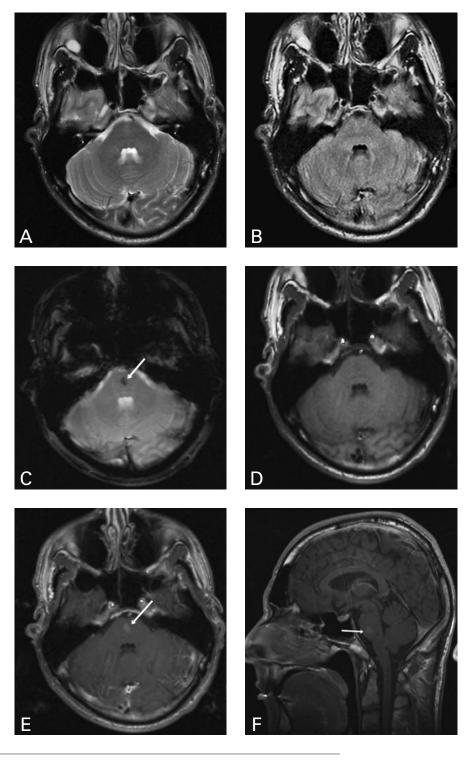
SYMPTOMATIC BRAIN CAPILLARY TELANGIECTASIA

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Key-word: Telangiectasia

Background: A 35-year-old patient presented to our hospital with persistent tinnitus and drowsiness.



1A 1B Fig. 1C 1D

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Work-up

MRI of the brain (Fig. 1) included an axial T2-weighted image (A) on which the lesion is hardly visible. On B (axial FLAIR image) a slightly hyperintense, small, rounded solitary lesion is seen centrally in the pons. On the axial gradient echo image (C), the lesion obviously exhibits hypointensity. On the axial T1-weighted image (D), a iso- to slightly hypointense small, rounded solitary lesion is seen centrally in the pons. Gadolinium-enhanced axial and sagittal T1-weighted image (E, F) shows homogeneous contrast enhancement of the lesion.

Radiological diagnosis

The imaging findings are consistent with *brain* capillary telangiectasia.

Discussion

Brain capillary telangiectasias are part of the vascular malformations of the brain besides pial arteriovenous malformations, cavernomas and venous angiomas or developmental venous anomalies. Brain capillary telangiectasias are not uncommon. They are small vascular lesions (ranging from several mm to 2 cm in diameter), usually located in the pons. Microscopically, they consist of irregular clusters of multiple thin-walled ectatic vascular channels interposed between normal brain parenchyma, without adjacent gliosis or hemosiderin deposition.

The clinical manifestations related to capillary malformations are variable, although typically they are a fortuitous finding on a brain imaging study. Occasionally they are seen in patients presenting with headaches, confusion, weakness, dizziness, visual changes, vertigo, tinnitus, or seizures, but without clear connection to the symptomatology.

Brain capillary telangiectasias have a characteristic MR-imaging appearance that correlates with their microscopic tissue composition. Lacking hemosiderin, capillary telangiectasias are not hypointense on conventional or fast spin-echo T2-weighted images but appear as isointense or hyperintense areas compared with the normal brain parenchyma. In T1-weighted unenhanced images, they exhibit iso- or hypointensity. Because telangiectasias are composed of sacs of stagnant

blood which haemoglobin presumably partially desaturates to deoxyhaemoglobin, they exhibit susceptibility dephasing on gradient-echo images. Being vascular lesions, they show mild contrast enhancement, creating a mesh of enhanced structures on a background of unenhanced brain parenchyma.

Nonenhanced CT does not depict capillary telangiectasias. Since they do enhance they can be visible on contrast-enhanced CT, but they may be overlooked due to insufficient enhancement or to artifacts in the posterior fossa. On angiography they are occult similar to cavernomas because of the lack of arteriovenous shunting.

The differential diagnosis of enhancing lesions in the pons includes neoplasms such as astrocytoma, metastasis or lymphoma, subacute infarction, active demyelination, or acute inflammatory processes such as acute disseminated encephalomyelitis, in addition to capillary telangiectasias. Only the capillary telangiectasias contain blood products, so only these would demonstrate increased susceptibility on GRE-images. Classic capillary telangiectasias can be distinguished from cavernous angiomas on MRI.

Cavernomas, which contain hemosiderin-laden macrophages and often are calcified, show marked signal intensity loss on GRE, called the "blooming effect". Its hallmark is a surrounding hypointense hemosiderin ring with an internal 'popcorn' appearance on T2-weighted images.

In conclusion, brain capillary telangiectasias are small, enhancing lesions that are usually located in the pons and that are often undetectable on conventional T1- and T2-weighted images. They lack the 'hemosiderin rim' of cavernous angioma and appear hypointense on GRE-images.

Bibliography

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