

Stroke Due to Chagas' Cardiomyopathy or Noncompaction

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With interest we read the article by de Mello et al¹ about a male patient with Chagas'-infection in whom left-ventricular hypertrabeculation/noncompaction (LVHT) was found¹. We have the following concerns:

Confirmation of stroke by CT-scan is insufficient. An MRI would have been more helpful to classify the lesion. How was a stroke-like-lesion excluded? Is information about the cerebral or extra-cerebral vasculature from carotid-ultrasound or MR-angiography available? Were causes of stroke other than atrial flutter (AFLU) or LVHT excluded? Did the patient have diabetes, arterial hypertension, previous stroke, myocardial infarction or hyperlipidemia? Chagas' itself may cause stroke, particularly in the anterior circulation and recur in 20% of cases. Was it considered as a cause? Did AFLU recur after ablation? Was oral anticoagulation therapy necessary after ablation? Did mitral regurgitation regress during follow-up?

Meningoencephalitis may be a manifestation in the acute phase of Chagas' and neuritis, sensory disturbances, dementia, confusional state or encephalopathy manifestations of the chronic stage. Did the patient undergo cerebrospinal-fluid investigations? Were there other manifestations of Chagas'?

The authors regard LVHT as congenital, an assumption that is plausible, but was not convincingly confirmed. Was acquired LVHT also considered, as it has been occasionally reported? Particularly, the link between Chagas' and LVHT should be discussed. Was there ever myocarditis, which has been occasionally reported together with LVHT? Since Chagas' may go along with myocarditis, it is not inconceivable that LVHT developed because of the disaggregation of the myocardium and subsequent hollowing of the tissue by the pressure of the surrounding blood.

Were genetic disorders associated with LVHT other than Barth-syndrome considered? LVHT has been particularly found in patients with chromosomal abnormalities, neuromuscular disorders and hereditary cardiomyopathies? Was there facial dysmorphism or bone abnormalities? Was there ptosis, double vision, muscle cramps, weakness, respiratory insufficiency or elevated muscle enzymes?

LVHT may occur also in other family members of an affected patient. Were relatives investigated? Did they also suffer from Chagas'? Were other cases with Chagas' and LVHT found?

A ratio of 6:1 on cMRI is exceptionally high. How do the authors explain this unusual value? Was it confirmed by transthoracic echocardiography? Were thrombus and tumors excluded?

As there are some unsolved issues with the presentation and work-up of this case, reevaluation of the patient and his relatives is warranted.

Keywords

Stroke; Chagas' cardiomyopathy; ventricular dysfunction left; brain ischemia

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Reference

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Reply

The association between left-ventricular hypertrabeculation/noncompaction (LVHT), as well as Chronic Chagasic Cardiopathy (CCC) with thromboembolic events has been well defined, including ischemic stroke. In the present report, the patient presented a previous history of stroke with complementary investigation and exclusion of other causes for the ischemic event, through Echo-Doppler evaluation of the vertebral/carotid arteries and transesophageal echocardiogram, which are routinely carried out in our institution. After the ischemic event, the patient received oral anticoagulation with adequate control of therapeutic levels. The patient did not present other comorbidities (diabetes mellitus, arterial hypertension and dyslipidemia), as well as coronariopathy, ruled out by the clinical history, cardiac magnetic resonance (cMRI) and coronariography. In a series from our service that has not been published yet, we observed that in CCC, the presence of an embolic event occurs predominantly in individuals with apical aneurysms. On the other hand, this patient presented three potential cardioembolic event inducers, among which one cannot ignore the presence of atrial flutter.

The main objective of the report was to present the association of extensive trabeculation, characteristic of LVHT and the fibrosis caused by the CCC. The report consists in the description of a patient with symptoms of heart failure associated with recent-onset atrial flutter (AFw). After the cMRI, transthoracic and transesophageal echocardiograms were performed, the patient was submitted to an electrophysiological study, with successful ablation

of the isthmus-dependent AF and sinus node disease documentation. The aforementioned imaging studies did not identify intracavitary thrombi or heart tumors. However, the presence of fibrosis typical of CCC and hypertrabeculations compatible with the manifestations of non-compaction was broadly demonstrated. After the ablation of the AFw and pacemaker implant (sinus node dysfunction), the patient has remained asymptomatic, while maintaining oral anticoagulation therapy and treatment for cardiopathy with captopril, carvedilol, furosemide and spironolactone. It is noteworthy the fact that the patient presented classic manifestations of CCC and an acute infection or infection reactivation due to immunodeficiency do not apply to the present case, particularly because the patient (and siblings) had been out of the endemic zone for more than 20 years and the signs (Romana's sign) and symptoms of acute infection had occurred during childhood. Other forms of presentation of the chronic form of CD, mainly digestive involvement, were ruled out.

The patient and his family members did not present any morphological manifestations that could lead to the suspicion of genetic disorders. We recall that the association between non-compaction and genetic alterations is not a mandatory one and its sporadic isolated form has been broadly documented in the literature¹. We have followed the patient routinely at the outpatient clinic for three years and he has not presented recurrence of atrial arrhythmia or ventricular arrhythmia recordings. The echocardiograms performed annually have shown worsening in ventricular function, which represents the evolution of the described cardiomyopathies.

Reference

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