

Artery of Trigeminal Nerve Ganglion

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Abstract

The artery to trigeminal nerve ganglion has been identified as a branch that arises from the extracranial segment of middle meningeal artery prior to entry into the foramen spinosum. The role of the artery in being the arterial supply to trigeminal nerve ganglion is supported by electrophysiological and clinical suppression of trigeminal nerve activity during selective intra-arterial injection of anesthetics.

Keywords

Trigeminal nerve ganglion; middle meningeal artery; external carotid artery; intra-arterial injection; trigeminal neuralgia

The artery to trigeminal nerve ganglion (located in the foramen ovale) arises from the extracranial segment of middle meningeal artery prior to entry into the foramen spinosum (see Figure 1). The middle meningeal artery is a branch of the internal maxillary artery. Internal maxillary artery is one of the terminal branches of the external carotid. The artery to trigeminal nerve ganglion courses anteriorly and medially at its point of origin from middle meningeal artery (toward the foramen ovale which is located anteriorly and medially to the foramen spinosum) (see Figure 2) [1,2]. The artery to trigeminal nerve ganglion originates proximal to the petrosal artery which courses posteriorly and proximal to intracranial division of middle meningeal artery into the anterior and posterior dural branches.

Previous studies have suggested that artery to trigeminal nerve ganglion is a branch of internal maxillary artery or its tributaries. Temporary sensory loss in part or all of the trigeminal nerve distributions has been reported with intra-arterial injection of lidocaine in the internal maxillary arteries when performed as provocative test prior to embolization of arteriovenous fistulas [3]. Additional evidence identifies middle meningeal artery as the source of artery to trigeminal nerve ganglion. Intra-arterial injection of dimethyl sulfoxide/ethylene-vinyl alcohol copolymer into the middle meningeal artery can trigger trigeminal-cardiac reflex by stimulation of trigeminal nerve [4–6]. The trigeminal-cardiac reflex consisting of bradycardia or asystole, systemic hypotension, and

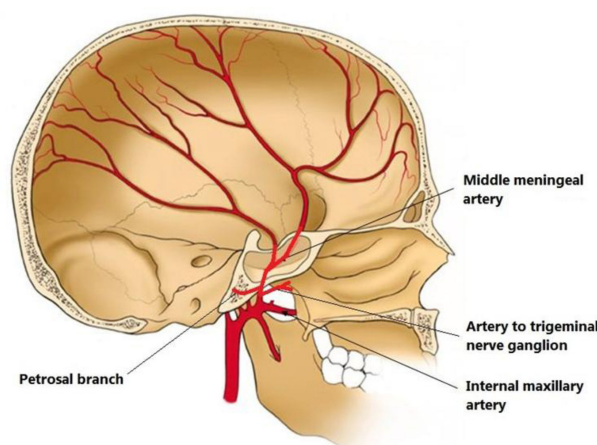


Figure 1. Illustration of artery to trigeminal nerve ganglion courses demonstrating origin from extracranial middle meningeal artery with a anterior and medial course toward foramen ovale.

apnea [7,8] is also observed during operative manipulation at the trigeminal nerve in proximity to cerebello-pontine region and spontaneously resolves after cessation of manipulation [9]. Resolution of trigeminal neuralgia has been reported following embolization of the middle meningeal artery-related arteriovenous malformations which also confirms the spatial co-localization of artery and nerve ganglion [10,11].

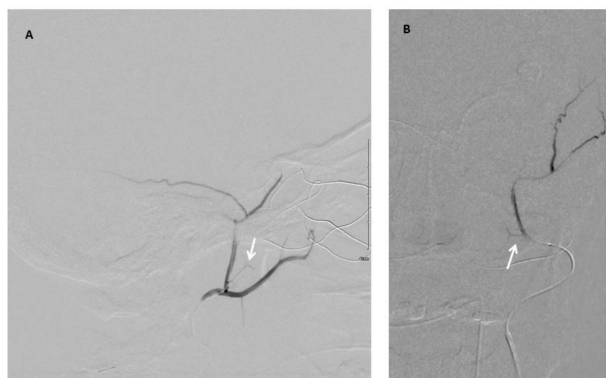


Figure 2. Selective angiogram with contrast injection in middle meningeal artery with images acquired in lateral (a) and anteroposterior (B) projections. the artery to trigeminal nerve ganglion is identified by arrows.

The most definitive evidence is based on a recent study by our group in which intra-arterial lidocaine in doses up to 50 mg was injected in the middle meningeal artery territory adjacent to the arterial branch that supplies the trigeminal nerve ganglion in three patients with refractory trigeminal neuralgia [12]. The latency and amplitude of R1 and R2 responses in the blink reflex were assessed before and concurrent with each incremental dose of lidocaine. In all patients, suppression of trigeminal nerve ganglion function was observed manifesting as latency prolongation and amplitude reduction of R1 and/or R2 responses. There appeared to be a dose-dependent effect with more pronounced effect on latency and amplitude of R1 or R2 responses seen with increasing doses of intra-arterial lidocaine. There was also clinical improvement concurrent with electrophysiological suppression of trigeminal nerve ganglion function. Two of three patients reported improvement in pain severity on 10-point numeric rating scale with pain free intervals of 5 and 3 days, respectively. There was improvement in facial hyperalgesia in all three patients in V1, V2, and V3 sensory dermatomes of the trigeminal nerve following intra-arterial lidocaine injection.

The artery to trigeminal nerve ganglion may be used in other therapeutic applications. Intra-arterial selective delivery of a high concentration of medication or neurotropic agents [13] may have value in treatment of migraine [14,15], regulation of cerebral blood flow and metabolism [16], cognitive function [17], and suppression of seizures, all of which have been related to the trigeminal nerve ganglion activity [18].

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