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Patent foramen ovale and MRI: Can imaging the brain tell us about the heart?

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Nothing fosters a heated discussion more than firmly held opinions that are based on incomplete information. Ever since the foramen ovale occasionally was found to be patent beyond the neonatal period, there has been a discussion about its relevance. There have been well documented cases of paradoxical embolism and even pathological specimens with thrombi trapped inside the hole. Some argue that the lesion is so dangerous that direct surgical closure or an implanted endovascular closure device is needed. Others still question the phenomenon altogether.

The most compelling evidence that paradoxical embolism through a patent foramen ovale (PFO) may be more than just a medical curiosity is the relatively consistent finding across dozens of case-control studies that patients with PFO are substantially over-represented among patients with cryptogenic stroke, compared both to patients with stroke of known cause and to age-matched non-stroke patients. Depending on the series, nearly 50% of patients with cryptogenic stroke have a detectable PFO compared to approximately 20% among control patients. According to Bayes' theorem, these numbers would suggest approximately two-thirds of cryptogenic strokes in patients with PFOs are attributable to the PFO, while in the rest the PFO is an incidental finding.[1] (The rate of incidental PFOs would be still lower in younger patients, or in patients with atrial septal aneurysm.)

Feurer and colleagues [2], however, appear unconvinced by this evidence. Against these prior studies, they submit their observations of the lesion patterns seen on MRI scans of patients with ischemic strokes with and without PFO. Their data show that MRI lesions in patients with PFO do not differ substantially from those without PFO, and in particular there is not a preponderance of scans showing "multiple ischemic lesions," a pattern they assert is often associated with cardioembolic strokes.

They conclude that their findings "do not provide any support for the common theory of paradoxical embolism as a major cause of stroke in PFO carriers." True enough. However, it is also true that their findings do not provide a serious challenge to this common theory. Simply put, the characteristic MRI findings of strokes caused by paradoxical embolism are unknown. Though sometimes implied, the widely-employed TOAST classification that the authors use to cope with the heterogeneity of stroke patients does not assume that there is homogeneity within a category – e.g. within cardioembolic stroke. For example, the mechanism, prognosis, and response to treatment of endocarditis-related stroke is very different from that related to atrial fibrillation, even though both are cardiogenic. Not enough is known yet about paradoxical embolism to infer that it is likely to be similar to

these other cardiogenic stroke types in terms of its typical neuroradiologic findings or other characteristics.

Indeed, the value of this study is that it suggests that the finding of multiple ischemic lesions is not especially common in patients with otherwise cryptogenic stroke and PFO, and is therefore unlikely to be a reliable sign for paradoxical embolism. This study is amongst the first cohorts of PFO patients to focus on radiological observations. Descriptions of the stroke patterns seen on MRI of patients with PFO and cryptogenic stroke (particularly younger patients and those with high-risk features such as atrial septal aneurysm and especially large shunts) may provide information that is ultimately helpful in identifying stroke patients with PFOs more likely to be pathogenic than incidental (i.e. unrelated to the stroke that led to its discovery). Endovascular closure for an incidental, non-pathogenic, PFO is not likely to reduce the risk of stroke recurrence—and may indeed increase this risk. Discriminating between pathogenic and incidental PFOs is thus crucial when advising patients about secondary prevention strategies, but current science is largely uninformative about how to do that. Neuroimaging data, once we understand the common patterns, may help to identify culprit PFOs and help us to ignore the innocent bystanders.

There are those who are convinced that the danger of a PFO is so great that they advocate “mechanical vaccination” against stroke. [3] There are many logical inferences involved in this recommendation: 1) paradoxical embolism is a stroke mechanism, 2) patients can be identified who have it, 3) the natural history without treatment is known, 4) the benefits of treatment are known, 5) the risks of the treatments are known, and 6) the comparative risk/benefit ratios for different treatments are known. Reliable data to support these leaps from suspicion of paradoxical embolism to confidence in the benefits of an interventional treatment are lacking. While we disagree with some of the authors main inferences regarding their results, we wholeheartedly endorse their final conclusion that questions concerning secondary stroke prevention need to be investigated further by prospective randomized clinical trials.

References

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