AMYLOID TUMOR OF THE GASSERIAN GANGLION

REPORT OF CASE

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Tumors of the gasserian ganglion producing trigeminal neuralgia or trigeminal pain are not common and to our knowledge an amyloid tumor of the gasserian ganglion has not been reported previously.

We should like to report a unique case of pain, paresthesias, and loss of sensation in the face produced by an amyloid infiltration of the gasserian ganglion.

REPORT OF CASE

A 42-year-old man was first seen at the Mayo Clinic on March 28, 1956, with the complaint of pain and numbness in the right side of his face. In 1945 he had consulted a dentist after losing a cap from his right upper incisor tooth. The dentist had induced local anesthesia by injection of the right upper alveolar nerve and repaired the tooth. The numbness of the local anesthesia remained. The next day the patient consulted the dentist who was unable to explain the persistent numbness. The patient was treated with injections of thiamine without benefit. Approximately 2 weeks after the onset of the numbness the patient had noted brief, recurrent, lancinating pains in the anesthetic region. These had occurred usually in the evening. In the following years the lancinating pains had decreased in frequency and severity but the patient noted the presence of a persistent, burning, dull pain in the anesthetic region. The part affected by the numbness had slowly increased in size, particularly in the preceding year. The numbness came to extend to below the right eye and over the entire cheek, and the burning pain became severer.

The results of general physical examination were normal. The abnormal findings were limited to the neurologic examination. The right corneal reflex was greatly reduced and the analgesia and anesthesia involved part of the face supplied by the entire 2nd division of the 5th cranial nerve and the superior portion of the 3rd division. The anesthetic region extended to the midline of the bridge of the nose and the upper lip. Weakness or atrophy of the muscles of the face and jaw was not evident.

Routine examinations of the blood and urine gave normal results. The blood serology test for syphilis was nonreactive. Roentgenograms of the skull, including stereoscopic views of the base of the skull, revealed decalcification of the middle fossa on the right side near the petrous tip. Electromyography revealed the motor-unit potentials in the right masseter muscle to be large in amplitude and reduced in number and occasional fibrillation potentials were observed. This was felt to be indicative of neurogenic atrophy.

Because of the history, and neurologic and roentgenographic findings, we made a diagnosis of a tumor involving the right gasserian ganglion. Although the pain that the patient had was of the type usually associated with trigeminal neuralgia, which of course has an unknown etiology, the objective neurologic findings indicated a lesion that was destructive of nerve fibers. Operation for removal of the tumor of the gasserian ganglion was advised. The patient was told prior to operation that he was expected to have more "numbness" rather than less of the right side of the face after operation, for the surgical extirpation of a tumor involving

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the gasserian ganglion usually necessitates removal of part or all of the gasserian ganglion with division of the posterior sensory root.

On April 2, 1956, with the patient under the effects of intratracheal anesthesia and in the upright position, subtemporal craniectomy was performed on the right side through a small perpendicular incision ⅓ inch anterior to the tragus; the middle meningeal artery was silver-clipped and divided, and the dura mater was stripped from the 3rd branch of the gasserian ganglion and from the gasserian ganglion as is done when the posterior root of the ganglion is to be exposed for root section for trigeminal neuralgia. Immediately it was noticed that the exposed portions of the 2nd and 3rd branches and the ganglion itself bulged convexly toward the craniectomy, and these structures appeared yellower than normal, not unlike the gross picture of a neurofibroma. The capsule of the ganglion was incised and the tumor, which was under pressure, began to separate the fibers; it appeared slightly darker than the ganglion but not so dark as a meningioma. The tissue, which had a gross appearance not unlike that of an epidermoid, was gradually removed piecemeal. By means of a small curet it was possible to scoop out the neoplastic tissue not only from the mesial side of the 2nd and 3rd branches and the ganglion but also from the mesial side of the posterior root and over the tip of the petrous bone just beneath the tentorium. In removing the tumor it was necessary to sacrifice the sensory and motor roots of the ganglion. Several pieces of Gelfoam were left extradurally to control some of the oozing about the ganglion and then the wound was closed in layers without drainage.

Grossly the tissue removed was larger in amount than that from the usual “tie” operation. Microscopic verification of such tissue is routine in our laboratory and accordingly the material was sectioned immediately on a fresh-freezing microtome and stained with Terry’s polychrome methylene blue. This stain exhibits a beautiful metachromatic effect with amyloid and the diagnosis of amyloidosis was immediately rendered to the surgeon. Permanent sections made from frozen and paraffin blocks and stained routinely with hematoxylin and eosin, with Congo red, and with methyl violet brought out the characteristics depicted in the accompanying photomicrographs (Figs. 1–4). At least 50 per cent of both the ganglion and its afferent nerve bundles was replaced by a substance having all the

Fig. 1. Amyloidosis of gasserian ganglion and its afferent nerve trunks. The granular homogeneous areas devoid of nuclei represent the amyloid deposits (hematoxylin and eosin, ×125).