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Neuropathologic chewing in comatosed children: a case report

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Abstract

Neuropathologic chewing, a complication which may present in the comatosed drowning victim, may result in severe self-inflicted trauma to the soft tissues of the oral cavity. Various clinical reports have documented this problem in adults. This report documents a case of neuropathologic chewing in a two-year-old.

Introduction

The comatosed patient may exhibit powerful ruminatory-like movements of the mandible which are difficult to control and which may result in severe self-inflicted trauma to the soft tissues of the oral cavity. Guyton (1968)[1] has termed this condition neuropathologic chewing. Various clinical reports have documented such cases.[2,3,4] However, little information is available regarding the occurrence or management of this problem in pediatric populations. The purpose of this report, therefore, is to present a case of neuropathologic chewing in a two-year-old drowning victim.

Case Report

K. B., a 23-month-old white male, was found unconscious at 8:45 am in the family swimming pool on the day of his first admission to the Children's Hospital of Philadelphia. Cardio-pulmonary resuscitation was initiated immediately by his parents and continued by a local rescue unit. The child was then transported to the emergency room of a local hospital where he was intubated and received intracardiac epinephrine as well as other resuscitative drugs. The patient was then transferred to the Pediatric Intensive Care Unit (PICU) of the Children's Hospital of Philadelphia where he was admitted at 1:45 pm. On admission, he was noted to be unconscious, hypertonic and non-reactive to pain. Other neurological findings included non-reactive constricted pupils, absent corneal and plantar reflexes, and increased intracranial pressure (ICP). His vital signs were stable and within normal limits.

Immediate treatment was directed at stabilizing the increased ICP. Therapy included: mannitol infusions; pentathol; decadron; and placement of a subarachnoid bolt. By the fifth day of his hospital course, his ICP stabilized and the subarachnoid bolt was removed without complication. The patient's neurological status had not improved and was further complicated by generalized clonic and tonic muscular activity suggestive of seizures. Treatment for this problem included IV valium and tranxene. On the following hospital day (day seven), the patient began spiking high fevers and exhibited marked generalized hypertonicity. Blood and urine cultures were negative. A chest film was also negative. The fevers were thought to be secondary to CNS deficits (i.e. hypothalamic dysfunction) rather than infection. It was also observed at this time that the patient exhibited active neuropathologic chewing which lacerated his tongue. The attending PICU physician requested a dental consult to evaluate and treat this problem.

Dental examination indicated that the patient had an incomplete primary dentition comprised of 16 erupted teeth (the second primary molars were not erupted). The only remarkable oral findings were generalized lacerations of all the surfaces of the anterior two-thirds of the tongue. In addition, these surfaces were coated with adherent white plaques (Figure 1). Oral cultures were obtained and the bacteriology report confirmed the clinical impression of moniliasis. Topical nystatin therapy (Mycostatin — 100,000u q.6.h.) was instituted. In addition, a recommendation was made to construct an intraoral appliance to abort the complications associated with the neuropathologic chewing.

On the following day, the patient had a tracheostomy as his upper airway reflexes were not intact. During the course of the next day, alginate impressions were obtained to fabricate working models for the intraoral appliance. This procedure was accomplished after the patient was paralyzed (10mg succinyl choline IV) and ventillated by the staff anesthesiologist. Upper and lower methyl methacrylate splints

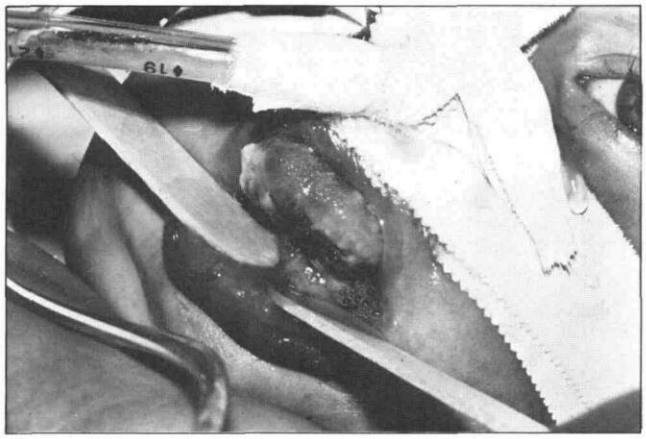

Figure 1. Lacerated tongue with adherent white plaques an initial presentation.

were constructed and cemented in place on the following day after the patient was paralyzed and artificially ventilated.

During the next few days, it was noted that the neuropathologic chewing worsened and the tongue wounds were not healing. In addition, the nursing staff had significant difficulty applying the nystatin secondary to the clenching and chewing. As the possibility of fungal sepsis[5] through a lingual portal was of concern, a mouth opening appliance to facilitate nystatin application and tongue healing was fabricated. Likewise, the potential complication of disseminated moniliasis precluded exodontia.[5] Finally, as K. B. was not swallowing, his pooled oral secretions resulted in drooling. Drooling may contaminate the tracheal stoma resulting in serious pulmonary infection. Therefore, mouth opening was also necessary for adequate suctioning.

New alginate impressions for working models were obtained after the patient was paralyzed and artifically ventillated. In addition, the patient's mouth was slowly opened with a ratchet type prop to the point of maximum opening and the distance between the incisal edges of the upper and lower incisor teeth was recorded (43 mm). The models were mounted on an articulator and a wire reinforced methyl methacrylate bite opening applicance was constructed 3 mm short of the recorded maximum opening. The appliance was inserted after the patient was paralyzed and artifically ventilated (Figure 2). Over the course of the next six days, the tongue lacerations healed and the moniliasis improved significantly. In addition, the patient was afebrile and his hypertonicity had improved markedly. The appliance was removed after being in place for seven days.

During the next seven days, the patient exhibited no signs of clenching or neuropathologic chewing. The nursing staff had no difficulty maintaining oral hygiene or applying nystatin. However, during the following week the patient developed spiking fevers associated with generalized hypertonicity and resumption of neuropathologic chewing. Treatment for this problem included IV valium and tranxene. In addition, the mouth opening appliance was reinserted to abort further trauma to the tongue and facilitate nystatin application. The white adherent plaques, previously localized to the tongue, had spread to the floor of the mouth (Figure 3). This lesion was also refractory to seven days of topical nystatin therapy. Oral cultures and a biopsy specimen from the ventral surface of the tongue ruled out a fungal etiology. The bacteriology report indicated that the oral samples were largely composed of *Pseudomonas aeruginosa* and coagulase-positive *Staphylococci.* Blood cultures were negative. After consulting with the Infectious Disease Service, the nystatin therapy was discontinued and the mouth was swabbed six times per day with Betadine.

This therapy was continued for the next six days. All intraoral lesions resolved during this period and the mouth opening appliance was subsequently removed after 13 days of being in position (Figure 4). Physical examination suggested no TMJ pathology or

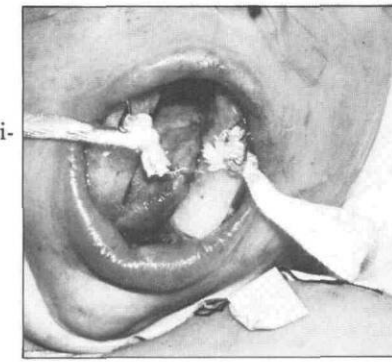

Figure 2. Appliance in position at initial insertion.

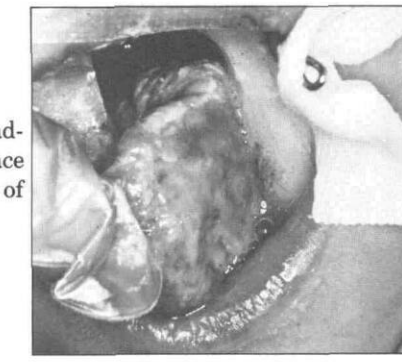

Figure 3. White plaques adhering to the ventral surface of the tongue and the floor of the mouth.

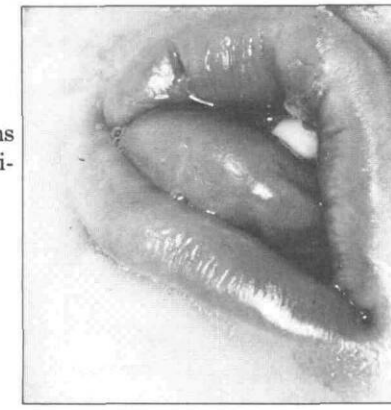

Figure 4. All tongue lesions resolved after six days of topical Betadine therapy.

intraoral soft tissue damage associated with the appliance. On the 43rd hospital day, the patient was transferred from the PICU to a rehabilitation facility where he was followed by the staff pedodontist. He exhibited no furuther clenching or neuropathologic chewing for the subsequent 6 months. His neurological status did not significantly improve since the original episode.

The Appliance

The appliance extended from the occlusal half of the distal surfaces of the first primary molars to the incisal half of the mesial surfaces of the primary cuspids. The superior and inferior aspects of the appliance interdigitated with the cusp tips of the primary molar and cuspid teeth. The buccal-lingual width of each methyl methacrylate block was approximately 10 mm. The bite was opened anteriorly about 40 mm. This opening provided access for nystatin and Betadine application, oral hygiene and suctioning. In addition, this degree of opening prevented the patient from dislodging the appliance. The appliance was also reinforced by two .036 stainless steel wires which were adapted to the palatal contours and connected the two methyl methacrylate blocks. An additional .036 stainless steel wire was attached to each block and extended extraorally. Trach tape was fastened to each extraoral wire and fastened on the patient's occipitat.

Discussion

The neurophysiological basis for neuropathological chewing has been discussed previously.[1,3,4] The placement of a bolus between the teeth results in a dropping of the mandible. Upon opening, the stretch reflexes of the muscles of mastication are stimulated, resulting in a rebound contracture. Subsequent to closure into the bolus, pressure receptors in the mouth cause an automatic opening of the mandible. This cycle will continue until the bolus is removed. With regard to neuropathologic chewing, the bolus is usually the tongue.

Earlier reports regarding the management of this problem have been directed at eliminating the tongue as a trigger mechanism. Devises that depress and stabilize the tongue in the floor of the mouth prevent the bolus effect and facilitate healing.[2,3,4] However, such appliances also impose constraints regarding the management of monilial and *Pseudomonas* infections of the tongue and floor of the mouth. In addition to interfering with topical chemotherapy, a tongue depressing stent may also complicate the clinical course of a lingual monilial infection. The relationship of oral moniliasis to foci of oral stasis (i.e. dentures) has been well documented.[6]

Sepsis was not the only complication of concern regarding the management of K. B.'s oral infections. Drooling may contaminate the tracheal stoma resulting in serious pulmonary sequela. Therefore, adequate oral access must be maintained to insure proper topical chemotherapy, oral hygiene, and suctioning.

An additional problem regarding K. B.'s management relates to the potential TMJ complications associated with mouth opening appliances. In this regard, dislocation and/or spasm of the muscles of mastication were of prime concern.[7] Dislocation was prevented by carefully recording the point of maximum jaw opening and constructing the appliance to effect an opening that did not exceed this point. Spasm of the muscles of mastication was not evident after appliance removal. This finding was, in part, probably due to the muscle relaxing agents (tranxene and valium) that K. B. was given.

Finally, this case also illustrates that neuropathologic chewing may be a self-limiting phenomenon. Although this patient exhibited several episodes of neuropathologic chewing during the first seven weeks of his hospital course, he demonstrated no signs of clenching or chewing over the past six months.

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