Temporal Nerve Neuropraxia and Contralateral Compensatory Brow Elevation

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Ali Hendi, MD, has indicated no significant interest with commercial supporters.

The temporal branch of the facial nerve is the most commonly injured nerve in facial surgery.[1] This is due to its superficial location over the temporalis fascia as it ascends from the main trunk of the facial nerve (cranial nerve VII) to innervate the frontalis muscle. A case of temporal nerve neuropraxia and compensatory contralateral brow elevation is presented.

Case Report

An 80-year-old woman presented for surgical treatment of an invasive squamous cell carcinoma on the left temple (Figure 1). The lesion had been present for several months and had been enlarging. The patient did not have any motor deficit in the forehead region. The tumor was removed in two stages using the Mohs technique (Figure 2). Nerve tissue was not seen on the Mohs slides. The defect was closed primarily after careful undermining in the upper subcutaneous plane (Figure 3). After surgery the patient had a noticeable ipsilateral brow ptosis. At 1 month follow-up, she complained of heaviness of her left brow. Her vision was not affected. On exam, there was persistent ptosis of the left brow and compensatory elevation of the right brow (Figure 4). Manual elevation of the left brow caused an automatic release of the compensatory brow elevation of the right brow (Figure 5). The patient was unable to return for follow-up. In a phone interview 4 months after surgery, she noted the position of the eyebrows was 90% improved.

Discussion

The frontalis muscle is the primary muscle responsible for elevation of the eyebrows. It is innervated by the temporal branch of the facial nerve on each side. The temporal branch of the facial nerve

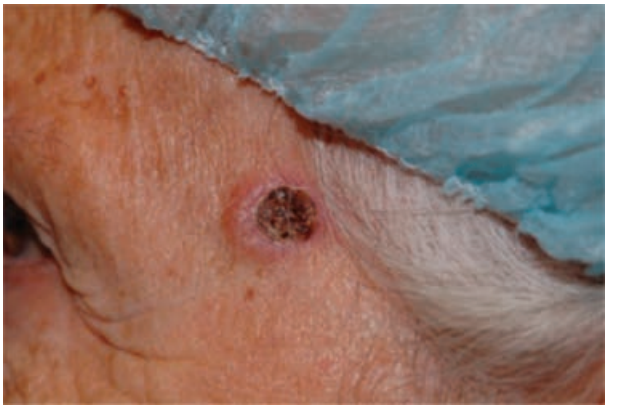

Figure 1. Squamous cell carcinoma on the left temple.

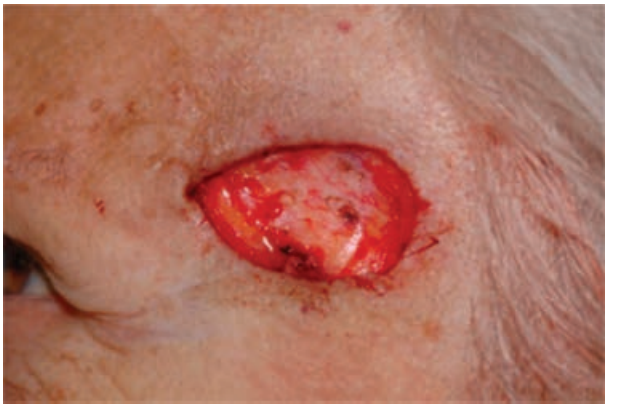

Figure 2. Defect on the left temple after tumor extirpation using the Mohs technique.

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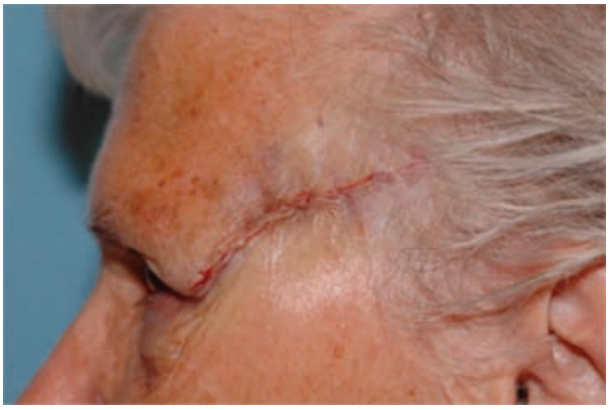

Figure 3. Primary closure of the defect.

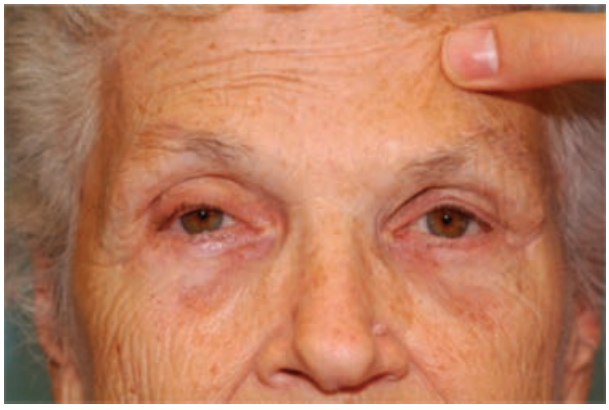

Figure 5. Release of compensatory right brow elevation with manual elevation of the left brow.

branches off the facial nerve trunk within the parotid gland. It then ascends superiorly to innervate the ipsilateral frontalis muscle. On its path to innervate the frontalis muscle, it lies superficial to the deep temporalis fascia, where it is at highest risk for injury.[1] It is for this reason, the temple is known as a "danger zone" in facial surgery. Transient temporal nerve paresis can be seen with infiltration of local anesthetic solutions in this region and should resolve within 24 hours. A motor nerve deficit that is reversible and lasts up to 6 months is likely to be neuropraxia. Neuropraxia is a temporary conduction deficit that is due to stretching or trauma of the nerve.[2] Permanent temporal nerve injury can be caused if the nerve is severed in the course of surgery. Neuropraxia can be differentiated from permanent nerve damage by electromyography and nerve excitability tests.[2] In this case, the absence of a nerve trunk on the Mohs slides would suggest that the temporal nerve was not severed. This patient was retrospectively diagnosed with neuropraxia based on the spontaneous near complete resolution of the deficit.

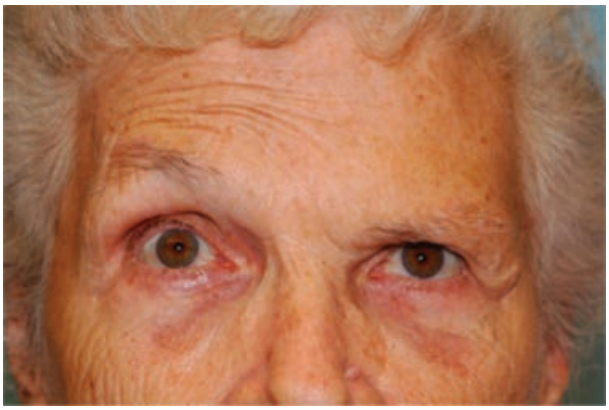

Figure 4. Left-sided brow ptosis and compensatory right brow elevation at 1 month postoperation. (Patient at rest, i.e., not trying to elevate brows.)

This patient also demonstrated a compensatory brow elevation on the contralateral side of the injury (Figure 4). The compensatory overstimulation of the contralateral frontalis muscle improves as the left frontalis muscle is manually raised (Figure 5). This central contralateral compensatory process[3] is due to the inability of the ipsilateral frontalis muscle to keep the brow in its natural position. This signals the brain to attempt to correct this by overstimulating the contralateral frontalis muscle. This phenomenon is documented in patients with Bell's palsy. A study of patients with Bell's palsy showed that 3 to 4 weeks after the onset of palsy there is an abrupt rise in the maximum compound action potential (MCAP) of the contralateral, healthy side as measured by electroneurography.[3] This increase in the MCAP plateaus and persists for about 3 months and at 4 to 6 months drops to lower levels which are still higher than the baseline. This was despite the fact that the mean time for the onset of recovery from Bell's palsy was 2 weeks. A similar central compensatory has been described for the vestibular system in animals.[4] Other animal studies have also shown motor cortex changes after peripheral facial nerve transaction.[5]

The patient presented could not return for follow-up; however, the compensatory process is likely to have resolved with the resolution of the neuropraxia of the left temporal nerve. This case illustrates two phenomena that the surgeon needs to be aware of: neuropraxia and central compensatory brow elevation after temporal nerve injury. Awareness of these phenomena can be reassuring to the surgeon and the patient.

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