Dysarthria-clumsy hand syndrome and multiple sequential acute limb embolisms as a form of presentation of aortic arch embolism

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Introduction. Aortic arch complex atheromatosis is a source of cerebral embolism. A percentage of lacunar infarct could be of embolic etiology, especially due to microemboli of the aortic arch.

Case report. We present the case of a 63-year-old hypertensive man suffering from dysarthria-clumsy hand syndrome for a right hemispheric minor ischemic stroke. The patient developed sequential acute thromboembolism of the left lower and right upper limbs. Computed tomography angiography revealed an aortic arch thrombus. Vascular surgery was successfully performed.

Conclusion. This case highlights the importance of considering embolic sources in lacunar syndromes, especially at the level of the aortic arch.

Key words. Acute ischemia syndrome. Aortic arch complex atheromatosis. Aortic arch embolism. Lacunar syndrome. Stroke. Thromboembolism.

Introduction

Cryptogenic or ischemic stroke of unknown cause account for approximately 25% of all ischemic strokes. There is convincing evidence that the majority of cryptogenic strokes are thromboembolic and can originate from any of several well-established potential embolic sources, including minor or covert cardiac sources, veins via paradoxical embolism, and non-occlusive atherosclerotic plaques in the aortic arch and cervical or cerebral arteries [1].

The importance of aortic arch complex atheromatosis as a source of cerebral embolism, which usually causes extensive non-lacunar cerebral infarctions, has recently been demonstrated [2]. The association between lacunar ischemia and aortic arch complex atheromatosis is not well established; however, it has been reported that there is a percentage of lacunar ischemia of embolic etiology (5-15%), especially in the context of microemboli from the aortic arch [3].

In fact, Adrià Arboix et al observed that aortic arch complex atheromatosis is observed in 18.3% of patients with a first lacunar ischemia [4]. This percentage is similar to that observed in the Rabinstein et al series, in which the frequency of aortic arch complex atheromatosis in 19% of patients with subcortical small cerebral infarcts was studied by transesophageal echocardiography [5]. In addition, one in five patients with lacunar syndrome do not have a lacunar ischemia; therefore, vascular neuroimaging is essential to exclude lesions other than lacunar ischemia [6].

We present a clinical case of a male patient who presented with dysarthria-clumsy left hand syndrome with sequential development of an acute thromboembolism of the left lower limb, and right upper limb 24 hours later, in the context of an incidental thrombus in the aortic arch. The patient provided written informed consent to the report of his case details and imaging studies.

Case report

A 63-year-old man, smoker and hypertensive, attended the emergency department for clinical manifestations of motor weakness in the left upper limb and speech articulation alteration of abrupt onset at 04:30 am, after waking up previously asymptomatic at 04:00 am, with no other associated somatic or neurological symptoms. The physical examina-

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Accepted:
30.08.24.

Conflict of interests:
None.

How to cite this article:
García-Granado JF, Galindo-Cordero Á, Hernández-Pérez S, Pérez-García MP, Pérez-Hernández P, de Varona-Frolov S, et al. Dysarthria-clumsy hand syndrome and multiple sequential acute limb embolisms as a form of presentation of aortic arch embolism. Rev Neurol 2024; 79:175-8. doi: 10.33588/rn.7906.2024203.

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Video 1. Focal endoluminal repletion defect at the apex of the aortic arch with triangular morphology occupying 20-30% of the vascular lumen, compatible with floating thrombi in the aortic arch.

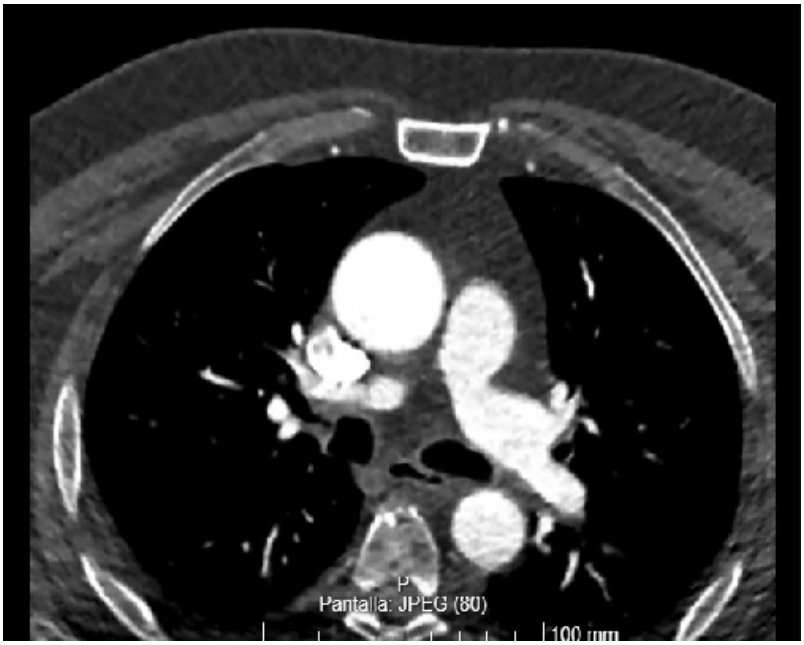

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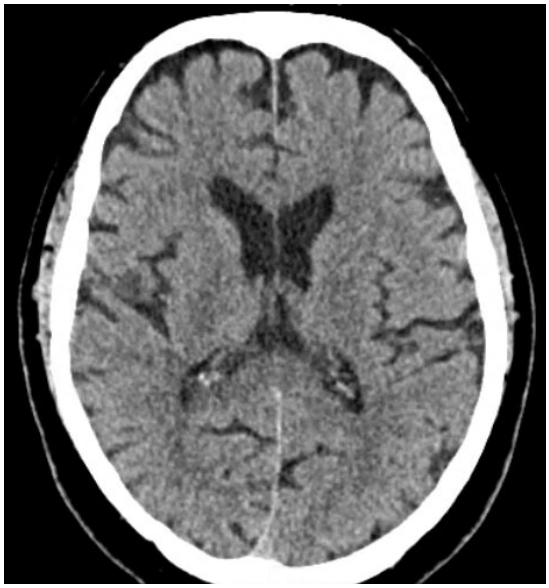
Figure. Cranial computed tomography, axial section showing a small hypodense lesion in the right posteroinferior frontal gyrus, which is compatible with acute ischemic stroke. Chronic lacunar infarction is also observed in the external capsule and bilateral lenticular nucleus.



tion was normal, and the neurological examination revealed mild dysarthria and motor balance of 4/5 in the distal region of the left upper limb according to The Medical Research Council Scale of the United Kingdom, with the rest of the neurological examination being normal. National Institutes of Health Stroke Scale (NIHSS) = 2.

Blood tests for ions, renal and liver function, hemogram, and coagulation were normal. Likewise, the cardiological examination by electrocardiogram and transthoracic echocardiography was normal.

Urgent neuroimaging by cranial computed tomography was performed, showing chronic lacunar ischemia in the external capsule and bilateral lenticular nucleus and a small hypodense lesion in the right posteroinferior frontal gyri of acute chronology (Figure). Likewise, neurosonological study revealed no alterations.

The patient was discharged with the diagnosis of lacunar syndrome dysarthria-clumsy hand in the context of minor ischemic stroke, clinically right hemispheric LACI (lacunar infarction) of probable hypertensive etiology, and it was decided to discharge him home with secondary preventive therapy with double antiplatelet therapy, antihypertensive drugs, and statins.

The patient returned to the emergency department 48 h later with abrupt onset of pain and hypoaesthesia in the left calf and back of the thigh, with no previous symptoms of intermittent claudication. Physical examination revealed pallor of the limb and weak femoral, popliteal, and left foot pulses, with the rest of the physical examination being normal.

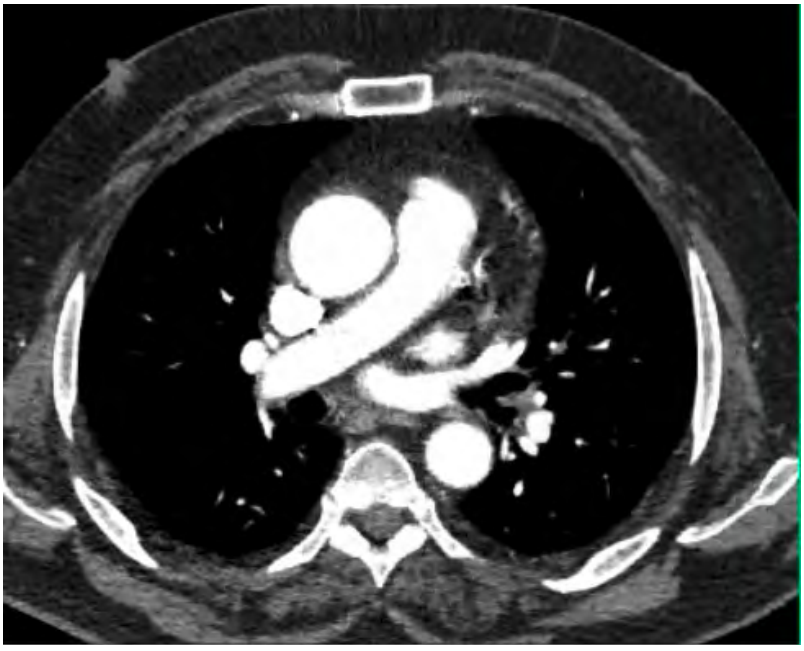
It was decided to extend the urgent neuroimaging study by means of computed tomography angiography, which ruled out extracranial and intracranial stenosis; however, a focal endoluminal repletion defect was observed at the apex of the aortic arch with a slightly triangular morphology that occupied between 20% and 30% of the vessel lumen, compatible with a floating thrombus in the aortic arch (Video 1), with subsequent pathological confirmation. Ultrasonography of the left lower limb showed an absence of arterial and venous Doppler ultrasound in the anterior tibial/posterior tibial region, with no popliteal arterial doppler signal and evidence of end-stage embolism in the bifurcation of the common femoral artery, as well as in the origin of the superficial femoral artery and deep femoral artery.

The patient was diagnosed with acute ischemia syndrome of the left lower limb, and transfemoral embolectomy was performed. He was discharged with enoxaparin at therapeutic doses and double

antiplatelet therapy because of the emboligenic mechanism.

However, he returned to the emergency department for the third time 24 hours later because of pain and hypoaesthesia in the right upper limb. Vascular examination revealed a right humeral rebound pulse, with no distal pulses, and ultrasound showed thrombus in the bifurcation of the humeral artery and in the radial artery. The patient was diagnosed with acute ischemia syndrome of the right upper limb, and transhumeral embolectomy was performed. Finally, in the follow-up computed tomography angiography scan one month later, the floating thrombus disappeared (Video 2) and anticoagulation was optimized.

Video 2. Control computed tomography angiography performed one month later showed complete disappearance of the floating thrombus in the aortic arch.



Discussion

Discussion of this case highlights the importance of considering embolic sources in lacunar syndrome. The patient presented with dysarthria-clumsy hand syndrome, initially attributed to lacunar ischemia, but developed subsequent systemic embolic complications. computed tomography angiography revealed a thrombus in the aortic arch, highlighting the relevance of aortic arch complex atheromatosis as a source of microemboli. This finding underscores the need for a thorough vascular work-up in patients with lacunar syndrome, particularly when it is associated with systemic embolic phenomena. Timely identification and treatment of aortic arch thrombi by transfemoral and transhumeral embolectomy were essential to prevent further complications. This case emphasizes the importance of vascular neuroimaging and continuous surveillance in patients with lacunar syndrome and possible embolic etiologies, thus improving prognosis and therapeutic management.

Conclusions

Aortic arch complex atheromatosis and microemboli from the aortic arch are considered sources of multiple systemic embolisms. In 20% of patients with lacunar syndrome, there is no underlying lacunar ischemia; therefore, not all lacunar syndromes are due to small vessel arteriopathy. It is advisable to extend the vascular neuroimaging study to include intracranial, extracranial, and aortic arch vessels. Early intervention and a multidisciplinary approach are vital for optimizing patient outcomes.

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Síndrome de disartria-mano torpe y embolias agudas secuenciales múltiples de las extremidades como forma de presentación de un trombo del cayado aórtico

Introducción. La ateromatosis del complejo del arco aórtico es una fuente de embolia cerebral. Un porcentaje de infartos lacunares podría ser de etiología embólica, especialmente debidos a microembolias del arco aórtico.

Caso clínico. Presentamos el caso de un varón hipertenso de 63 años con síndrome de disartria-mano torpe por un ictus isquémico *minor* hemisférico derecho. El paciente desarrolló un tromboembolismo agudo secuencial de los miembros inferior izquierdo y superior derecho. La angiografía por tomografía computarizada reveló un trombo en el arco aórtico. La cirugía vascular se llevó a cabo con éxito.

Conclusión. Este caso destaca la importancia de considerar las fuentes embólicas en los síndromes lacunares, especialmente en el arco aórtico.

Palabras clave. Ateromatosis del complejo del arco aórtico. Ictus. Ictus isquémico. Síndrome lacunar. Trombo del cayado aórtico. Tromboembolismo.