VERTEBRAL ARTERY STENOSIS: A NARRATIVE REVIEW

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Burle et al. (St. George University School of Medicine, Clarksville, USA) review the anatomic, pathophysiologic, and therapeutic features of vertebral artery stenosis (VAS). They are responsible for 20% of subsequent ischemic cerebrovascular accidents (ICA). The causes of VAS, in addition to calcification and atherosclerotic lesions, are dissections, fibromuscular dysplasia, giant cell arteritis, neurofibromatosis, and bone compression. The most frequent symptoms are vertigo, vision disorders, nystagmus, dizziness, loss of consciousness, nausea, and ataxia. They can lead to stroke, infarction, vertebrobasilar insufficiency, and sudden death without diagnosis and treatment.

The vertebral artery is anatomically divided into four segments (V1-V4): the ostial segment (from its origin to the transverse foramen of the sixth cervical vertebra), the transverse segment (to the second vertebra), the suboccipital segment (until it crosses the dura mater) and the fourth or intracranial segment, which extends to its junction with the contralateral vertebral artery to form the basilar artery.

The authors review the causes of calcifications and atherosclerotic lesions in the extracranial segments.

Lesions caused by dissection are infrequent in the general population but are one of the leading causes of stroke in those under 45, with an incidence of 10-25%. They can be traumatic, associated with previous lesions, or spontaneous; the latter are seen more frequently in older adults. Possible causes include chiropractic maneuvers, spontaneous cranial movements, cervical trauma, oral contraceptives, and fibromuscular syndromes such as Ehlers-Danlos type IV and osteogenesis imperfecta type I. Dissections usually occur in segment V3 because of its proximity to the atlantoaxial joint, which is involved in the rotational movement of the head.

Fibromuscular dysplasia is a hereditary, non-atherosclerotic, non-inflammatory vascular condition that can affect any vessel and is the third most common cause of vertebral artery injury. It is caused by abnormal deposition of collagen fibers. It can affect the intimal, medial, or adventitial layers, with the medial layer being the most frequently involved, manifesting as alternating areas of dilatation and stenosis. The forms affecting the intimal or adventitial layers, which are less frequent, are stenotic.

Another rarer cause of AVS is giant cell arteritis, an autoimmune disorder that affects the vasculature by inflammation of the internal elastic membrane. Arteries with this granulomatous process, which in 75 to 100% of cases affects the vertebral arteries, present as thrombotic obstruction, necrosis, or stenosis. Neurofibromatosis type 1 is an autosomal dominant disorder affecting several tissues (brain, bones, and vessels) and can manifest as stenosis, occlusion, aneurysms, and arteriovenous fistulas, is also described.

External compressions can produce VAS at any point along its course, the most frequent site being the C1-C2 level. Primary causes include neck muscle hypertrophy, osteophytes, fibrous bands, idiopathic hyperostosis, cervical spondylosis, spondylolisthesis, herniated discs, and other mobility disorders and tumors. A particular case is the bow hunter's syndrome due to compression of the V3 segment, which typically occurs in cases of a dominant vertebral artery occluded by head rotation.

VAS can cause symptoms such as vertigo, ataxia, diplopia, speech disorders, bilateral hemianopsia, syncope, headaches, tinnitus, other neurological symptoms, and an increased risk of stroke or transient cerebral ischemia. The most severe symptoms are usually seen when the lesion is bilateral. The true incidence of VAS is unknown because of the large number of asymptomatic cases.

Subclavian steal syndrome is a special case of flow compromise resulting from injury to the subclavian artery in the vicinity of the origin of the vertebral artery, leading to a reversal of flow in the ipsilateral vertebral artery.

Because of its complex, tortuous, variable anatomy, with extracranial and cranial segments, VAS often offers difficulties in diagnosis. Doppler examination has low sensitivity compared to other noninvasive diagnostic methods, such as computed tomography and magnetic resonance imaging. Digital angiography is the most accurate method, although it is more invasive and risky.

In addition to preventing and treating risk factors common to other arteriopathies, it is suggested that patients with VAS due to atherosclerosis or compression receive antiplatelet medication. The WASID (warfarin-aspirin symptomatic intracranial disease) study concluded that warfarin would be more effective for stroke prevention in patients with symptomatic VAS.

In surgical treatment, the authors point out the technical difficulties, poor success, and complications of endarterectomy. Reconstructive surgeries have better results, especially when the involvement is proximal. Although surgical treatment of VAS has some degree of success, it is a technically challenging procedure with frequent postoperative complications and deaths. Direct surgery is performed less frequently because of advances in endovascular techniques. Currently, angioplasty-stenting of symptomatic VAS is considered a safe, durable, and effective method, and drug-eluting stents are preferred because of their lower restenosis rate.