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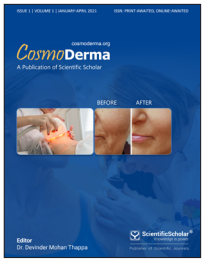

Letter to the Editor

Temporary facial paralysis following a local lignocaine injection

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Dear Sir,

Local anesthetics such as lignocaine are widely used in dermatologic practice due to their rapid onset, effectiveness, and safety profile. Although adverse events are rare, transient facial nerve paralysis is an unusual but important complication that can cause considerable distress. We report a case of temporary facial nerve palsy following local lignocaine administration for a minor dermatologic procedure, with emphasis on anatomical considerations, clinical differentiation, and management. A middle-aged woman underwent elective removal of a benign mole on her left cheek. The procedure was performed under local anesthesia using 1 mL of 2% lignocaine with adrenaline. Injection was given subcutaneously with standard aseptic precautions. The excision was uneventful, and the patient was discharged with routine post-operative instructions. Approximately 4 h later, she returned with sudden-onset left facial weakness. Examination revealed lower motor neuron facial nerve palsy: inability to close the left eye fully, asymmetry of facial movements, and left oral commissure drooping. She was afebrile and neurologically intact, except for the facial weakness. A focused assessment excluded other cranial nerve involvement. Given the timing, small anesthetic volume, and site of injection, a diagnosis of transient facial nerve blockade due to anesthetic diffusion was made. The patient was reassured, and no pharmacologic intervention was necessary. Complete recovery occurred spontaneously within 12 h [Figure 1a-c].

The facial nerve (cranial nerve VII) exits the stylomastoid foramen and branches into temporal, zygomatic, buccal, mandibular, and cervical divisions. These innervate the muscles of facial expression and course superficially through the cheek and periauricular area. Injections in these regions[1] risk unintended nerve blockade, especially with deeper or high-volume administration.[2] Although this phenomenon is more often reported in dental or maxillofacial contexts, it may occur during dermatologic procedures involving the midface.[3,4] Factors that influence risk include concentration and volume of anesthetic, depth of injection, and vasoconstrictive additives such as adrenaline that delay systemic absorption. It is critical to distinguish this reversible iatrogenic condition from other causes of facial paralysis, such as Bell's palsy, cerebrovascular accidents, or Ramsay Hunt syndrome. In this case, the absence of systemic signs, vesicular rash, or other neurological deficits, along with a clear temporal link to the procedure, supported a diagnosis of anesthetic-induced facial nerve palsy.[5] Imaging or further investigations are generally unnecessary if the presentation is classic and recovery is swift.

Management is conservative. Patients should be reassured that this is a self-limiting condition with expected full recovery. Nonetheless, clinicians must be aware of this possibility and take

Figure 1: (a and b) Four hours after surgery, (c) 12 hours after surgery.

steps to minimize risk. This includes using minimal effective anesthetic volume, injecting slowly, and avoiding deep injections in known danger zones–especially near the parotid gland, zygomatic arch, and mandibular angle. It is also advisable to inform patients undergoing facial procedures of this rare risk during pre-operative counseling. This case reinforces the importance of anatomical knowledge, careful injection technique, and calm clinical judgment when faced with unexpected post-operative facial weakness. Raising awareness of this benign complication can help reduce unnecessary referrals and alleviate both patient and practitioner anxiety.

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