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EDITORIAL COMMENT

Invasive versus non-invasive coronary microvascular assessment in hypertrophic myocardiopathy – Are we measuring the same thing?

Avaliação invasiva e não-invasiva da doença microvascular na miocardiopatia hipertrófica – Estaremos a medir a mesma coisa?

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Hypertrophic cardiomyopathy (HCM) is the most common genetic heart disease inherited as an autosomal dominant trait. Its main characteristic is left ventricular hypertrophy that occurs in the absence of other conditions that trigger this change.[1]

Disease penetrance and the age of phenotypic development range over generations; initial presentations may vary from asymptomatic forms found in routine echocardiography to manifestations of heart failure or sudden cardiac death.[2] Despite having a low long-term mortality, the burden of morbidity in Portugal remains considerable. Age at diagnosis is relatively advanced, emphasizing both the need for better diagnostic strategies and better treatments targeting symptomatic control and the natural history of the disease.[3]

Clinical scores have been developed to provide prognostic and functional assessments and to enable risk stratification.[2] Additionally, high-resolution imaging methods, such as cardiac magnetic resonance (CMR), represent an important complement to echocardiography in different clinical scenarios.[4] CMR is becoming increasingly used in HCM, especially for the identification of other important hallmarks of the disease: myocardial fibrosis (which is usually more prominent in the most hypertrophic segments) and coronary microvascular disfunction (CMD). Both these features have been proven to be directly linked to, but also to be independently associated with poor outcomes in HCM patients.[5]

In this issue of the Journal, Rosa et al.[6] present part of their research into CMD in patients with HCM, using both regadenoson stress-rest CMR and the invasive assessment of the index of microcirculatory resistance (IMR) during coronary catheterization. For the first time, IMR has been used for the assessment of CMD in HCM patients without epicardial coronary artery disease, adding an important contribution to the scientific knowledge in his field. Despite the inherent limitations associated with the number sample size, this pilot study has the advantage of the simultaneous use of two very different techniques (CMR and IMR) for the assessment of CMD. This approach enables a direct comparison of the findings and opens up an interesting discussion on how to correlate and interpret the results.

As the authors correctly state, CMD is one of the less understood pathophysiological features of HCM. Several methods can be used for the assessment of CMD but there is no universal gold-standard and the different methods are probably measuring different markers of the same reality. Therefore, it is interesting to note that patients with abnormal IMR seemed to have more significant tissue abnormalities as defined by CMR, namely fibrosis and increased extracellular volume (as assessed by late gadolin-

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ium enhancement and T1-mapping). And, in my view, it is particularly exciting to interpret the somehow discordant results obtained with the two techniques. The concordance between IMR and CFR was found in only six patients (43%) and the prevalence of CMD by IMR was significantly lower than the detection of ischemia using stress CMR. The authors raise the hypothesis that lower IMR values may be found in HCM patients compared with other diseases, and postulate that HCM may run its course with a reduction in coronary flow reserve secondary to near maximal baseline vasodilation of the microcirculation, instead of an increase in coronary microvascular resistance due to a narrowing of small vessels or external compression. This type of CMD would be detected using CMR, which is sensible for myocardial perfusion flow reserve, but not for IMR, since microvascular resistance would not be significantly. Another interesting point is that CMR is able to simultaneously assess the cumulative global impact of CMD in the whole myocardium, while IMR and the other invasive techniques such as coronary flow reserve measurement have the advantage of being vessel-specific but may lose some sensitivity in a global microvascular tree assessment.

As good research often does, this study raises more questions than it gives answers. Nevertheless, it is another piece of a puzzle that is far from being complete and invites us to conduct further research into the potential of IMR and the value of CMD assessment in patients with HCM. We will be sitting on the front row to see what this approach may bring to the management of these patients, especially for the diagnosis, symptom control and prognosis-modifying treatment.

Conflicts of interest

The author has no conflicts of interest to declare.

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