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## METASTASIC BREAST CARCINOMA TO THE MANDIBLE

 $A.\ Belmehdi,\ S.\ Chbicheb$  Dental School, Mohammed  $V^{th}$  University in Rabat, Rabat, Morocco

**Corresponding Author:** Akram Belmehdi, **MD.** 

Affiliation: Dental School, Mohammed V<sup>th</sup> University in Rabat, Rabat, Morocco.

E-mail: akram.belmehdi@gmail.com

**Tel.:** + (212) 662296555

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A 48-year-old female patient had reported to the department of oral surgery with a complaint of swelling, discomfort and paresthesia in the lower right posterior region of jaw since 3 months. Her medical history revealed a modified radical mastectomy with axillary lymph node dissection for invasive ductal carcinoma of the right breast. Extra- oral examination was not relevant except the presence of a right submandibular lymphadenopathy which was present with a palpable lymph node of size  $2 \times 2$  cm approximately. Intra- orally expansion of buccal cortex was noticeable in the right molars region and overlying mucosa was normal. Orthopantomogram (OPG) examination revealed a pyramidal irregular osteolytic lesions suggestive of partial bone destruction on right body of mandible, involving the inferior alveolar nerve (IAN) (**Figure 1**). Panoramic and serial cross-sectional CT-scan showed a large hypodense image with  $2\times3$  cm dimension and loss of lingual cortex (**Figures 2, 3**). Under local anesthesia biopsy of the lesion was taken. And based on histopathology, final diagnosis was consistent with metastatic lesion to the mandible from breast carcinoma. The patient was referred back to her oncologist to start radiotherapy.

Metastases in the oral cavity are rare and comprise approximately 1% of all oral malignancies (1). In descending order, the oral locations to which metastasis most commonly occur are the jaws, periodontal tissue (mandible/maxilla), and the tongue, however, involvement of both the mandible and the maxilla is rare (2). The most common symptom suggestive of metastatic disease is paresthesia of lower lip and chin (3). Metastases of breast cancers identified to cause osteolytic lesions secrete biological mediators along with interleukin IL-11, IL-8, and IL-6, parathyroid hormone-related protein (PTHrP), that induce osteoclast mediated bone resorption through activation of the RANK/RANKL/ OPG signaling pathway. These mediators up-regulate the expression of RANKL and down-regulate the expression of L (OPG) by osteoblasts and other stromal cells, as consequence of promoting osteoclast differentiation and activation, culminating in bone resorption (4).



Figure 1: Orthopantomogram showing a pyramidal radiolucent lesion



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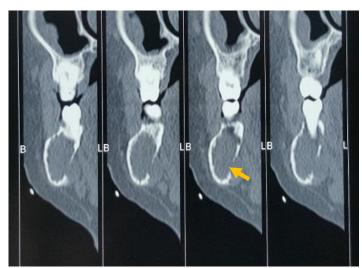


Figure 2: CT-scan panoramic view showing the dimension of the lesion with involvement of the IAN



Figure 3: Cross-sectional CT-scan showing the expansion of the lesion with destruction of the lingual cortex

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