Neurovascular compression syndrome

The pathophysiology of neurovascular compression syndrome has not been fully elucidated.

Over the course of decades, Neurovascular compression (NVC) has become established as the cause of nearly all cases of classic or typical trigeminal neuralgia, HFS, and glossopharyngeal neuralgia (GPN) when strict diagnostic criteria are applied. These conditions are also amenable to high rates of cure by the atraumatic alleviation of culprit vascular compression via Jannetta's MVD procedure. However, the NVC theory has also been invoked by some as a potential cause of other conditions such as non-episodic facial pain (e.g., type 2 TN), Bell's palsy, tinnitus, vertigo, hypertension, and diabetes.

see also Hemilaryngopharyngeal spasm.

Evidence of an association between these conditions and culprit vascular compression is less robust, and MVD surgery has not been generally accepted as effective in their treatment. There remains an appropriately great burden of proof when proposing an NVC etiology, just as Jannetta faced [1].

The symptoms of trigeminal neuralgia are believed to arise from neurovascular compression near the trigeminal root entry zone of the trigeminal nerve; however, this still remains theory [2] [3].

A 72-year-old -female presented with trigeminal neuralgia (TN) and radiological evidence of neurovascular compression on the affected side. She had complete resolution of her pain for 7 years after treatment with GKRS. The patient experienced recurrence and underwent repeat GKRS, this time resulting in another 3 years of pain relief. After the second recurrence, repeat intracranial imaging demonstrated resolution of neurovascular compression.

GKRS is an important treatment option for TN, although the mechanisms behind pain relief from this procedure still remain unclear. While prior histological and radiological studies point to ablative mechanisms for pain relief, this case report suggests that GKRS may result in a decompressive effect in TN due to changes in neurovascular architecture. Despite this finding, TN is known to occur and recur in the absence of neurovascular compression; thus, further work is necessary to understand the etiology of TN and its treatments.

In this case, Moosa et al. demonstrated that vessel-nerve relationships may change over time in TN patients treated with GKRS, which raises the possibility that GKRS could release a neurovascular compression [4].

A case of posterior cerebral artery aneurysm, which caused the isolated trigeminal neuralgia in a 48-year-old woman, was operated on through microvascular decompression and the aneurysm was wrapped [5].

S100B in Neurovascular compression syndrome

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