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CERVICAL OSTEOCHONDROSIS AND ISCHEMIC STROKE: VERTEBROBASILAR MECHANISMS, RISK ASSESSMENT AND PREVENTION

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Abstract: *Cervical osteochondrosis, described in the international literature as cervical spondylosis or degenerative cervical disc disease, is among the most common musculoskeletal conditions of adults, and ischemic stroke remains a leading cause of death and disability. This article reviews how degenerative changes of the cervical spine may contribute to ischemia in the vertebrobasilar territory, summarizes epidemiological evidence of an association, and outlines a practical approach to recognition, imaging and prevention in clinical practice. The evidence supports an association with posterior circulation stroke rather than proof of a direct causal role, and management should therefore combine vascular risk control with careful spinal assessment.*

Keywords: *cervical osteochondrosis, cervical spondylosis, ischemic stroke, vertebral artery, vertebrobasilar insufficiency, posterior circulation, prevention.*

Ischemic stroke and degenerative disease of the cervical spine are both very frequent in middle-aged and older adults, so they inevitably coexist in many patients. The clinical question is whether the coexistence is coincidental or whether osteochondrosis of the cervical spine can itself contribute to cerebral ischemia. The question is most relevant for the posterior circulation, which is supplied by the vertebral arteries and the basilar artery and accounts for roughly one fifth of all ischemic strokes [1]. The vertebral arteries pass through the transverse foramina of the sixth to the second cervical vertebrae, running directly beside the uncovertebral joints and facet joints, which are the very structures altered by degeneration. This close anatomical

relationship provides a plausible route by which spinal disease could affect blood flow to the brainstem, cerebellum and occipital lobes. The aim of this article is to summarize the current understanding of this relationship and its implications for everyday practice.

Degeneration begins in the intervertebral disc, which loses water content and height, and is followed by osteophyte formation at the uncovertebral and facet joints, thickening of ligaments and segmental instability. Several mechanisms have been proposed by which these changes may compromise vertebrobasilar flow. Marginal osteophytes may narrow the transverse foramen and compress or angulate the vertebral artery, particularly during rotation or extension of the neck, producing a positional, so-called bow hunter type of occlusion. Case reports describe positional occlusion of a vertebral artery due to cervical spondylosis as a rare cause of stroke on waking [2], and a series published in the European Spine Journal presented cervical spondylosis as a rare but curable cause of vertebrobasilar insufficiency [3]. Chronic repetitive mechanical irritation may also injure the arterial wall and promote intimal damage, thrombus formation and distal embolization, and irritation of the periarterial sympathetic plexus has been suggested to provoke vasospasm, although this mechanism is less well established.

Epidemiological data support an association. In a nationwide, population-based cohort from Taiwan that followed 27,990 patients with cervical spondylosis and the same number of matched controls for about six years, patients with cervical spondylosis were 1.46 times more likely to develop posterior circulation ischemic stroke, with a hazard ratio of 1.50 in those with myelopathy and 1.43 in those without it [4]. The same study reported no significant increase in risk after anterior decompression surgery but a markedly higher risk, of 4.23, after posterior decompression [4]. A more recent retrospective review of ischemic stroke patients likewise reported a higher burden of cervical disc degeneration in stroke patients than in comparison groups [5]. These findings must be interpreted with caution. Observational studies cannot exclude shared

risk factors such as age, hypertension, diabetes and smoking, which promote both vascular and degenerative disease, and administrative databases cannot show that a given stroke was caused mechanically by a given spinal level. The association is nevertheless consistent enough to justify attention in clinical practice.

Recognition depends on a careful history. Symptoms suggesting vertebrobasilar ischemia include vertigo, diplopia, dysarthria, drop attacks, nausea, ataxia, visual field disturbances and transient loss of consciousness, especially when they are provoked by turning or extending the head, and they should never be attributed to cervical osteochondrosis alone until stroke has been excluded. Because posterior circulation strokes are easily missed on non-contrast computed tomography, patients with acute vertigo or gait imbalance and vascular risk factors require magnetic resonance imaging with diffusion-weighted sequences, together with vascular imaging of the neck and intracranial arteries by CT angiography, magnetic resonance angiography or duplex ultrasonography [1]. When positional symptoms are present, dynamic imaging of the vertebral arteries in neutral and rotated positions can demonstrate transient occlusion, and radiographs or MRI of the cervical spine show the level of foraminal narrowing. Acute treatment of confirmed stroke follows standard guidelines, including timely thrombolysis or thrombectomy when indicated, and is not modified by the presence of spondylosis [1].

Prevention rests mainly on control of established vascular risk factors: blood pressure, glycemia, lipids, smoking cessation, physical activity and antithrombotic therapy according to the mechanism of the event [1]. For patients with symptomatic spinal compression of a vertebral artery, conservative measures such as avoidance of provoking neck positions, a cervical collar for short periods and physiotherapy are used first, and surgical decompression or stabilization is reserved for carefully selected cases with a demonstrated hemodynamic mechanism [3]. Manual therapy deserves particular caution. Forceful manipulation and sudden neck rotation in patients with degenerative

disease and vascular symptoms may injure the vertebral artery and should be avoided until vascular imaging has excluded significant pathology. Patient education is equally important: people with neck pain and dizziness should be advised to seek urgent care if new neurological symptoms appear, because early treatment determines outcome.

In conclusion, cervical osteochondrosis is associated with an increased risk of posterior circulation ischemic stroke, and several anatomical and hemodynamic mechanisms make this association biologically plausible, although a direct causal role has been demonstrated only in a minority of patients. Clinicians should consider vertebrobasilar ischemia in patients with positional dizziness or brainstem symptoms, should image the vessels as well as the spine, and should combine rigorous vascular risk control with cautious management of spinal disease. Prospective studies that use standardized imaging of both the cervical spine and the vertebral arteries are needed to define which patients are truly at risk and which treatments reduce that risk.

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