Sphenopalatine Ganglion Block for Management of Painful Idiopathic Orbital Inflammatory Syndrome: A Case Report

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Introduction

Idiopathic Orbital Inflammatory Syndrome (IOIS) is a chronic disease characterized by the presence of flogistic and even immature osteoid tissue inside of orbita. Patients present proptosis, edema, orbital and facial pain, and redness. Proptosis usually grows with the degree of inflammation fibrosis, and mass effect. It is possible to find optic neuropathy, alterations in the motility of the eye, ptosis. Patients may also experience headaches, asthenia, and general malaise. The diagnosis is based on anamnesis, clinical characteristics, exclusion of other pathologies. Orbital Magnetic Resonance Imaging (MRI) and TC are the most important diagnostic tests. Biopsy is rarely practiced mainly due to the low risk-benefit ratio; the possibility of injury to the orbital and periorbital anatomical structures is high. The main therapy is represented by corticosteroids, administered for a long time until the improvement or remission of symptoms [1].

The Sphenopalatine ganglion (GSP) is a complex anatomical structure, present bilaterally within the facial massif. Its fibers provide sensitivity to various structures and regulate their secretory and motor activity. We refer to: Nasopharyngeal and oropharyngeal mucosa, lacrimal glands, oral cavity in its upper region and nasal cavity. The fibers of the sphenopalatine ganglion also innervate the meninges and cerebral vessels.

Specifically, the sphenopalatine ganglion is located behind the lateral insertion of the middle nasal concha and is covered with a thin layer of mucosa in the pterygopalatine fossa. The latter, together with the GSP, are anteriorly in relationship with the maxillary sinus; posteriorly with the pterygoid process; medially with the palatine bone; laterally with the pterygomaxillar fissure [2].

Another set of axons that cross the pterygopalatine ganglion is made up of the somatic sensory fibers of the maxillary branch of the trigeminal. The maxillary nerve emits somatic sensory branches cross the pterygopalatine ganglion to form the palatine nerves. They conduct the somatic sensations of the gums, hard and soft palate, oral cavity, tonsils and uvula [3]. Due to its sensory afferents, the sphenopalatine ganglion has been the target of treatment for chronic headache and atypical facial pain syndromes [4].

Case Presentation

We report an interesting case of a patients affected by IOIS with a disabling pain syndrome. She reported orbital and temporal pain, continuous, with a VAS steadily more than 7, insomnia and poor quality of life. At the CT scan of the facial massif the patient presented osteodysplastic alterations involving the small sphenoid wing, the frontal scale, part of the ethmoid labyrinth and the left anterior clinoid with fibrous tissue and immature osteoid. There were also alterations of the orbital pyramid. All the lesions were present on the left, where the painful symptoms were manifested.

The patient had practiced pharmacological therapy with opioids, antiepileptics drug, NSAIs and corticosteroids with very poor results. Upon obtaining informed consent, we proposed to the patient to undergo a cycle of sphenopalatine ganglion anesthetic blocks. We asked the patient to express the number of daily pain attacks, their intensity and duration. We administered the HIT-6 questionnaire to the patient to assess the impact of pain on quality of life. The same information was requested at the end of each therapy cycle. The patient received the treatments at the AORN Monaldi pain therapy department in Naples, as an outpatient procedure. In each session the patient was placed in the supine position with the chin raised; 0.3 ml of 0.5% bupivacaine were administered through the left nostril by means of a special device present in the company for over a year and
already used for treatment of migraine and trigeminal neuralgia. The procedure was practiced in the same way contralaterally. The patient was observed for 30 min and then sent home with an appointment for the next block.

We subjected the patient to sphenopalatine ganglion block once a week for three weeks and repeated the cycle 15 days after the last administration. Fifteen days after the end of treatment, the patient reported a decrease in mean pain (mean VAS 3.6 ± 0.7) and a reduction in the number of daily painful episodes (2 ± 0.5) and in duration (30 ± 25 min). The consumption of NSAIDs was reduced and the quality of life was defined as improving (HIT-6 score from 60 before treatment to 50 after).

Discussion

Ocular inflammation syndrome is a benign but disabling disease. The painful symptoms that often accompany this clinical condition are often very intense, continuous and capable of compromising normal daily activities. In the specific case it was a young patient, active at work and in good health conditions for the rest. However, the frequent painful attacks were unresponsive to all available pharmacological treatments and had led the patient to a depressive state. The analysis of the anatomy of the sphenopalatine ganglion and of its sensory afferents allowed us to hypothesize its block in the management of the orbital inflammatory syndrome. The good response to treatment represented an interesting therapeutic option. Such experience obviously requires further investigation and a large-scale application to validate its effectiveness.

References