more branches of the trigeminal nerve. Light tactile stimulation may trigger such an attack. Although the contribution of central and peripheral mechanisms to the aetiopathogenesis of trigeminal neuralgia still remains unclear, the concept of vascular compression of the trigeminal root as the main causative factor in idiopathic “tic douloureux” has achieved widespread acceptance. Trigeminal neuralgia may also afflict patients with multiple sclerosis. In these cases, the demyelination of central trigeminal pathways is the accepted aetiology, and the presence of a T2 hyperintensity along the intrapontine course of trigeminal fibres is generally considered a contributory factor to microvascular decompression. We recently saw a case of successful microvascular decompression in a patient without multiple sclerosis, despite an intrapontine trigeminal lesion.

This 66 year old previously healthy man presented with a 6 year history of intense, paroxysmal, electric shock-like pain in the territory of the second branch of the right trigeminal nerve. The pain was triggered by washing his face and shaving and it lasted for a few seconds. Painful attacks, initially rare, gradually increased in frequency and intensity and spread to the first trigeminal branch. When admitted to our hospital 1200 mg carbamazepine was ineffective in relieving the pain. Before admission phenitoin, baclofen, and lamotrigine had been tried without success. Neurological examination was negative and in particular there were no gross sensory deficits in his right trigeminal territory. Magnetic resonance imaging showed a T2 hyperintensity of intrapontine trigeminal fibres and nucleus (fig 1), without evidence of vascular conflicts with the trigeminal root entry zone. A controlateral, smaller, symmetric lesion was also evident. Multiple sclerosis and Lyme disease were ruled out by clinical history and appropriate investigations. The patient had no vascular risk factors. Holter monitoring, neck vessels colour echo Doppler, transthoracic echocardiographic examination, and brain MR angiography did not disclose alterations suggesting a possible ischaemic origin.

At operation, performed through a keyhole retrosigmoid craniectomy, the root entry zone of the nerve was found crossed by an “intratrigeminal” vein, which was electrocoagulated and divided. No other vascular contacts could be detected by careful exploration of the intracystern tract of the nerve. The postoperative course was uneventful. Paroxysmal pain slowly faded away during subsequent weeks. Carbamazepine could be completely withdrawn after 2 months.

This case is intriguing for two reasons: firstly, because we obtained an apparently paradoxical therapeutic answer in a case where microvascular compression should have been generally contraindicated; secondly, because of the rarity of the T2 hyperintensity of intrapontine trigeminal fibres and nucleus in patients without multiple sclerosis.

Although the intimate aetiopathogenetic mechanisms of trigeminal neuralgia still remain unknown, peripheral lesions affecting the trigeminal nerve entry zone (tortuous vessels, meningoepulmonary aneurysms, arteriovenous malformations, lipomas, epidermoid cysts, oseomas, etc) and multiple sclerosis are certainly involved in the mechanisms causing paroxysmal pain. Demyelination of trigeminal fibres at the level of trigeminal root entry zone in case of vascular compression and demyelination of intrapontine trigeminal fibres in case of multiple sclerosis (personal observation of MRI in more than 80% of cases) may result in ephaptic, abnormal transmission of impulses. A neuropathological origin was surgically and pathologically confirmed in a patient with multiple sclerosis and trigeminal neuralgia by Lazar and Kirkpatrick.1 The anapathological evidence of demyelination of intrapontine trigeminal fibres in a patient with multiple sclerosis and trigeminal neuralgia was provided by Crooks and Miles.2

Recent studies have hypothesised that vascular compression (and possible consequent demyelination) of the trigeminal root and demyelination of intrapontine trigeminal fibres due to multiple sclerosis can coexist and perhaps cooperate in provoking pain paroxysms. Hence, the classic distinction between the supposed “intracranial” mechanism for trigeminal neuralgia associated with multiple sclerosis and the “all peripheral” mechanism for the trigeminal neuralgia related to vascular compression should be overcome in favour of a unique (at least with trigeminal neuralgia and multiple sclerosis are included), mixed central-peripheral mechanism in which abnormal impulses coming from demyelinated axons (multiple sclerosis, vascular compression, and any other possible cause of demyelination along the central and the peripheral course of gasserian ganglion fibres) modulate the nuclear activity.

An alternative view of this case might be the one advocating the hypothesis that pain relief might be due to surgical damage to the trigeminal root. This view has been strongly challenged by the results of recent studies on sensory effects of microvascular decompression. In our patient, too, no evidence of sensory deficits could be found postoperatively.

Generally a minimum myelin damage, without any gross nerve hypofunction, is involved in the aetio-pathogenesis of trigeminal neuralgia. In rare cases demyelination is so widespread along trigeminal fibres to be visualised by MRI and only patients with multiple sclerosis show the classic T2 hyperintensity along the intra-axial trigeminal pathways. So the best of our knowledge this case is the third reported in which such a lesion was found in a patient without multiple sclerosis.

In the two previous cases1,2 a pontine ischaemia was supposed: in one, multiple cerebral ischaemic lesions with widespread cortical atrophy were found; in the other the pontine lesion was small but an MRI scan showed a focal stenosis of the vertebral artery. A few patients with small pontine infarcts were reported with trigeminal symptoms, but without paroxysmal pain.

In our case the MRI evident T2 hyperintensity of intrapontine trigeminal pathways was considered a demyelinating or an ischaemic lesion of unknown aetiology. This case suggests that the presence of a pontine trigeminal lesion is not an absolute contraindication for microvascular decompression in cases of drug resistant typical trigeminal neuralgia.

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**References**


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**Reversal of tissue hypoxia by a single intraventricular dose of sodium nitroprusside in a patient with severe medically refractory cerebral vasospasm after subarachnoid haemorrhage**

A 29 year old man was referred to our department from a local hospital in November 2000, for treatment of acute subarachnoid haemorrhage. At examination, he presented with stupor and a Glasgow coma scale score of 7 and was intubated and artificially ventilated. Diagnostic angiography was performed the same day and demonstrated a ruptured anterior cerebral artery aneurysm. The complex configuration of the aneurysm precluded embolisation as a treatment option. Surgery was
performed the next day and the aneurysm was successfully clipped. After surgery, the patient received the calcium channel blocker nifedipine intravenously at a dosage of 2 mg/hour and moderate hypertensive haemodilution using isotonic solutions to prevent vasospasm induced brain ischaemia. Transcranial Doppler flow velocities were less than 140 cm/s from day 1 to 5 postoperatively. At day 6, Transcranial Doppler flow values increased up to 200 cm/s indicating severe cerebral vasospasm. Cerebral angiography was performed demonstrating 80% vasospasm on the left internal carotid artery (C1 segment). Because the patient remained in a medically induced coma, a Clark-type intraparenchymal brain tissue oxygen sensor was implanted in the left middle cerebral artery territory to monitor brain tissue oxygenation. Initial values showed tissue hypoxia (tissue oxygen pressure <10 mm Hg), therefore, aggressive hypertensive hypertensive haemodilution therapy was initiated to improve cerebral circulation. After temporary improvement, the next day brain tissue oxygen pressure decreased below 5 mm Hg and emergency endovascular balloon dilatation of the C1 segment of the internal carotid artery was performed. Again, tissue oxygen improved temporarly but critical tissue hypoxia developed 4 hours after balloon dilatation. Brain CT was performed and showed no signs of established cerebral infarction. At a brain tissue oxygen value of 2 mm Hg, a total dose of 40 mg sodium nitroprusside was administered over a period of 30 minutes via the ventricular catheter. The dosage was chosen from an earlier clinical report. Seventy minutes after administration of a single dose of 40 mg sodium nitroprusside, cerebral oxygenation improved permanently.

Figure 1 Left frontal brain tissue oxygen pressure (pTIO₂), mean arterial pressure (MAP), and intracranial pressure (ICP) in a patient with cerebral vasospasm after subarachnoid haemorrhage. Low pTIO₂ values indicating critical brain hypoxia prompted emergency endovascular balloon dilatation of the vasospastic C1 segment of the left internal carotid artery, which only temporarily improved cerebral oxygenation. After intraventricular administration of a single dose of 40 mg sodium nitroprusside, cerebral oxygenation improved permanently.

References

Transcranial magnetic stimulation alleviates truncal ataxia in spinocerebellar degeneration

Spinocerebellar degeneration is an inherited or acquired neurodegenerative disorder characterised by steadily progressive cerebellar ataxia, dysarthria, and gait disturbance. These symptoms restrict daily activities. However, no satisfactory therapy has been established. Transcranial magnetic stimulation (TMS), originally introduced to the medical field to evaluate the function of the CNS, is recently becoming a therapeutic tool for neuropsychiatric disorders, such as major depression1 and Parkinson’s disease.2 We also reported the efficacy of TMS for inherited spinocerebellar