ROLE OF VERTEBRAL HYPOPLASIA IN POSTERIOR ISCHEMIC STROKE

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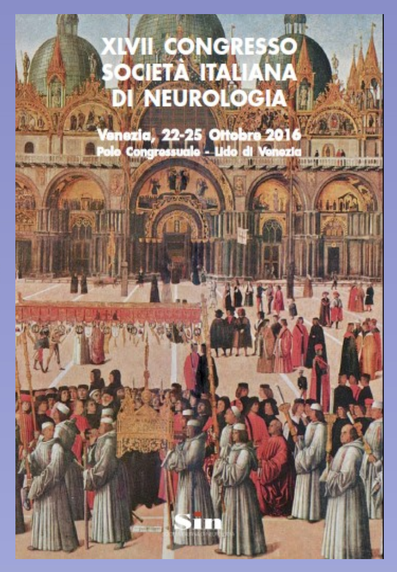

INTRODUCTION

Vertebral artery hypoplasia (VAH) is a common congenital anatomical variation, which has been reported in approximately **10% of normal individuals**; although often regarded as a clinically meaningless finding, in recent years increasing evidence is suggesting that VAH might contribute to posterior circulation ischaemic events. Moreover, it has been hypothesized that the unequally mechanical forces resulting from asymmetric VA flow might cause morphological alterations in the vertebrobasilar arterial system and asymmetrically induce the development of infarcts before or after the vertebrobasilar junction[1]. This study aimed to investigate the prevalence and pathogenetic role of VAH among ischemic events occurring in territories supplied by the vertebro-basilar system.

PATIENTS & METHODS

We have analyzed all patients admitted to our **Stroke Unit over a six-year period (2009-2015) with a documented posterior acute ischemic lesion**. Brain lesions were categorized as proximal, middle, and distal intracranial posterior circulation territories according to the topographic classification described by Caplan et al.[2] The patients were then divided into two groups based on the presence of VAH (VAH+, VAH-), which was **first evaluated by ultrasound** (defined as a diameter smaller than 2,5 mm with a compensatory controlateral caliber, but within normal range velocity values) and then **confirmed by MRA/CTA/DSA**. We analyzed the clinical outcome, risk profile and etiology of posterior strokes in the two study groups (VAH+, VAH-).

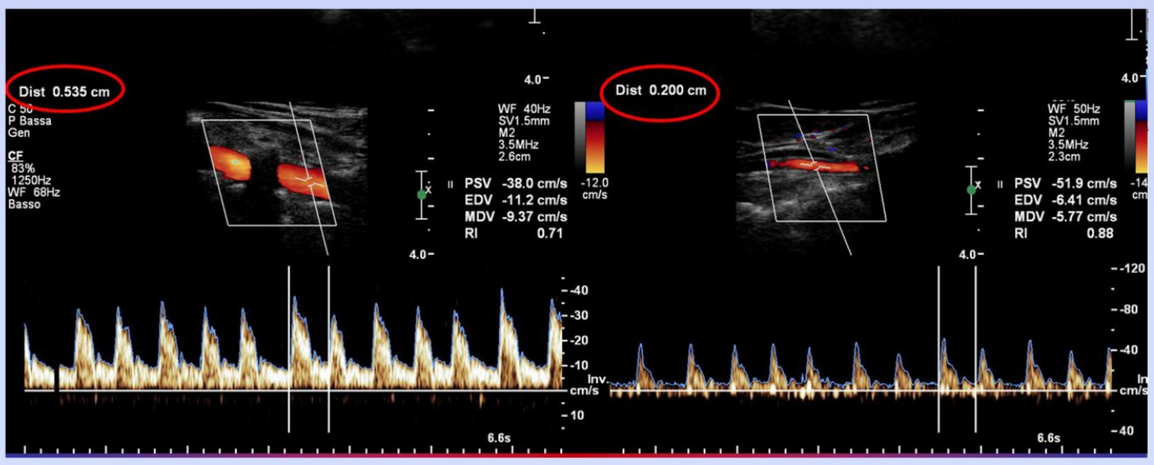

Figure 1. US findings of VAH: reduced diameter (<2.5 mm) along the entire course of the vessel with reduced diastolic velocity and increased controlateral diameter (> 4mm) and normal velocity.

RESULTS

Out of 156 posterior circulation strokes, **VAH was present in 54 (34,6%) cases** (Table 1). VAH of the right artery was almost three times as represented as compared to left artery (74,1% vs 25,9%), confirming previous reports; it has been postulated that since the left subclavian artery branches directly from the aortic arch it undergoes higher shear stress during development, potentially leading to a left VA dominance in the posterior circulation[3].

The two subgroups of patients did not present significant differences in the distribution of gender, age, clinical outcome and stroke subtype. A smoking history was significantly associated with VAH ($p < 0.001$).

Posterior ischemic strokes occurring in the presence of vertebral hypoplasia were located in a **more proximal territory ($p < 0,01$)** compared to the VAH- patients who presented mainly with more distal strokes. These findings suggest a hemodynamic/atherotrombotic mechanism in the former and an embolic mechanism in the latter group.

Prevalence of **VAH among ischemic events in the proximal territory** was 27,8%, occurring **more often on the same side**: 11 out of 14 events (Table 2).

CONCLUSION

The present study demonstrates that **vertebral artery hypoplasia is more frequently seen among patients with posterior circulation ischemia than in the general population**. Moreover, **unequal VA flow seems to be an important contributor of proximal posterior circulation ischemic stroke, occurring more often on the same side of VAH.**

	VAH (+)	VAH (-)	
N° of patients (%)	54 (34,6) Right, 40 (74,1) Left, 14 (25,9)	102 (65,4)	
Male, n (%)	37 (68,5)	69 (67,6)	
Mean age, years ± SD	63 ± 13	62 ± 14	
Median mRS score at discharge	1	1	
Proximal territory, n (%)	15 (27,8)	27 (26,5)	
Middle territory, n (%)	24 (44,4)	29 (28,4)	
Distal territory, n (%)	18 (33,3)	62 (60,8)	OR 0,3 [0,2-0,6], **p < 0.01**
Hypertension, n (%)	38 (70,4)	72 (70,6)	
Dyslipidemia, n (%)	22 (40,7)	37 (68,5)	
Diabetes mellitus, n (%)	14 (26,0)	22 (21,6)	
Smoking habit, n (%)	21 (38,9)	15 (14,7)	OR 3,7 [1,7-8,0], **p< 0.001**
Etiopathogenesis:			
Embolic, n (%)	14 (25,9)	35 (34,3)	
Atherothrombosis, n (%)	18 (33,3)	22 (21,6)	
Dissection, n (%)	6 (11,1)	14 (13,7)	
Lacunar, n (%)	4 (7,3)	8 (7,8)	
Other, undetermined, n (%)	12 (22,2)	23 (22,5)	

Table 1. Comparison of patients with and without vertebral artery hypoplasia

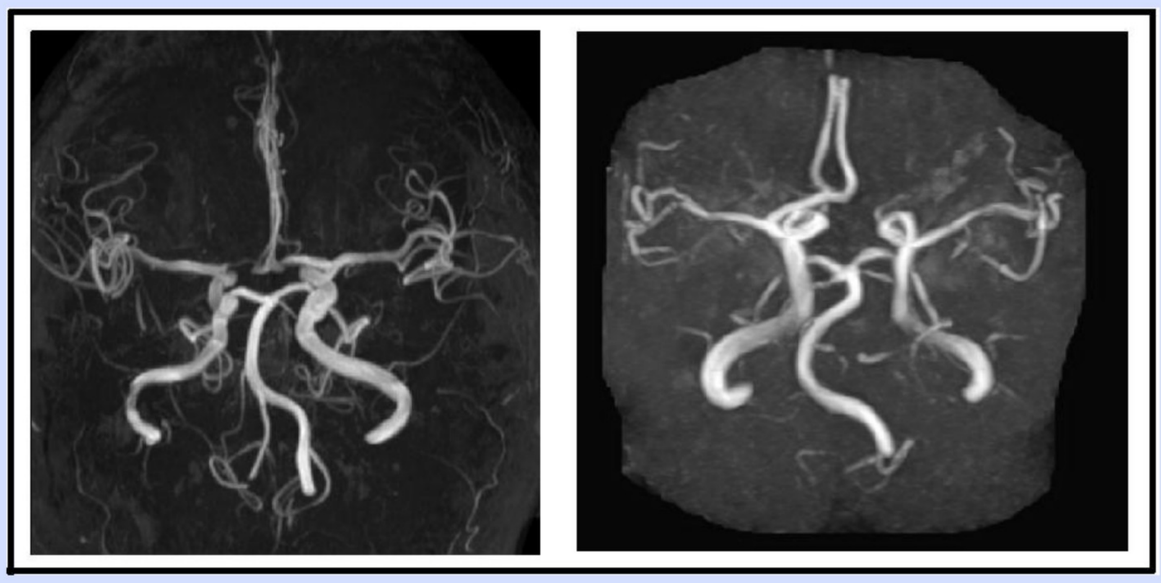

Figure 2. MRA showing VAH of right vertebral artery and lateral displacement of BA.

	Total	VAH ipsylateral to ischemic lesion	VAH contralateral to ischemic lesion
N° of patients, n (%)	49,(100)	29 (59,2)	20 (40,8)
Proximal territory, n(%)	14,(100)	11 (78,6)	4 (21,4)
Middle territory, n (%)	21,(100)	12 (57,1)	9 (42,9)
Distal territory, n (%)	15,(100)	6 (40,0)	9 (60,0)

Table 2. Prevalence of the side of the VAH

REFERENCES

1. *Vertebral artery dominance contributes to basilar artery curvature and peri-vertebrobasilar junctional infarcts; Hong et al, J Neurol Neurosurg Psychiatry (2009);*
2. *New England Medical Center Posterior Circulation registry; Caplan et al, Ann Neurology (2004);*
3. *The vascular territories in the cerebellum and brainstem: CT and MR study; Savoiardo et al, AJNR Am J Neuroradiol. (1987);*