Indications for Anticoagulation in Cardiac Amyloidosis

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In cardiac amyloidosis, amyloid infiltrate at the atrial level promotes atrial dilation and contractile dysfunction as well as a higher prevalence of atrial fibrillation which, when associated with ventricular diastolic and systolic dysfunction, favors blood stasis and the consequent development of intracardiac thrombosis (ICT) and cardioembolic events.

In a study of 116 autopsies of patients with cardiac amyloidosis, conducted at the Mayo Clinic, the presence of ICT was demonstrated in 33% of hearts, with a significantly higher prevalence in patients with the AL form of amyloidosis than in those with other forms (56% versus 16%, p < 0.001).1

When evaluating 324 patients with cardiac amyloidosis using cardiac magnetic resonance, we found an ICT prevalence of 6.2%, with 90% located in the atrial appendage. Among the patients, 70% had atrial fibrillation, and 30% had sinus rhythm, with a similar prevalence of ICT in the AL (5.2%) and ATTR forms (7.2%).2,3

Morphological and functional changes caused by atrial amyloid infiltrate favor the development of atrial fibrillation, with a prevalence ranging from 29% to 60%, depending on the population, and it is more prevalent in the ATTR form, given that it affects an older population. The presence of atrial fibrillation poses a high risk for the development of ICT and stroke, especially in patients with AL amyloidosis.4

Approximately 20% to 30% of patients who have ICT, as well as 27% of patients with cerebral ischemic events are in sinus rhythm.3,5

The probable mechanism responsible for the development of thrombotic events and thromboembolism in patients in sinus rhythm would be the presence of atrial myopathy due to amyloid infiltrate and high ventricular filling pressures, which induce atrial contractile dysfunction and favor blood stasis and ICT formation.5,6

By means of logistic regression analysis, several factors have been identified that are related to greater predisposition to ICT and thromboembolic events, such as biventricular systolic dysfunction, restrictive pattern of diastolic dysfunction, atrial dilatation, degree of atrial amyloid infiltration, and high NT-proBNP levels. In the presence of these factors, we must carry out a specific evaluation as to the possible benefit of anticoagulation for preventing cardioembolic events.2

In patients with cardiac amyloidosis who have ICT or atrial fibrillation, anticoagulation treatment is absolutely indicated, due to the benefits in reducing thromboembolic events and improving survival.8

New anticoagulants and coumarin are similar in terms of benefits in reducing stroke and increasing survival, as well as in terms of increasing the incidence of bleeding events. Approximately 55% to 68% of patients taking coumarin have difficulties reaching the stable therapeutic target of the international normalized ratio, especially in the elderly population, which we must take into account when making decisions regarding the choice of new anticoagulants as an alternative for anticoagulation.9-11

Even though anticoagulation in patients with amyloidosis has been associated with an increased risk of bleeding events due to amyloid angiopathy, especially in patients with the ATTR form, who are at risk of intracranial hemorrhage, this risk does not outweigh the benefits of preventing thromboembolic events; therefore, anticoagulation should be carried out when it is indicated.12-13

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