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CASE REPORT

Mandibular ramus-related Stafne's bone cavity

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Mandibular bone depressions located on the lingual/buccal aspect of the mandibular ramus are the rarest variants of the so-called Stafne's bone cavities, or major salivary gland-related depressions, with only 17 cases reported in the literature including both clinical cases and archaeological specimens. We report the case of a 14-year-old male patient who sought clinical assistance complaining of a hard expansion on the lower left premolar–molar region. Apart from a unilocular radiolucent lesion between the lower left second premolar and first molar, a panoramic radiograph showed another radiolucent lesion located in the right mandibular ramus, at the level of the mandibular foramen. Computed tomography (CT) revealed an expansile lesion in the left mandibular body, later diagnosed as a simple bone cyst through surgical exploration. The three-dimensional CT volume rendering reconstructed image showed that the second lesion, located on the lingual aspect of the ascending ramus, was an actual cortical bone defect, which was diagnosed as a mandibular ramus-related Stafne's bone cavity. Considering the young age of the patient, the size of the defect, the recognizedly slow development of mandibular bone defects and, above all, the location of the bone defect under discussion, we believe it to have a congenital rather than a developmental origin (*i.e.* it was caused by a focal failure during intramembranous ossification of the mandible). If this is the case, mandibular bone depressions should not be seen exclusively as salivary gland-related bone defects. *Dentomaxillofacial Radiology* (2004) **33,** 63–66. doi: 10.1259/dmfr/39682286

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Case report

A 14-year-old male patient complained of a slight deformity on the left side of the mandible, between the second premolar and first molar. He was referred to a private clinic for panoramic radiography. The panoramic view (Figure 1) showed a unilocular radiolucent area located between the roots of the left second premolar and first molar, extending downward to the mandibular canal. There was no well defined cortical outline, even though the margins of the lesion were clearly delineated. The longest axis of the radiolucent area was vertically placed, its largest width seen below the apexes of the dental roots, and its caudal expansion jeopardized the superior wall of the mandibular canal. Although the roots of the second premolar and first molar displayed a slight divergence, there was no sign of resorption.

A second radiolucent area could be seen in the right mandibular ramus, at the level of the mandibular foramen, extending to the posterior border of the mandibular ramus and involving the mandibular foramen anteriorly. This lesion presented a well defined contour but no cortical outline. The lesion was somewhat irregular, almost triangular in shape, with rounded edges.

As the second step in a sequential diagnostic approach, computed tomography (CT) was performed. The multiplanar reconstructed images (Figure 2) showed the first unilocular area as a lesion showing slight expansion and thinning of the lingual and buccal cortices, with no invasive features. Although the density of the bone lesion's content (45–55 HU) did not exclude the possibility of a tumour, our first diagnostic impression was that of a cyst. Surgical exploration revealed the presence of blood content with no fibrous capsule, suggesting a diagnosis of a simple bone cyst.

With respect to the lesion in the right mandibular ramus, the three-dimensional (3D) CT volume rendering reconstructed image (Figure 3) showed it was an actual cortical

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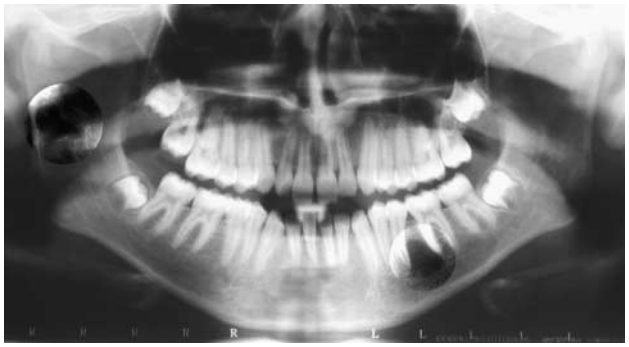

Figure 1 Panoramic radiograph displaying two radiolucent lesions, one in the region of the lower left second premolar and first molar and the other in the right mandibular ramus

bone defect located on the lingual aspect of the ascending ramus. It is important to notice that the 3D CT volume image displayed a defect showing a more irregular shape and a larger area, measuring 22.9 mm (longest vertical axis) × 15.0 mm (longest horizontal axis). On the other hand, the two-dimensional CT image (Figure 4) clearly showed that the defect was shallow and totally covered by the medial pterygoid muscle. For this reason, there was no contact between the corresponding parotid gland and the medial aspect of the mandibular ascending ramus.

Discussion

After a worldwide literature review, Philipsen et al[1] concluded that, in light of current knowledge, variants of the so-called Stafne's bone defects could have a common origin: the pressure exerted by hyperplastic/hypertrophic salivary glands on bone surfaces. However, in describing possible locations for the posterior variant (a submandibular gland-related bone defect), they mention an occurrence of the lesion on the lingual surface of the mandibular angle, in the area where the medial pterygoid muscle is attached, an area in which there is no possibility of contact between the submandibular gland and the bone surface. For this reason, ascribing one sole cause to this kind of bone defect seems inadequate to us – and this is something that may become even more evident when the anatomical relationship between the parotid gland and the ascending ramus of the mandible is taken into consideration.

Only rarely have bone cavities been described in association with the parotid gland, in a total of 6 clinical cases[2,3] and 11 archaeological cases.[4–6] The first five cases of bone defects located on the ascending ramus of the mandible were described by Wolf.[2] A relationship with the parotid gland, however, can be admitted only for the one case in which the defect affected the continuity of the posterior border of the ascending ramus, *i.e.* case No. 3, in which the gland is in close contact with the bone, from the condylar neck (below the area of attachment of the temporomandibular joint capsule) to the mandibular angle. Couly,[7] in turn, had already drawn attention to the possibility of parotid tumours being the cause of lordosis of the posterior border of the ascending ramus. And, indeed, this is a possibility that should be taken into account: since the parotid gland is, both posteriorly and medially speaking, associated with the sternocleidomastoid, digastric,

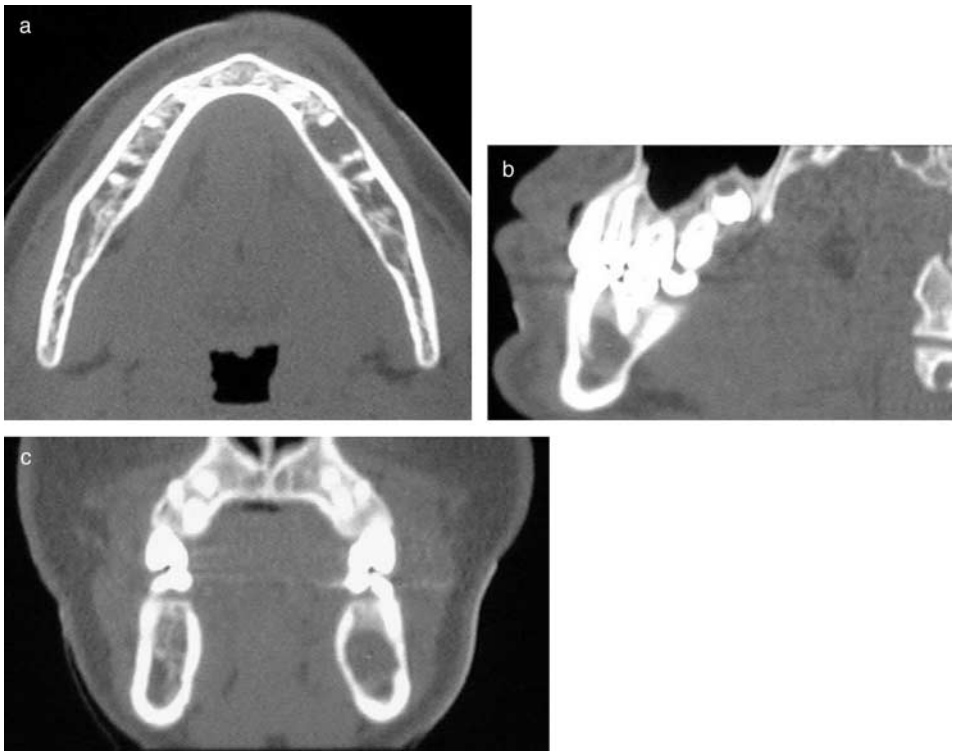

Figure 2 Multiplanar reconstructed images displaying the mandibular bone cyst in (a) axial, (b) sagittal and (c) coronal view

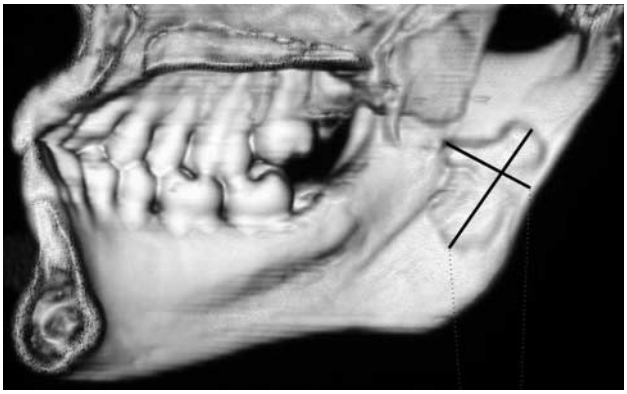

Figure 3 Three-dimensional CT volume rendering reconstructed image showing a lingual mandibular ramus-related Stafne's bone cavity larger than observed in the panoramic radiograph

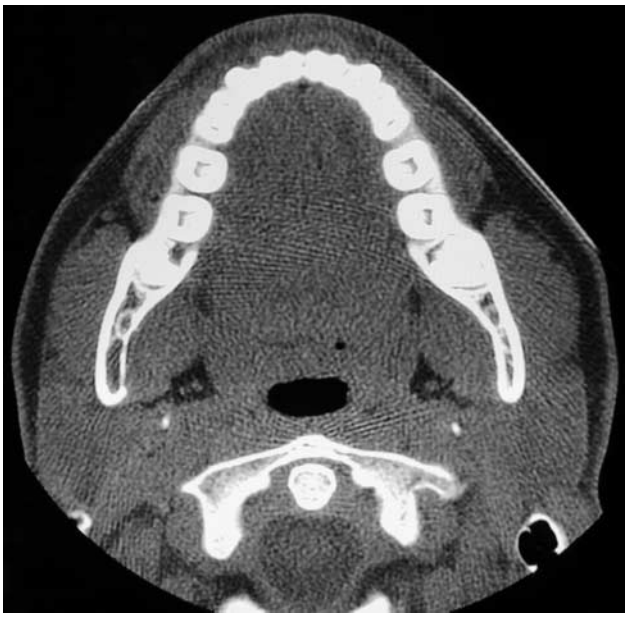

Figure 4 Two-dimensional CT axial view at the level of the ramus lesion showing a thinner lingual cortex in which there is no contact between the parotid gland and the lingual or buccal aspects of the ascending ramus

stylohyoid, styloglossus and stylopharyngeal muscles. When subjected to an increase in volume, it is forced to expand anteriorly, laterally and medially. On the other hand, it is necessary to consider that the connective tissue that binds the gland to the posterior border of the ascending ramus characteristically allows the glandular tissue to glide along the bone surface during mandibular movements. This would most likely prevent any significant pressure on the posterior border of the mandibular ramus that could bring about cavitation, especially when the thickness of this bone cortex is taken into account.

With regard to the other four cases, the examinations performed to determine the location of the bone defects and to establish their relationship with the glandular tissue (namely stereoscopic radiography and sialography) could only just corroborate the closeness among the anatomical structures in the area owing to their lack of precision. On the other hand, it must be acknowledged that the areas where the lesions occur are, totally or partially, free from being covered by muscle, the medial pterygoid on the lingual aspect or the masseter on the buccal aspect.

In the case presented by Barker,[3] there is indeed no covering by the medial and lateral pterygoid or masseter muscles or by the joint capsule in the region where the condition had been detected. In that case, there is actual contact between the parotid gland and the bone on the buccal and lingual aspects. However, the flat images that illustrate his work have not allowed him to determine whether the condition was buccal or lingual. Thus, based on sialography, it seems natural to speculate about an apparent relationship between the glandular tissue and the bone defect, but in fact it would be inappropriate to conclude that this relationship is not equivocal.

Shields[6] describes the only case where the bone defect is located on the buccal surface of the ascending ramus. He is right to emphasize that in this area the superficial lobe of the parotid gland is in close contact with the bone surface, since it is located below the area of attachment of the capsule and the lateral ligament of the temporomandibular joint, and behind and above the masseter muscle. However, owing to the fact that what had been observed was a dry mandible, here too the relationship between the bone defect and the parotid gland can only be suspected.

Ultimately, a relationship between the parotid gland and cavitary bone defects in the ascending ramus seems highly improbable, even considering the areas in which there is contact between glandular tissue and bone surface. The reason for this is that the gland is covered solely by skin laterally and medially it adjoins the parapharyngeal space. Thus, hypertrophic/hyperplastic conditions associated with the parotid gland would tend either to cause the skin to distend or to force the glandular tissue to project inwardly, towards the parapharyngeal space – which means the glandular tissue could have no way of exerting enough pressure on the bone surface to cause cavitation.

A strong argument for the developmental origin of mandibular bone defects is the fact that they occur primarily among middle-aged individuals. A few rare cases of such defects have been reported among individuals in their teens, but none among younger individuals. Considering the young age of the patient (it is the first case of the variant associated with the ascending ramus reported among individuals under 20 years), the size of the defect (twice the area of the largest of the defects reported in the literature), the recognizedly slow development of mandibular bone defects (such a severe lesion would require a long time to develop) and, above all, the location of the bone defect under discussion (*i.e.* an area covered by the medial pterygoid muscle), we believe it to have a congenital rather than a developmental origin.

According to Mann and Tsaknis,[4] bone defects located in the sulcus of the mandibular foramen could be associated with neoplasms of neural or vascular origin or even with aneurysms of the inferior alveolar artery. However, they have also emphasized that such conditions

could not be capable of exerting enough pressure to erode the bone surface of the ascending ramus laterally, since they would naturally tend to spread to medial soft tissues. In spite of the logical inference, it is our belief that the mandibular movements, as well as the successive distensions and contractions of the medial pterygoid muscle, which overlaps the lingual surface of the ascending ramus and the mandibular foramen almost entirely, would press, both significantly and repeatedly, any lesion that might be found in the area against the bone surface. As a result, superficial erosion and development of a bone defect would follow. If it were interposed between the muscle and the bone surface and covered the area of the mandibular foramen, the sphenomandibular ligament could have a similar contributory effect.

Minowa et al[8] have formulated the latest hypothesis regarding the posterior variant, according to which bone cavitation follows from erosion brought about by acquired vascular lesions. However, the present case does not confirm it, first because the region affected by the lesion is totally protected and free from trauma (a determining aetiological factor in the occurrence of vascular lesions in young patients) and second because in this region there is no significant artery near or in contact with the bone surface (the mylohyoid artery lies anteriorly). Furthermore, the small depth and the large area of the lesion contrast with the classical characteristics of Stafne's bone cavities.

Following such considerations, we could conclude by saying that, in regions where there is no contact between major salivary glands and bone surfaces, mandibular bone defects can have either a developmental origin, as long as they are not related to the glands, or a congenital origin, being the result of a focal defect during the mandibular intramembranous ossification process, as suggested by the present case. Thus, mandibular bone depressions should not be seen exclusively as salivary gland-related bone defects.

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