Through the last two decades, there has been a significant progress on the understanding of the nature of various congenital vascular malformations (CVMs), and the confusion based on old name-based eponyms and classification is now sufficiently cleared. Further, contemporary concept on the CVMs and its management by a multidisciplinary approach are now fully established.

However, current knowledge is not sufficient enough to correct many unfair prejudices with a proper answer, and questions such as the aneurysm of congenital origin as the result of a birth defect remain to be answered.

Among the arteriovenous malformations (AVMs), aneurysm formation along the feeding artery and/or draining veins is not rare at all; it is a ‘well anticipated’ haemodynamic reaction/consequence to such ‘high flow’ lesions.

Together with a tortuosity, saccular or fusiform dilatation of the vessels is a common complication along the natural course of the progress of such lesion, when left alone.

Therefore, all the extratruncular AVM lesions should be considered as candidates for aggressive control ‘before’ they progress to cause such potentially limb-threatening condition (e.g., aneurysmal rupture).

However, such extratruncular lesions are the embryonic tissue remnant following the developmental defect along the “earlier” stage of embryogenesis, so that all of them possess mesenchymal cell characteristics of an evolutionary power to grow when stimulated (e.g., female hormone, menarche, pregnancy, trauma, surgery and so on). 

Therefore, complete destruction of the ‘nidus’ of the lesion is warranted for effective control of the lesion, and the once popular approach with ligation of the feeding artery due to misunderstanding is now condemned, as it only provokes the lesion to enhance neo-vascular recruitment to make the condition worse.

As the authors correctly pointed out, without proper control of the AVM lesion itself as the primary cause of the aneurysm, recurrent as well as de novo aneurysm development is an unavoidable consequence. Further, a life-/limb-saving surgical ligation of the ruptured aneurysm alone would rather cause more stimulation and should be followed by appropriate management of the AVM (e.g., ethanol sclerotherapy). Contrary to this ‘secondary(?)’ aneurysm as the consequence of the AVM lesions, a ‘primary’ arterial/venous aneurysm is relatively rare and classified separately to ‘truncular’ arterial malformation (AM)/truncular venous malformation (VM).

‘Truncular’ malformation is an outcome of birth defect along the “later” stage of embryogenesis so that it no longer possesses mesenchymal cell characteristics as extratruncular lesions. Instead, various conditions involving the vessel wall, such as a dilatation, occlusion/stenosis and defective vessel structures (e.g., web and membrane), would cause only
haemodynamic impacts depending upon their severity, extent and location (e.g., absence of femoral artery).

References