"Top of the basilar" syndrome: A comparison of clinical and MR findings

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Summary. Among 100 patients with an infarction of the brain reported on MR and clinically confirmed there were 4 with widespread lesions of the temporal and occipital lobes, thalamus, midbrain, pons and cerebellum, all supplied by arteries originating around the top of the basilar artery. Clinically these patients presented the "top of the basilar" syndrome, which is caused by a disturbance in circulation at the top of the basilar artery. Which brain areas are involved may be deduced theoretically from the vascular anatomy. These lesions can, we believe, be clearly detected using MR, because of its sensitivity to ischaemic disturbances and in the posterior fossa. We report our 4 patients here to illustrate the clinical presentation and MR findings of the "top of the basilar" syndrome.

Key words: Top of the basilar artery syndrome - Cerebral infarctions - Magnetic resonance

General

The top of the basilar artery (TOB) is the place where the basilar artery divides into the two posterior cerebral arteries (PCA). Disturbances in the circulation in this area can be caused by aneurysm, embolism or thrombosis with partial or total occlusion of the lumen leading to a syndrome to which attention has been drawn lately in the literature [1-3]. Our patients illustrate the clinical presentation of this "top of the basilar" syndrome and their magnetic resonance (MR) findings. For a better comprehension of this matter, we will first discuss the vascular supply in the region and review relevant literature. We will then consider the possible value of MR in this syndrome.

Vascular territories around the top of the basilar

The areas involved after a disturbance in the circulation in the TOB-area can be deduced theoretically from the pattern of vascularization (Fig. 1). The following arteries and brain areas are involved [4-7]:

PCA, supplying the medial part of the temporal lobe (memory) and the occipital lobe (vision). This artery becomes of even greater importance in persons with an incomplete circle of Willis.

Small branches supplying the thalamus. The following branches can be distinguished:

Fig. 1. Vascular supply around the TOB. ICA: internal carotid artery, BA: basilar artery. For further explanation and abbreviations see text (choroidal arteries not shown)
TTA: Thalamotuberal arteries (polar arteries). They arise from the posterior communicating artery (PCoA), and supply the posterior part of the hypothalamus, and the anterior part of the thalamus. These areas contribute to the circuit of Papez, a part of the limbic system. Furthermore the TTA supply the anterior part of the crus posterior of the capsule interna [4], containing efferent motor pathways, especially those innervating the arm. In 50–75% of the cases the TTA are thought to be absent [2], in which case their function is adopted by:

TPA: Thalamoperforate arteries (paramedian arteries). They arise from the first part of the PCA, before merging with the PCoA. By some authors this part is called the basilar communicating artery, or the mesencephalic artery. Its embryonic origin is supposed to differ from the rest of the PCA. The TPA supply the median and paramedian parts of the midbrain, e.g. the cerebral peduncles (efferent motor pathways), the nucleus nervus III (ocular movements) and the reticular formation (arousal). They also supply the medial part of the thalamus. The right and left TPA often communicate or even have a common stem [5, 8].

TGA: Thalamogeniculate arteries. They arise from the PCA after merging with the PCoA and supply the lateral part of the thalamus. The nuclei in this region have, among other things, a sensory function.

PChA: Posterior choroidal arteries, arising from the PCA and supplying the geniculate bodies (auditory and visual relay nuclei), the pulvinar and the dorsolateral thalamus (integration). The PChA anastomose with the AChA (anterior choroidal arteries).

Small branches originating from the rostral part of the basilar artery, supplying the rostral brainstem.

Vascular supply of the cerebellum (motor coordination and equilibrium), the superior cerebellar artery (SCA) in particular.

Literature review

The name TOB-syndrome was coined by Caplan [1] to distinguish a separate entity from the rather vague concept of “vertebro-basilar ischaemia”. The clinical TOB-syndrome includes an array of visual, oculomotor, and behavioral disturbances. Motor disturbances are less conspicuous. Caplan divided the symptoms into two groups. Those resulting from rostral brainstem-infarction, and those resulting from PCA-involvement. He identified his cases by means of CT-scan, angiography or autopsy. Only a number of the patients had full angiography.

Segarra [2] described the syndrome of the mesencephalic artery. His line of investigation goes back to a tradition of investigation of vascular territories of the thalamus, mostly of French origin, and already initiated in 1925 by Foix and Hillemand [9]. The key symptom of the syndrome of the mesencephalic artery was found to be akinetic mutism. The TOB-syndrome has a broader definition, hence it includes not only the syndrome of the mesencephalic artery, but includes symptoms of other vascular territories as well [1].

In 1987 the TOB-syndrome was evaluated by Sato [3]. In his series all patients had to have changes at least on CT-scan, but preferably also on angiography, in the area near the basilar bifurcation. The neurological symptoms were roughly classified according to the state of consciousness and the degree of disablement. It is not certain whether his cases are comparable to the syndrome originally described by Caplan. Of the 13 patients who underwent angiography, 11 (85%) showed stenosis or occlusion within an area with a diameter of 2 cm around the TOB. Other atherosclerotic changes were found in 9 patients, of whom 3 also had a megadoliche basilar artery. In 8 patients (62%) recanalization was thought to have occurred and therefore they were suspected of having had an embolic rather than a thrombotic occlusion. An embolic occlusion was postulated by Caplan as well.

The role of MR

The lesions associated with the TOB-syndrome can be diagnosed with advantage on MR, because of its greater sensitivity in this region [10, 11], where CT-scan suffers from artifacts caused by beam hardening [12]. MR has a greater sensitivity in detecting acute and subacute infarctions in general [13]. Ischaemic areas are shown on MR from 4 hours onwards, and sometimes as early as 30 min [14], the changes being caused by a disturbance in metabolism of the neural tissue, accompanied by cytotoxic and later by vasogenic edema. This results in prolonged relaxation times of the affected tissue and a corresponding change in signal intensity (SI). Up to 6 h T1 is strongly prolonged, leading to a low SI on T1-weighed (T1W) images. Later T1 prolongation decreases, leading to an intermediate SI. In both instants there is a marked increase of T2, leading to a high signal on T2W images. Late (chronic) changes in signal are caused by replacement of the normal brain tissue by repair tissue (gliosis) or even by cyst formation. Pure gliosis results in a low SI on T1W, but is often accompanied by a high SI on T2W images because of the increase in extracellular water.