

## **Trigeminal neuralgia due to pontine infarction: A case report**

**V. Pizza\***, **E. Lamaida<sup>^</sup>**, **A. Agresta\***, **G. Bandieramonte<sup>o</sup>**, **A. Volpe<sup>o</sup>**, **R. Galasso<sup>o</sup>**, **L. Galasso<sup>o</sup>**,  
**A. Bisogno<sup>^</sup>**, **A. Capasso<sup>^</sup>**.

\*Neurophysiopathology, <sup>^</sup> Neurosurgery and <sup>o</sup> Neuroradiology Unit, S. Luca Hospital, Vallo della Lucania (Sa), <sup>^</sup> Department of Pharmaceutical Science, University of Salerno, Italy

### **Abstract**

**Brain stem lesions are known to cause facial pain. But, trigeminal neuralgia (TN) due to ischemic lesion of the pons are very rare. We report a case with a pontine infarct at the root entry zone (REZ) of the right trigeminal nerve resulting with a classical trigeminal neuralgia. C.M.A. is an old woman (75 years) that suddenly presented an acute symptomatology typical of classical trigeminal neuralgia. Neurological examination revealed only slight hypoesthesia on the right V2 dermatomes. There was a trigger point at the right upper lip. Magnetic Resonance cranial investigation showed a hyperintensity in the FLAIR and DP/T2 and a hypointensity in T1 sequences latero-pontine dx, at the root entry zone (REZ) of the right trigeminal nerve. The patient was treated with pregabalin at dosage of 150 mg/die/os in two administrations and antiaggregant drugs. The resolution of symptomatology was registered later three weeks. Isolated trigeminal neuralgia is a rare debut form of pontine infarct and the mechanism of TN in these cases is still a matter of debate. Central theories on the etiology of TN mainly focus on the increased neuronal activity in the trigeminal nucleus. The treatment of secondary TN is a symptomatic control of pain with anti-epileptic agents. Besides, in a patient with pontine infarct, prevention of further strokes should be done. In our patient was obtained a complete resolution of the symptomatology with pregabalin and was effected a preventive stroke therapy with acetylsalicylic acid.**

**Key words:** Trigeminal Neuralgia, Pontine Infarction, Ischemic Lesion

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### **Introduction**

Trigeminal neuralgia has an annual incidence of 4.5 per 100000 cases. It is characterised by recurrent episodes of intense lancinating pain in the distribution region of mandibular and jaw (rarely opthalmic) rami. The onset is usually in middle age and later life, but young adults and children can be affected too. Rarely attacks last more than few seconds or 1-2 minutes, but they may recur repeatedly within few seconds, so intensely that the patient gives unintentionally a start, and from this the term "tic douloureux" [1-5].

Often, but not always, they are precipitated by mild stimuli applied to particular area of the face (so-called trigger zones), sometimes located anywhere within the territory of the affected nerve, such as eating, drinking, washing, shaving etc.

As a rule, a summation of spatial and temporal impulses followed by a refractory period of 2-3 minutes is necessary to precipitate attacks. This mechanism, typical of

other neuropathic pains, suggests a pathogenesis included in the field of allodynia [1-5].

In most cases trigeminal neuralgia has no defined cause, other times it is provoked by the compression of the trigeminal nerve root, usually within a few millimetres of entry into the pons, i.e. the root entry zone. In a few cases, trigeminal neuralgia is due to a demyelinating disorder. Other rare causes include infiltration of the nerve root, the gasserian ganglion or the nerve by a tumour or amyloid. Rarer are cases due to small infarcts or angiomas in the pons or medulla.

Finally, once all of these possibilities have been excluded, there remains a small proportion of patients in whom the aetiology is undetermined [1-5].

Here, we report the case of a patient affected by a pontine infarction localized in the root entry zone of the fifth cranial nerve, who referred a typical trigeminal neuralgia as initial symptom.

### Case Report

C.M.A is a 75 year-old woman suffering from high blood pressure and heart complaint, who has suddenly presented a clinical symptomatology typical of a trigeminal neuralgia at her right hemiface. The neurological exam has showed a mild hypoesthesia in the territory of the second branch of right trigeminal nerve, with trigger points located at the corner of her upper lip. Magnetic Resonance of the encephalon has showed a focus of altered signal, hyperintense on DP/T2 and FLAIR sequences and hypointense on T1 sequence, at lateropontine level, in the



Fig. 1

**Figure 1:** Axial FLAIR: focus of hyperintensity, coarsely ovalar, in the lateral right pontine portion, extended to the REZ of cranial nerve V.

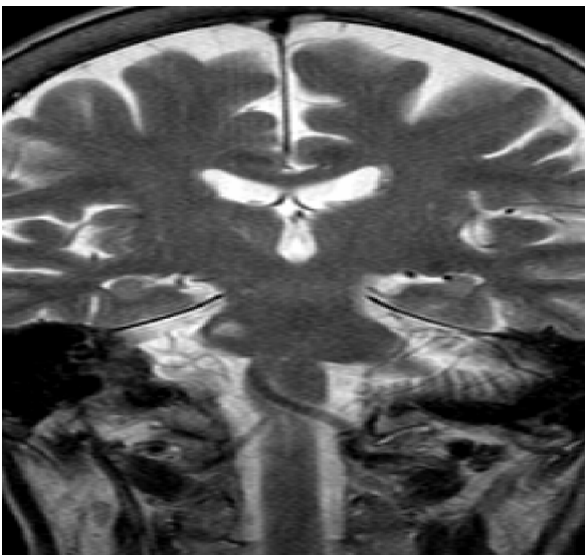


Fig. 2

**Figure 2:** Coronal FSE T2: the presence of a focus of hyperintensity of the right pontine hemisection is confirmed

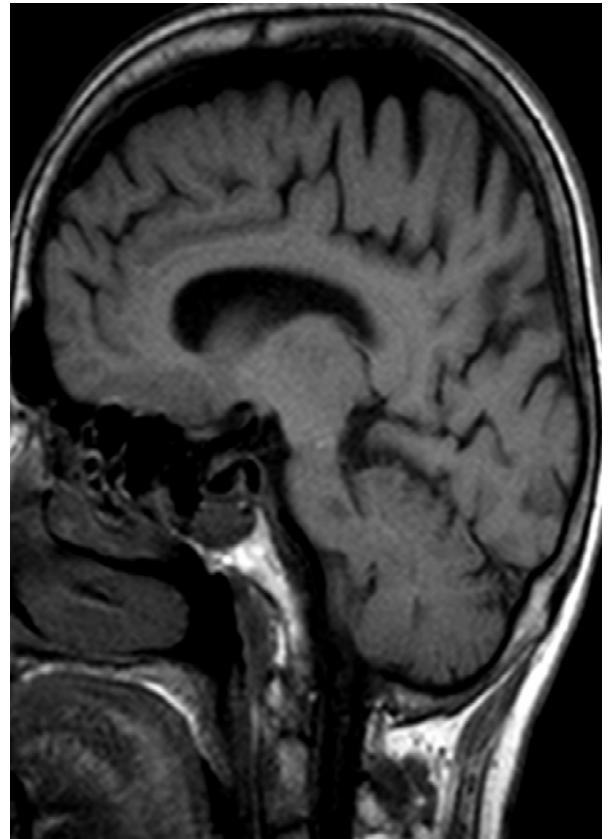


Fig. 3

**Figure 3:** Sagittal FSE T1: the focus of altered signal appears hypointense in this sequence

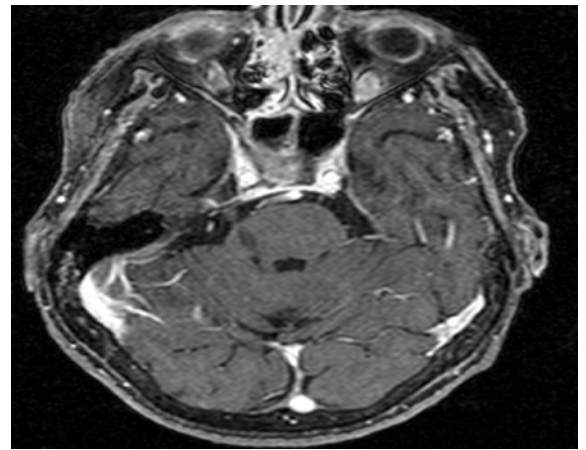


Fig.4

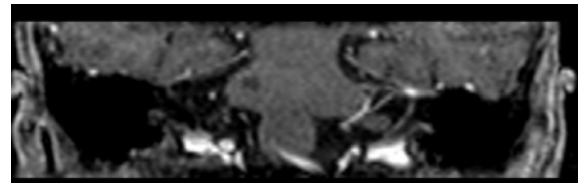


Fig. 5

**Figures 4 –5** Axial (04) and coronal (05) FSE T1 with contrast medium: no significant intensification of signal after contrast medium injection.

origin of the V cranial nerve, of oblong morphology, extended from the REZ to the lateral recess of the IVth ventricle (Figures 1-5). The patient has been subjected to a therapy with Pregabalin at an oral dosage of 150 mg/day in two administrations combined with antiaggregant drugs (acetylsalicylic acid, 300 mg/day). The complete resolution of the symptomatology has been achieved within 3 weeks of initiating therapy.

## Discussion

Rarely the initial symptom of an ischaemic pontine lesion is an isolated trigeminal neuralgia. The physiopathogenetical mechanism of trigeminal neuralgia is still a matter of debate, with absolutely inconclusive results. Demyelination of sensory fibers is usually thought to underlie trigeminal neuralgia, implicating in most cases the roots of nerve and being often precipitated by a vascular compression. Other causes of trigeminal neuralgia in which demyelination is present include multiple sclerosis and probably masses occupying the posterior cranial fossa space. The examination of trigeminal nerves roots of patients affected by vascular compression has highlighted focal demyelination in the region of compression, with a narrow apposition of demyelinated axons and an absence of intervening glial processes. A juxtaposition of axons and foci of nerve root demyelination have been demonstrated also in multiple sclerosis patients with trigeminal neuralgia [1-5].

Experimental studies indicate that these arrangements favour the ectopic generation of spontaneous nerve impulses and their ephaptic conduction to adjacent fibres, and that spontaneous nerve activity is strictly correlated to lesions associated with pulsatile vascular activity [1-5].

Decompression of nerve roots produces a rapid relief of symptoms in most patients with trigeminal neuralgia associated to vascular compression, probably because the resulting separation of demyelinated axons reduces their spontaneous activation and their ephaptic spread [1-5].

The role of remyelination in the initial symptomatic recovery after decompression is still unclear. However, remyelination may help to ensure that relief of symptoms is due to decompression, that may also be responsible for the spontaneous remission of the neuralgia [1-5].

By promoting a rapid recovery of nerve conduction, vascular decompression positively reflect on myelinated fibres outside the region of demyelination. Trigeminal neuralgia can occur in association with several other syndromes apart from vascular compression and even in this field clinical and electrophysiological observations sup-

port the concept that demyelination and ephaptic spread underlie the pathological event [1-5].

Therefore, a central point is represented by an enhanced neural activity in the nucleus of the fifth cranial nerve. Symptomatic treatment of these forms is based on the administration of antiepileptic drugs able to control pain, such as phenytoin, clonazepam, carbamazepine, gabapentin and more recently pregabalin,. In vascular forms rarely observed few data are available to estimate the efficacy of these drugs. In that sense our report appears to be greatly interesting, as a therapy with pregabalin has been conducted together with an antiaggregant treatment based on acetylsalicylic acid administration. In our case we have noticed an excellent response to pregabalin, with a rapid complete remission of symptoms, thus integrating and supporting the physiopathological introductory statements discussed.

## References

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## Correspondence:

Anna Capasso  
Department of Pharmaceutical Sciences  
University of Salerno, Via Ponte Don Melillo (84084)  
Fisciano, Salerno, Italy  
E-mail: annacap@unisa.it