Posttraumatic Pneumosialodenitis of the Submandibular Gland

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Abstract

There are multiple etiologies for facial subcutaneous emphysema, including facial trauma, dissection of air along facial planes from a pneumomediastinum, infection, and entry of air through defects in teeth due to decay, trauma, or dental procedure. In addition, air can be forced through lacerations of the oral or pharyngeal mucosa by increased intraoral pressure. An infrequently reported cause of subcutaneous emphysema is autoinsufflation of the parotid duct with extravasation of air into the surrounding tissue. In this report, the authors present a 14-year-old patient treated at the Department of Oral and Maxillofacial Surgery of the Hospital Escuela Universitario due to an increase in size in the buccal region and left masseterine space of 2 months of evolution. Presenting with a sudden evolution, without any possible explanation, which caused displacement of adjacent structures without damage to the bone or surrounding tissues. After a series of echographic and tomographic studies, it showed that inside the submandibular gland there was a hypodense image with gas density (-1100 UH) expanding the gland and its respective excretory duct.

Keywords

► sialoadenitis
► salivary glands
► submandibular gland
► pneumothorax
► pneumomediastinum
► maxillofacial

Case Report

A 14-year-old male patient with no pathological medical history was referred to Hospital Escuela Universitario to the Oral and Maxillofacial Surgery Department due to an increase in size in the buccal region and left masseterine space of 2 months of evolution; oral aperture was compromised and made it difficult for the patient due to the increase in volume which was even more noticeable when closing the mouth (►Figs. 1 and 2).
The increase in volume was spontaneous with recurrent episodes without any apparent cause. Upon palpation, crepitation sounds were heard in the affected area. When exploring salivary glands by means of bimanual palpation, patient referred mild pain on the left submandibular gland and there was also presence of frothy saliva at the exit of the Wharton’s duct.

When performing mandibular opening movements, the increase in volume seems to disappear momentarily. Patient did not report having difficulty while breathing or recent fever. Laboratory blood tests were within normal ranges. Patient came with a radiographic study, a posteroanterior skull radiography (Fig. 3) in which a radiolucent area at the mandibular level with well-defined edges is observed.

<table>
<thead>
<tr>
<th>Table 1 Situations or maneuvers reported to cause pneumosialodenitis</th>
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<tbody>
<tr>
<td>• Wind instrumentalists(^5,6)</td>
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<td>• Balloon and glass blowers(^5-7)</td>
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<td>• Bicycle tire inflation by mouth(^8)</td>
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<td>• Dental procedures using air-powered equipment(^9)</td>
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<tr>
<td>• Cough in COPD(^10)</td>
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<td>• Nose blowing(^11)</td>
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<td>• Whistling(^12)</td>
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<td>• Valsalva maneuver to clear ears(^13)</td>
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<td>• Spirometry(^14)</td>
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Abbreviation: COPD, chronic obstructive pulmonary disease.

Fig. 1 Initial frontal photography.

Fig. 2 Initial left and right lateral photographies.

Fig. 3 Posteroanterior skull radiography.
Ultrasound and contrast computed tomography of the facial skeleton and thorax were requested.

Tomography report stated that inside the submandibular salivary gland, a hypodense image, gas density (-1100 UH) was observed, dilating the gland and also the Wharton’s duct, measuring $3.95 \times 4.47 \times 4.68$ cm considering emphysematous sialoadenitis (►Figs. 4–9).

It was decided to give the patient a follow-up and treatment with antibiotics. Procaine Penicillin in doses of 1.2 million U.I. one each 24 hours for 10 days as prophylactic means was administered.

At the end of the antibiotic therapy, a slight decrease in volume was observed. Patient was evaluated every 4 weeks for 2 months after antibiotic therapy was finished (►Figs. 10 and 11).

During the follow-up, a satisfactory evolution was observed, showing a gradual decrease from the initial volume,
just as the literature mentions it to be self-limiting pathology. Subsequently, follow-up was performed every 3 months until full remission of the pathology. Currently, patient is in control every 3 months.

**Discussion**

When reviewing the literature, it can be recognized that the presence of pneumosialadenitis has a higher incidence in the parotid glands. As possible causes of appearances and recurrences of parotid involvement, there is a weakening of the buccinator muscle that does not prevent the retrograde flow toward the Stensen duct and the presence of an abnormal dilation in the ducts path. It is suspected that at the submandibular level, the possible causes would be a dilatation of the duct as anatomical variation or secondary to a trauma.

**Conclusion**

Pneumosialadenitis is the insufflation of air inside the duct and the affected salivary gland, presenting more frequently in the parotid glands. Its treatment varies and is based on whether there is an infectious process, if the intraglandular air is extravasated to become subcutaneous emphysema or its high recurrence. These transient events can be treated with prophylactic antibiotics with observation, but recurrent cases must be managed by means of surgical techniques of relocation of the secretory duct or even partial or total removal of the affected gland.

**Conflicts of Interest**

None.

**References**