Is the smaller of paired arteries more vulnerable to occlusion? Vertebral Artery Hypoplasia.

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Although vertebral artery hypoplasia (VAH) is not currently considered an independent stroke risk factor, emerging evidence suggest that may contribute to ischemic events, especially when other risk factors coexist. In the present review we thoroughly discuss the relationship between VAH and posterior cerebral ischemia.

Imaging studies reveal reduced blood flow ipsilateral to the hypoplastic vertebral artery, despite difficulties and controversies

The prevalence of VAH is roughly estimated from 1.9% to 11.6%, as neither consensus on a standardized measuring system nor on the cut-off diameter (range 2.0 mm-3.0 mm) for the VAH has been achieved to date, therefore the comparison of data from different research groups is difficult if not impossible. However, results from two different ultrasound protocols indicate that right sided VAH is twice as common as left sided VAH. Another serious obstacle for standard angiographic techniques that leads to VAH underdiagnosis is the difficulty in differentiation between congenital variants and secondary thrombosis.

Severe unilateral hypoplasia was detected in 5.3% of the total study population and in 12.3% of the hypoplastic subgroup in a MRI scanning protocol of patients with cervical pain. In a retrospective analysis of color Doppler ultrasonography data, healthy subjects with VAH had significantly lower mean flow volume (MFV) in the hypoplastic VA and slightly increased MFV in the contralateral one, when compared...
to those without VAH. The mean net flow volume (MnFV) -the sum of the MFV of bilateral VA- was significantly lower, while the prevalence of VA flow volume insufficiency was respectively higher in subjects with unilateral VAH. Moreover, both the MFV and the MnFV were found to have a strong positive relation with the diameter of the VA.

Case reports indicate that vertebral artery hypoplasia may lead to posterior circulation ischemia, when combined with conventional risk factors

Giannopoulos et al have reported three cases of young adults (average age 38 years) with lateral medullar ischemic events associated with ipsilateral hypoplastic vertebral artery on MRA. All three patients had two additional atherosclerotic or non-atherosclerotic risk factors for stroke, suggesting that vertebral artery hypoplasia combined with other conventional risk factors may provide an optimal background for brainstem ischemia.

Locked-in syndrome due to brainstem infarction has been reported by Orimo et al in a 36 year old male with hypertension and hypercholesterolemia. Brain MRI imaging showed pons, medulla oblongata and right cerebellum infraction, while cerebral angiography revealed bilateral vertebral artery hypoplasia, a persistent right primitive trigeminal artery (PTA) and retrograde blood flow of basilar artery from the PTA.

Recently, Tai et al have presented a case of bilateral medial medullary infarction in a 60-year-old man with VAH, uncontrolled hypertension, hypercholesterolemia and a single episode of transient ischemic attack 15 years ago. MRA confirmed the presence of a hypoplastic left vertebral artery with atherosclerotic changes, occlusion after the posterior inferior cerebral artery (PICA) and stenosis of the proximal basilar artery. Bilateral hypoplasia, an uncommon vertebral artery anomaly with an estimated frequency of 0.75%, has also been revealed in a 67 old female patient with cerebellar infarction by Mestan et al. However, the angiogram in this particular case report failed to display any evidence of vertebral atherosclerotic occlusive disease.

Large cohort studies confirm the case report assumption

Results from a large multicenter study suggested that cerebellar infarction was surprisingly more strongly related with vertebral artery abnormalities compared to the abnormalities of cerebellar arteries that distribute directly to the ischemic areas. More specifically, two thirds of the patients with superior cerebral artery (SCA) or anterior inferior cerebral artery (AICA) infarct had unilateral vertebral artery occlusion or severe stenosis.

A retrospective analysis of 529 patients with ischemic stroke by Park et al demonstrated that even though VAH is a common finding in the asymptomatic population, it is highly associated with posterior circulation strokes (PCS). Perren et al confirmed that among 725 first-ever stroke patients, those with PCS had significantly more frequent VAH (13%) compared to those with stroke in other territories (4.6%). Similarly, Chuang et al reported that the overall incidence of unilateral VAH, measured in a study group of 191 acute ischemic stroke patients, was 11.5% and this percentage was significantly higher in cases of brainstem or cerebellar infarction. More than half of the study group patients had a
single vascular risk factor and 79.4% of them had an additional vascular risk factor.11

Both studies by Park et al and Chuang et al converge that the majority of patients with VAH related ischemic stroke is based on large-artery atherosclerosis. A hypoplastic vertebral artery, because of its decreased flow volume and flow velocities, could presumably be more susceptible to prothrombotic or atherosclerotic processes than normal vertebral arteries11,12. Moreover, blood rheology at a low shear rate was found significantly impaired in symptomatic patients with VAH compared to those VAH patients who were asymptomatic, providing evidence that altered blood rheology in the vertebral artery could be associated with posterior cerebral ischemia.14

Finally, VAH has also been related with an increased probability of stenosis/occlusion of the distal intracranial portion of the ipsilateral VA and also with basilar artery hypoplasia or stenosis, which further increase the risk of posterior circulation ischemia.

Conclusion

The afore mentioned may explain why the smaller of paired arteries are more vulnerable to occlusion, especially if vascular factors are present, and further support the view that arterial size does matter indeed.16

Therefore the posterior circulation is presumably more vulnerable to ischemia in patients with VAH, particularly in those with severe hypoplasia. Most of these individuals remain asymptomatic, but the stroke risk increases further when additional atherosclerotic factors coexist. It should be emphasized that vertebral hypoplasia not only associates with a decrease in blood flow, but also with the progress of atherosclerotic or prothrombotic changes, which finally both contribute to cerebral ischemic events (figure 1).

References