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MANAGEMENT OF MANDIBULAR PARESTHESIA: A LITERATURE REVIEW

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ABSTRACT
Many systemic and local factors can cause paresthesia, and it is rarely caused by infections of dental origin.

INTRODUCTION

Paresthesia is characterised as a prolonged state of impaired sensation or persistent anaesthesia that lasts longer than anticipated. It is typically an unavoidable consequence for patients having oral surgery, including implant implantation, but it is also frequently mentioned in cases of dental negligence.

Paresthesia due to periapical infection may be caused by

The accumulation of purulent exudate in the mandibular bone can result in mechanical pressure and ischemia related to the inflammatory process (edema), or it can cause local pressure on the mental nerve that causes paresthesia due to periapical infection. Secondly, the toxic metabolic or inflammatory products of bacteria can cause paresthesia.
(1) enough force exerted by a hematoma that develops later.

The "electric shock" feeling that results from the needle making contact with the nerve may be all that is needed to trigger paresthesia. Because dentistry uses fine gauge needles for local anaesthetic injections, nerves seldom become severed during the procedure. Though it has been suggested that paresthesia is more likely after blocks using 4% local anaesthetic solutions, the exact aetiology of paresthesia following local anaesthetic injection remains unknown.[2,3]

PATIENT MANAGEMENT

1. Comfort the sufferer

The doctor should have a direct conversation with the patient.

Describe the onset of paresthesia and the anticipated resolution period.

Schedule a patient examination appointment.Document the event in the dental file.

2. Patient assessment

Talk to the patient about the paresthesia phenomena.

Describe how paresthesia can take months to go away and sometimes it can stay that way forever.

Assess the patient's level and severity of paresthesia.

Enter the results of the examination in chart

3. Observe the patient again

Reevaluate the patient within a month, then every one to two months after that, or more frequently if necessary, for as long as the paresthesia lasts. Even a slight improvement in the symptoms is frequently a clue that the condition will eventually completely resolve.

Paresthesia does not cause muscular paralysis; rather, it is purely a sensory phenomena[5-8].**Signs and symptoms of paresthesia.**

Most of the time, the nerve damage is discovered as a postoperative problem rather than during the dental surgery.

In the affected area, the patient will experience altered, decreased, or even entire loss of sensation. There could be involvement from one or more senses (taste, touch, pain, proprioception, or perception of temperature).

The specific region that is impacted is the one that the injured nerve serves. Regarding the mandibular or lingual nerves, this refers to a part of the lip, chin, palate, or tongue of the individual.

How long does the numbness/sensory loss last?

For those patients who are affected, one of 3 scenarios will play out.

- In most cases, the paresthesia is transient, resolving on its own after just a few days or weeks.
- In some cases, the condition is best classified as being persistent (lasting longer than 6 months).
- For a small number of cases, the loss is permanent.

Treating permanent paresthesia.

Testing/mapping paresthesia.

As a way of documenting the extent of a patient's condition, both initially and as recovery occurs, the affected area can be mapped.

To do so, different types of sensory tests are performed, and those regions (lip, facial skin, tongue, etc...) that respond with no or altered sensation are recorded.

Light Touch: To test whether a little cotton ball can be felt and whether the patient can tell which way the ball is moving, it is gently stroked against the skin. Using this test to analyse moist tissues (such as the lining of the mouth) might be challenging.

Discrimination between sharp and dull: Areas are prodded using a pin or other sharp object. If the patient is experiencing any kind of feeling, it is inquired as to whether it is dull or keen. Additionally, a comparison is conducted with the patient's unaffected side at the same spot.

CONCLUSION

In conclusion, the pathogenesis of delayed paresthesia in our patient is believed to involve bacterial infiltration of the neural sheath and a transient conduction blockage brought on by fibrinolysis's breakdown by free radicals.

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