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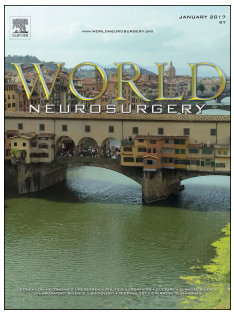

Microvascular decompression for treatment of trigeminal neuralgia in a patient with a facial nerve schwannoma

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Introduction

Classic trigeminal neuralgia (TN) comprises an unpredictable facial pain disorder frequently caused by vascular compression of the fifth cranial nerve at the root entry zone (1,2). Newly diagnosed or suspected TN should be investigated with neuroimaging, preferably thin slice axial and coronal MRI scanning, to investigate for possible secondary etiologies such as multiple sclerosis, herpes zoster, or space-occupying lesions (3,4). When space-occupying lesions are identified, surgery is often pursued with the intention of alleviating the concomitant TN (5). In this setting, the tumor is usually felt to be the most likely causative etiology for the pain and investigation for other possible causes, such as vascular compression, may not always be pursued. We present a case of a patient who developed a facial nerve schwannoma that was found to coexist with vascular compression of the trigeminal nerve. The resolution of the patient's TN following microvascular decompression (MVD) and petrous apex resection illustrates the importance of pursuing other actionable pathology even in the setting of evident tumor mass-effect.

Case Report

A 51-year old woman presented from an outside institution with intractable lancinating left hemi-facial pain distributed across all three divisions of the fifth cranial nerve. She additionally reported hypesthesia in a V2 and V3 nerve-distribution along with an increasing sensation of fullness in her left ear. There were no changes in balance or perceived hearing loss. Notably, the patient denied a history of facial nerve paresis, spasm, dysgeusia or dry eye. Physical examination was normal, including intact facial nerve function bilaterally. MRI revealed a 1.6 cm contrast-enhancing mass in the left cerebellopontine angle with extension into the internal auditory canal (IAC), consistent with a left vestibular schwannoma (**Figure 1**). No asymmetrical enhancement of the labyrinthine or geniculate segments of the facial nerve was identified. The cisternal component resulted in mild displacement of the trigeminal nerve root. In addition, a branch of the left SCA demonstrated coexisting nerve root compression (**Figure 2**). After reviewing options with the patient, tumor resection through a left retrosigmoid craniotomy with attempted hearing preservation and MVD of the left trigeminal nerve was planned.

Intraoperatively, the entire dorsal surface of the tumor capsule, as well as the superior and inferior surfaces, was stimulated at low threshold (0.2 mA, 50 ms duration, monopolar stimulation) with confirmatory firing of the facial nerve. Further, the probe was bent in order to stimulate as much of the ventral surface of the tumor as possible, and this also elicited facial nerve firing. In total, over 270 degrees of tumor surface stimulation elicited facial nerve firing, unequivocally proving that this was in-fact a facial nerve schwannoma. Careful mapping of the tumor surface failed to identify a safe entry window for intracapsular tumor debulking. Taken in combination with the patient's