

The mystery of intracranial aneurysm formation

O mistério da formação de aneurismas intracranianos

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Intracranial aneurysms are a common and potentially devastating condition. They are found in approximately 1% of autopsies and 7% of patients referred for catheter angiography for indications other than subarachnoid hemorrhage¹. Pathogenesis is not completely understood, but it is generally accepted that both genetic and hemodynamic factors play a role. As a group, a number of known heritable disorders account for only 5% of aneurysm cases, such as Ehlers-Danlos syndrome, autosomal dominant polycystic kidney disease, Marfan syndrome and others². A series of recent genome-wide association studies have identified candidate genes related to increased risk of aneurysm formation^{3,4}. While some of these genes are related to wall structure, others are related to hemodynamic wall stress⁵. Thus, hemodynamic factors are still thought to play the dominant role in aneurysm formation, justifying their preferential location in arterial bifurcations.

In this issue of *Arquivos de Neuro-Psiquiatria*, Silva Neto et al.⁶ shed further light into the pathophysiology of aneurysm formation. In this angiographic case series, 169 patients diagnosed with intracranial aneurysms were compared with 256 patients referred for multiple reasons to catheter angiography without intracranial aneurysms. A series of common anatomic variants of the circle of Willis were found to be related to different aneurysm locations. Aneurysms in the posterior communicating artery (PCoMA) were associated with fetal-type PCoMA; and anterior communicating artery (ACoMA) aneurysms were associated with A1 hypoplasia. Both variants increase flow to arterial bifurcations, theoretically increasing the odds of aneurysm formation. Moreover, a careful analysis of the carotid syphon was performed, measuring the angle between the intracavernous and supraclinoid segments of the carotid artery. A lower angle (which also increased local blood flow distally) was associated with higher occurrence of aneurysm.

Some emphatic points in Silva Neto's work are the large sample size and biological plausibility of the findings. As in any single-center case series, authors must recognize that their findings may not generalize to other unselected samples. The authors do not explain their criteria for excluding 57 cases (from a sample of 512 patients, 30 did not have reports, 169 had aneurysms and 256 were controls, leaving 57 unaccounted patients). Calculation of the syphon angle is subject to an unknown measure of inter-rater variability and some bias may be expected from raters, as they were not blind to aneurysm status or location. Still, the authors are to be congratulated for their efforts in studying a significant population and for increasing our knowledge on the origin of intracranial aneurysms in a Brazilian population.

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