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The double lumen: a pathognomonic angiographic sign of arterial dissection?

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Abstract A case is reported which questions the absolute significance of the "double-lumen sign" in the diagnosis of arterial dissection. I suggest that when demonstrated in isolation, this sign should be interpreted with caution, giving consideration to the possibility of arterial fenestration, and appropriate diagnostic measures implemented.

Key words Dissection, arterial · Duplication, arterial · Fenestration, arterial

Introduction

Intracranial vertebral artery dissection is being recognized with increasing frequency as a cause of subarachnoid hemorrhage [1, 2]. The prognosis in these cases is serious, since rebleeds, with their possibly dire consequences are said to occur in 18 –40 % of cases, usually in the acute stage [1, 3]. Multiple angiographic signs of arterial dissection have been described. Nevertheless, the only signs recognized as pathognomonic are demonstration of the true lumen and the intramural dissection (the "double-lumen" sign) and of vessel dilation in the arterial phase, with retention of contrast medium in the false lumen during the venous phase [2, 4]. The absolute significance of the double lumen sign is questioned by this case report.

Case report

A 40-year-old man developed sudden severe suboccipital headache while riding his bicycle. The headache persisted and became associated with nausea, neck pain and stiffness. CT revealed diffuse subarachnoid hemorrhage and the patient was transferred for neurosurgical care. He was alert and oriented; his blood pressure was 118/70 mm Hg. The right pupil was larger than the left but both reacted to light. Apart from slight neck stiffness the rest of his examination was normal. The patient said the pupillary inequality had been present since childhood; otherwise, his medical history was noncontributory. Complete blood count, electrolytes, blood urea, creatinin, liver function tests, and coagulation profile were all normal. A four-vessel cerebral angiogram was performed, with aortic arch views which revealed normal origins of both common carotid and left vertebral arteries, with a very hypoplastic right vertebral artery. Intracranially, the anterior circulation presented no abnormalities. However, while the left posterior communicating artery was clearly seen, the right was not. Right vertebral artery injection revealed a double lumen extending from the point of dural penetration to the vertebrobasilar junction (VBJ). No right posterior inferior cerebellar artery (PICA) was demonstrated. A common arterial trunk, originating from the basilar artery just distal to the VBJ, seemed to supply the territories of the right anterior inferior cerebellar artery (AICA) and PICA (Fig. 1). The right superior cerebellar artery (SCA) originated from the basilar artery as an upper vessel continuing as the superior vermian artery and an inferior one supplying the territory of the marginal branch. Left vertebral artery injection revealed a dominant vertebral artery (Fig. 2).

With the diagnosis of a ruptured right vertebral artery dissection as the source of the subarachnoid hemorrhage, a right suboccipital exploration was carried out. Instead of evidence of arterial dissection, two normally appearing vertebral arteries, coursing in close apposition with one another, were seen. There was no evi-

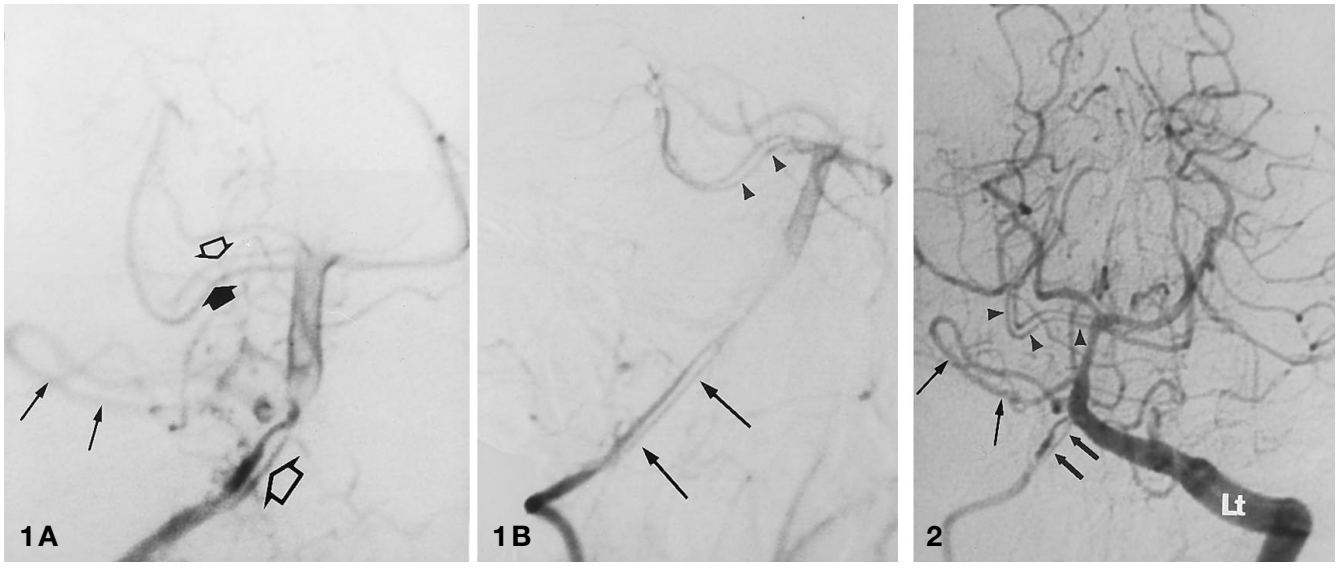

Fig. 1 A, B Right vertebral angiogram. **A** Towne's projection demonstrates the vertebral artery "double lumen" *(large open arrow)* and the common arterial trunk, originating from the basilar artery and supplying the territories of the AICA and PICA *(long arrows)*. The individual origins of the superior vermian and marginal branches directly from the basilar artery are also seen *(small open and solid arrows)*. **B** Oblique projection. The "double lumen" is clearly seen *(solid arrows)*. The independent origin of the superior cerebellar branches is again visualized *(arrowheads)*

Fig. 2 Left vertebral angiogram. Towne's projection. A dominant left vertebral artery is demonstrated *(Lt)* with reflux into the right vertebral artery, again showing the "double lumen" *(large arrows)*. The common arterial trunk supplying the right AICA and PICA territories *(small arrows)* and the origins of the right superior cerebellar arteries from the basilar artery *(arrowhead)* are again seen

dence of a PICA arising from either. The right cerebellar tonsil was supplied by a transdural arterial branch. Repeat four-vessel cerebral angiogram 11 days after the bleed confirmed the findings of the initial study, with a mild degree of vasospasm. The patient's condition continued to improve and he was able to return to his previous occupation without limitations.

Discussion

During brain angiogenesis and thereafter, functional end arteries irrigate constant functional territories. Variations in the origin and course of arteries leading to these end arteries, being only conducting vessels, are compensated for by the development of collateral routes [5, 6].

Embryologically, the basilar artery evolves from the gradual fusion of the two paramedian longitudinal neural arteries, a process completed by the 12 mm embryo stage. The intracranial segment of the vertebral arteries originates from the caudal, still plexiform, segments of these longitudinal neural arteries [7].

The vertebral artery duplication reported herein extended from the point of dural penetration to the VBJ. This vascular segment corresponds precisely to a transient embryological vessel, the accesory vertebrobasilar anastomosis, which normally regresses at the 14 mm embryo stage [7]. It is thus conceivable that persistence of this vessel could have resulted in the vertebral artery duplication as well as in the frequently observed supply of the PICA territory by the AICA. The hypoplastic right cervical vertebral artery, in series with the duplication of its intracranial segment, resulted in an occipitocerebellar pattern of vascularization (proatlantal variant, type I) [6, 8]. Blood was supplied to the right cerebellar tonsil by the transdural arterial branch noted at surgery [9], as previously described in similar circumstances [8]. It was not demonstrated angiographically in the present case because no selective injections were done.

Fenestration or partial duplication of the vertebral artery is found at autopsy, and in angiographic series, with a prevalence of approximately 1 % [10, 11]. About 70 % are in the cervical segment, most at the atlantoaxial level, while the rest occur intracranially [10, 12].

While the majority of vertebral artery fenestrations represent asymptomatic incidental findings, in about 20 % of cases they are detected in association with symptomatic intracranial aneurysms unrelated to the fenestration [11]. In the present case, detailed angiography and inspection at surgery failed to detect an aneurysm as the source of the subarachnoid hemorrhage.

Regarded as a pathognomonic sign of arterial dissection [2, 4], the double-lumen sign consists of seeing contrast medium in the true and false lumina. Its appearances may vary. It can appear as a widened segment of an artery, representing the false lumen, with a much

thinner channel coursing within it, the true lumen. It can also present as a localized bleb-like outpouching of the arterial wall. Finally, a double lumen can be seen as two roughly parallel channels filled with contrast medium along a segment of the affected artery. This last variety may be confused with an arterial duplication, as in the present case. Review of the few reported cases of vertebral dissection exhibiting a double-lumen sign revealed that it is usually seen together with other angiographic signs of dissection, such as irregular, localized widening of the vertebral artery and/or retention of contrast medium at the site of the dissection, in delayed phases. Although angiography in arterial dissection shows dynamic changes which can evolve for up to 3 months [13], this criterion may not be of practical use for diagnostic confirmation in the acute phase, since it can only be applied retrospectively. MRI has proved helpful in diagnosis of arterial dissection [4, 14], but it is not always available or feasible in the acute phase.

The assumption has to be made that this case represents the coincidence of subarachnoid hemorrhage of unknown origin and an asymptomatic vertebral duplication. Differential diagnosis between this and a ruptured vertebral artery dissection may be difficult, and in this case prompted a negative surgical exploration. Demonstration of an isolated double-lumen sign, even in the presence of subarachnoid hemorrhage, should therefore raise the question of arterial duplication. To resolve the issue, MRI should be obtained, since it can provide unequivocal evidence of arterial dissection [4, 14]. If MRI is unavailable or not feasible, a repeat angiogram, tailored specifically to resolving this dilemma, may be done not less than 24–48 h after the first. The disclosure of changed appearances in the area of interest would support the diagnosis of arterial dissection. In the absence of such change, different projections may assist in establishing the diagnosis of duplication, avoiding unnecessary surgery.

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