administered anticoagulation treatment, after ruling out haemorrhagic transformation in a follow-up CT scan. She was discharged with a diagnosis of cardioembolic stroke.

Our patient presented advanced interatrial block (AIB), and subsequently developed AF and stroke. AIB is an ECG pattern first described by Bayés de Luna³ in the late 1970s, and is characterised by conduction delay through the Bachmann bundle, the main conduction pathway between the 2 atria (Fig. 2). Partial interatrial block is more common, and is characterised exclusively by prolonged P-wave duration (> 120 ms) in the inferior leads. AIB involves retrograde activation of the left atrium from the coronary sinus, which results in abnormal electrical activity (P-wave duration > 120 ms, associated with a negative terminal component of the P-wave in inferior leads).⁴ The association between AIB and paroxysmal AF is known as Bayés

syndrome.⁵ Atrial fibrosis and atrial dyssynchrony constitute the anatomical substrate for AIB and AF, promoting systemic embolism, although atypical forms have also been described in patients with diabetes or chronic kidney disease, with no impact on the risk of AF or stroke.⁶ The concept of atrial failure has been proposed to underscore the association between this entity and thromboembolic events, with AF being considered a marker of atrial dysfunction, rather than a direct emboligenic mechanism.⁷ Speckle-tracking echocardiography is a novel technique enabling the assessment of atrial fibrosis in order to predict emboligenic events.⁸ Although no clinical trial performed to date supports the use of anticoagulants in patients with AIB but not AF, a thorough ECG study is essential. Detecting AIB allows us to identify patients at high risk of embolism who may benefit from prolonged monitoring (external loop recorder, implantable Holter monitor, portable mHealth system); this strategy may in future contribute to deciding whether anticoagulant or antiarrhythmic treatment is needed.⁴

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Rapidly progressive dementia with generalized myoclonus in an adult: Do not forget subacute sclerosing panencephalitis



Demencia rápidamente progresiva con mioclonías generalizadas en un adulto: no olvides la panencefalitis esclerosante subaguda

Dear Editor,

Persistence of measles infection in children and young adults can rarely lead to a fatal progressive neurodegenerative disorder known as subacute sclerosing panencephalitis (SSPE).^{1–4} Amongst the other known neurological manifestations of measles infection, SSPE is the rarest and most severe.^{1–4} However, measles infection is amenable to prevention by highly effective live vaccination.^{1–5} The latency period between measles infection and development of clinical features of SSPE is variable and can be up to 2–3 decades.^{1–4} Earlier affection and intrafamilial cases of SSPE have a shorter latency.^{1–4} The clinical course is characterized by subacute to chronic progressive cognitive impairment, behavioral abnormalities, movement disorders (especially myoclonus), seizures, visual disturbances, and ataxia.^{1–3} Although few reports of spontaneous remissions of SSPE have been documented,^{6,7} this disease is considered universally fatal. Diagnosis of SSPE is difficult in developed countries because of extreme rarity of the condition and atypical and non-specific initial presentation because the sufferers come rather early in the course of disease.^{1–3,8} On the contrary, in underdeveloped/developing countries, the delay in diagnosis occurs due to low health-related literacy in general population, significant lack of knowledge and training of primary care physicians regarding these disorders, inadequate health-seeking behavior of the general population, and inadequate investigational backup in peripheral health care setups.^{1–3,8}

We herein report a case of SSPE in a 36-year-old previously healthy man with questionable vaccination status in childhood who presented with rapidly progressive early onset dementia and subcortical myoclonus. Neuroimaging revealed altered signals involving bilateral thalamus

and brainstem widening the list of differential diagnoses; however, clinical history, examination findings, and high anti-measles IgG titers in serum and cerebrospinal fluid (CSF) clinched the diagnosis, which was supported by classical Radermecker's complex in electroencephalography (EEG).⁹

A 36-year-old previously healthy man from a middle socio-economic family of rural West Bengal (India) was brought to the out-patient department with complaints of rapidly progressive behavioral abnormalities (inattentiveness, forgetfulness, mood disturbances, aggressive behavior, lack of interest in surroundings, calculation difficulties, difficulties in recognizing relatives and common objects, and loosing ways during navigation) and visual difficulties for last 3–4 months. The family members also gave history of objects falling from his hands unknowingly, and gradually evolving abnormal involuntary jerky "shock-like" movements involving all the limbs and torso for the last 2 months. Past medical history and history of long-term drug exposure or addiction in any form were unyielding. The family members revealed that one of his brothers also had succumbed to a similar illness within 2 years of developing similar symptoms at the age of 18 years. On enquiry, his mother described an episode of "fever with confluent rash" with spontaneous recovery in all her children, in their childhood, over a period of 2 weeks suggestive of measles. Neurological examination revealed multi-domain cognitive impairments with subcortical myoclonus.

Brain magnetic resonance imaging (MRI) showed non-enhancing bilaterally symmetrical altered intensity lesions, hyperintense on T2-weighted imaging and FLAIR sequences, involving thalamus and brainstem (Fig. 1). EEG revealed periodic discharges suggestive of Radermecker complexes (Fig. 2). Contrast enhanced MR angiography and MR venography excluded common vascular insults. Accordingly, relevant tests were done, which excluded metabolic disorders (blood glucose, thyroid, liver and renal function tests, electrolytes, vitamin B1 and B12, arterial blood gas analysis, serum lactate, CSF lactate, lactate/pyruvate ratio, and anti-thyroxine/thyroglobulin antibody were normal), toxic encephalopathy (toxicology screen), acute infectious encephalopathies (neuro-viruses panel, including SARS-CoV-2 and HIV 1, 2, were negative). Autoimmune/paraneoplastic encephalitis panel was negative. CSF study revealed lymphocyte-predominant pleocytosis (cell count: 25/ μ L), increased total protein (108 mg/dL), and raised IgG index.