Cervical Artery Dissection: An Important Cause of Stroke in Young

Cervical artery dissection (CAD) is an important cause of stroke in young, accounting for approximately 25% of cases in individuals below 45 years of age.[1,2] CAD encompasses both carotid artery dissection and vertebral artery dissection, the former being twice as common.[3,4] Trauma through motor vehicle accidents or neck manipulations (barber, chiropractic, and whiplash) is the most common cause of CAD and can affect extracranial (more common) and intracranial arteries. The presence of vascular risk factors is less prevalent in patients with CAD (younger age group). However, risk factors such as hyperhomocysteinemia, presence of preceding infections/fever, and migraine are more prevalent in them.[5]

Chakraborty *et al*.[6] describe a multicenter, retrospective case series of patients with posterior cerebral artery (PCA) dissections. They have described 14 such patients, enrolled over a study period of one year. Interestingly, 13 of these patients presented with subarachnoid hemorrhages (SAHs) and only one presented with an infarct. Most of them underwent endovascular coiling to treat the dissecting aneurysms to prevent rebleeds and had a good functional outcome.

Arterial dissections cause a tear in the intima of the vessel wall and lead to neurological deficits mainly by two mechanisms: (a) ischemia (hypoperfusion or thromboembolism) due to luminal compromise caused by tracking of the intramural hematoma between the layers of the blood vessel and (b) SAH caused by tracking of the intramural blood outward through the adventitia leading to the formation of aneurysms (which rupture).[5] Ischemic presentations are more common. However, the occurrence of SAH is more common in intracranial dissection as compared to extracranial dissection. This is because the intracranial arteries lack the presence of an external elastic lamina, making it easier for the blood to track outward.[5] The most common sites for extracranial dissection are as follows: the origin of the internal carotid artery from the common carotid artery and the V2–V3 segment of the vertebral arteries. Intracranial arterial dissections are more random, and isolated PCA dissections are extremely rare. These PCA dissections can be associated with SAH, as described by the authors,[6] and portend a poorer prognosis than ischemic insults.

Stroke due to CAD needs to be differentiated from that caused by atherosclerotic disease. The presence of neck pain is a useful indicator toward dissection as the dissected blood irritates the pain-sensitive nerve endings present in large arteries.[6] Vascular imaging (computed tomographic or magnetic resonance angiography) can also provide vital clues toward dissection being the etiology. These include the presence of multifocal and generalized atherosclerotic disease compared with focal findings in dissections. Also, the latter can have the concomitant presence of intimal flaps, crescent sign, double lumen, or aneurysms.

Randomized controlled trials[7,8] have established antiplatelets to be noninferior to anticoagulants and tend to be the first choice for physicians, especially in intracranial dissections due to the fear of SAH. Also, unlike atherosclerotic disease, stroke recurrence is rare in patients with CAD and predicts a better prognosis. Ischemic strokes associated with dissections are not contraindications to thrombolysis or endovascular thrombectomy (provided other eligibility criteria are met), and patients with SAH associated with aneurysms should undergo endovascular treatment for them. Repeating vascular imaging to look for recanalization of previously thrombosed arteries remains debatable as studies have not found any difference in functional outcomes between patients who achieve recanalization and those who do not.[9]

Ayush Agarwal, M. V. Padma Srivastava

Department of Neurology, AIIMS, New Delhi, India

Address for correspondence: Prof. M. V. Padma Srivastava, Department of Neurology, Chief, Neurosciences Centre, All India Institute of Medical Sciences, New Delhi, India.
E-mail: Vasanthapadma123@gmail.com

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