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LETTER TO THE EDITOR

Interatrial block and stroke: Waiting for evidence to decide anticoagulation☆

Bloqueo interauricular avanzado y accidente cerebrovascular: esperando la evidencia para decidir anticoagulación

Dear Editor:

We read with great interest the article by López Perales et al.,[1] who present a masterful description of a clinical case to address a long-standing debate in cardiology: should anticoagulation be administered to patients with advanced interatrial block (IAB) but no clinical evidence of atrial fibrillation (AF)?

IAB is a disorder of conduction between both atria caused by replacement fibrosis in the Bachmann bundle.[2] It has traditionally been classified as partial, advanced, or intermittent,[3] although a new category, atypical advanced IAB, has recently been included.[4] Several studies have shown an association between advanced IAB and subsequent development of AF, which is known as Bayés syndrome.[2] However, recent evidence suggests that the presence of IAB (and particularly advanced IAB) may increase the risk of cardioembolic events, mainly stroke.[5] Carrillo-Loza et al.[6] showed that presence of IAB predicts stroke recurrence in patients with history of embolic stroke of undetermined source, revealing the following independent risk factors for stroke: advanced AIB ($P < .001$), male sex ($P = .028$), and age over 50 years ($P = .039$).[6]

Several mechanisms have been reported to play a role in the pathophysiology of thromboembolic events secondary to IAB, including atrial dyssynchrony, endothelial damage, and atrial fibrosis, all of which appear sequentially and promote activation of the clotting cascade (Fig. 1).[7] Atrial fibrosis, an important marker of atrial dysfunction, may be studied with such techniques as two-dimensional speckle tracking echocardiography, a non-Doppler technique enabling quantification of atrial deformation based on standard two-dimensional images and the measurement of longitudinal strain (ε, expressed as a percentage) and longitudinal strain rate (SR, expressed as the number of deformations per second) of the left atrium during a cardiac cycle.[8] This happens

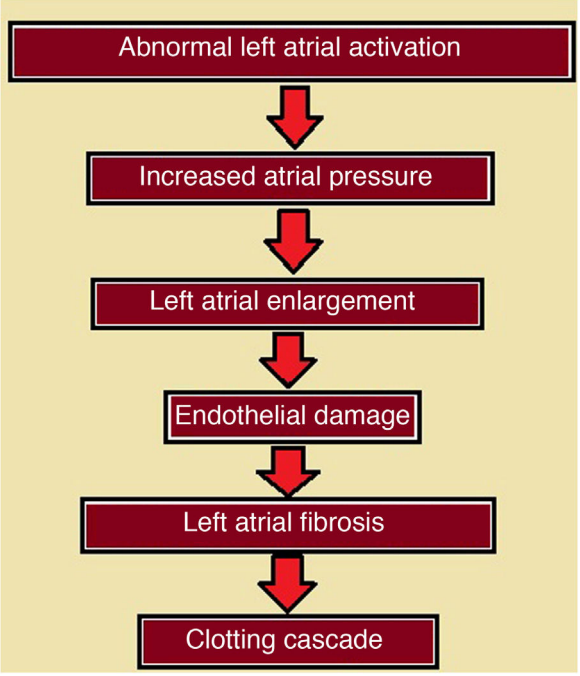

Figure 1 Schematic representation of the sequential process by which presence of interatrial block may trigger a series of phenomena leading to the activation of the clotting cascade and consequently the development of thromboembolic events. Modified from Martínez Sellés et al.[7]

to have been the technique used by López Perales et al.[1] in their excellent report.

The BAYES registry, a recent multicentre study conducted between 2017 and 2020, found IAB to be a powerful predictor of AF and stroke in non-hospitalised older patients with underlying structural heart disease. The study included data from 556 patients, who were classified into 3 groups (group A, P wave < 120 ms; group B, partial IAB; group C, advanced IAB). Patients were followed up for a mean of 694 days: 16.7% developed AF, 5.4% presented stroke, and 6.1% died during follow-up. The incidence of AF in groups A, B, and C was 24%, 29%, and 40%, respectively, and the incidence of stroke was 9%, 9%, and 12%. After performing univariate and multivariate analyses, the authors concluded that advanced IAB was an independent predictor of AF and stroke, and that P-wave duration showed a linear correlation with increases in the incidence of AF, stroke, and mortality (Fig. 2).[9]

Despite the available evidence, the answer to the question posed by López Perales et al.[1] of whether oral coagulation should be administered in patients with no evidence of AF is ''no,'' or at least ''not for the time being.'' Two studies on the topic are currently underway:

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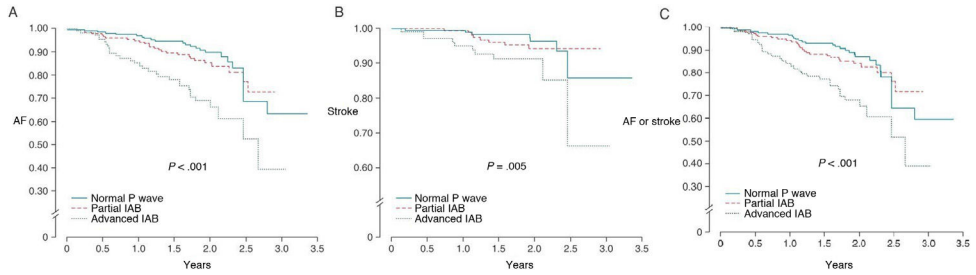

Figure 2 Kaplan-Meier curves from the BAYES registry representing the percentage of patients who develop atrial fibrillation (A), stroke (B), and atrial fibrillation or stroke (C), as a function of time. Group A: solid blue line. Group B: dashed red line. Group C: dashed green line. AF: atrial fibrillation; IAB: interatrial block. Reproduced with authorisation from Martínez-Sellés et al.[9]

Atrial Cardiopathy and Antithrombotic Drugs in Prevention after Cryptogenic Stroke (ARCADIA)[10] and Anticoagulation Management in Interatrial Block with Long-term Evaluation (AMIABLE). Both seek to evaluate the efficacy of anticoagulation in preventing stroke recurrence (in the former) and first stroke (in the latter) in patients with atrial dysfunction, including IAB.[5]

In this context, ambulatory external electrocardiography monitoring is becoming increasingly important in the study of patients in whom AF is strongly suspected.[11] Such long-term monitoring techniques as Holter monitoring over periods of weeks and ambulatory cardiovascular telemetry are examples of the wide range of non-invasive monitoring methods showing great effectiveness in different clinical scenarios.[11] When correctly used, these tools may be extremely useful in the study of patients with IAB, enabling early detection of AF and, consequently, early implementation of oral anticoagulation with a view to preventing cardioembolic events.

Conflicts of interest

The authors have no conflicts of interest to declare.

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Bilateral quadriceps tendon rupture as unusual cause of acute paraparesis☆

La rotura del tendón cuadricipital bilateral como causa inhabitual de paraparesia aguda

Dear Editor:

The differential diagnosis of bilateral lower limb weakness includes a wide range of disorders; in these cases, central and peripheral causes of weakness (vascular, neoplastic, traumatic, or neuromuscular causes) must be sequentially ruled out. Unilateral quadriceps tendon rupture is rare, with a prevalence of 1.37 cases per 100 000 population; it is more common in men and in the fifth decade of life, and is associated with thyroid or kidney disorders.[1—6] Bilateral quadriceps tendon rupture is even rarer, with only isolated cases reported in the literature.[2—7]

We present the case of a 50-year-old man with personal history of arterial hypertension and obesity who was receiving doxazosin, bisoprolol, enalapril, and hydrochlorothiazide. He visited the emergency department due to thoracolumbar trauma following an accidental fall at his home from a height of approximately 2 metres. After the fall, he reported weakness in both lower limbs, which prevented him from walking. He did not report pain, sphincter dysfunction, or loss of sensitivity. He was assessed by the traumatology department; the initial examination revealed no signs of fracture or joint lesions, and radiography of the joints and lumbar region revealed no alterations.

A lumbar spine MRI scan also detected no significant alterations. Three weeks later, the patient visited the neurology department due to persistent inability to walk and a burning sensation in the suprapatellar region bilaterally, with kneecap swelling and quadriceps atrophy.

The clinical examination revealed 4+/5 strength in hip flexion and knee extension, and 5/5 strength in distal lower limb muscles, with no claudication in antigravity movements, bilateral flexor plantar reflex, and abolished patellar and Achilles reflexes bilaterally. Tactile and vibratory sensitivity were preserved. Gait could not be assessed due to the patient's inability to stand. No alterations were observed in the upper limbs, cranial nerves, or higher cortical functions (Fig. 1).

An electromyoneurography study did not detect neuropathy in the peroneal, tibial, or sural nerves, or any sign of underlying plexopathy. However, it did detect bilateral chronic denervation with polyphasic potentials in proximal leg muscles (adductor longus and vastus lateralis muscles bilaterally), and possible disuse muscle atrophy, with no signs of chronic or active denervation, at the distal level (L4-S1) bilaterally (Fig. 1).

A contrast-enhanced thoracolumbar MRI scan revealed left subarticular T6-T7 disc protrusion, causing mild spinal cord compression, with no signs of myelopathy, as well as smaller protrusions at L4-L5 and L5-S1 (Fig. 2).

We performed another physical examination, noticing a gap in the quadriceps bilaterally (suprapatellar depression or ''hack sign''), which was more pronounced in the right knee, and bilateral joint effusion (Fig. 1). An MRI scan of both knees revealed a full-thickness tear in both quadriceps tendons (> 90% of fibres in the right tendon and complete rupture in the left), with signs of calcific enthesopathy in the preserved fibres, and haematoma in the area of the rupture (Fig. 2).

In view of these findings, the patient was referred to the traumatology department and underwent surgery for tendon repair, progressing favourably after the intervention.

Bilateral quadriceps tendon rupture is rare even after accidental falls, and requires early diagnosis and surgical management. Its prevalence increases with age, and it may be associated with history of hyperuricaemia, thyroid or kidney disorders, diabetes, and corticotherapy.

Our patient had no history of any of these conditions. We first screened for any causes of acute compression (spinal cord compression, vertebral fractures, dural fistulas, psoas haematoma, and post-traumatic plexopathy), detecting no abnormalities. We also considered Guillain-Barré syndrome or acute nutritional polyneuropathy, but electromyography and laboratory tests yielded no results supporting those diagnostic hypotheses. Repeated physical examinations by different specialists detected a rare sign, which guided diagnosis.

The diagnostic triad of quadriceps tendon rupture is acute pain, inability to extend the knee, and a palpable suprapatellar gap.[8—10] An MRI scan of both knees confirmed the diagnosis. Treatment for quadriceps tendon rupture is conservative in case of partial rupture, whereas full-

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