Mandibular metastasis in a patient with follicular carcinoma of thyroid

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Abstract

Mandibular metastasis due to thyroid carcinoma is not very frequent and the cases described in the literature are few. Due to its bloodstream dissemination, most of them are a consequence of the follicular variant of thyroid carcinomas. We are presenting a case in which the metastatic lesion of mandible was detected before diagnosis of primary malignancy.

Keywords: Follicular carcinoma of thyroid, mandible, metastasis

Introduction

Metastatic tumors of oral cavity are very rare, accounting for 1% of neoplasm in the area and their primary origin can be anywhere.^[1] Most such patients were previously diagnosed with primary neoplasm. The literature states that in about 30% of cases of patients with gnathic bone metastases, the primary tumor is asymptomatic and not diagnosed.^[2] The most common primary tumors leading to mandibular metastasis were lung in men and breast in women.^[3] These metastatic lesions (or tumors) usually are carcinomas rather than sarcomas.^[4]

Mandibular metastasis due to thyroid carcinoma is not very frequent and the cases described in the literature are few. Due to its bloodstream dissemination, most of them are a consequence of the follicular variants of thyroid carcinomas.^[5] We are presenting a case in which the metastatic lesion of mandible was detected before diagnosis of primary malignancy.

Case Report

A 40-year-old female patient reported to the oral medicine and radiology department with complaint of growth arising through nonhealing extraction socket since 3 months.

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Three months back the patient developed diffuse extraoral swelling over lower left side of the face. After consultation with a local dentist, the patient underwent extraction of lower left first, second, and third molars. However, the swelling persisted even after extraction. Few days after extraction, the patient noticed a small growth arising from extraction socket. It was tender and used to bleed spontaneously. No history of paresthesia/numbness with the concerned region was reported.

The patient also revealed the presence of a small lump in the neck region, which she noticed 3–4 months prior; however, she neglected it as it was asymptomatic.

The patient was averagely built and had been cleaning her teeth with *mishri* (fine black powder of roasted tobacco leaves) since last 30–35 years.

General examination revealed the presence of lump in the midline of anterior region of neck; it was about $3.5 \text{ cm} \times 3 \text{ cm}$ in size, soft to firm in consistency and was nontender. It used to move with swallowing.

Extraoral examination revealed swelling in the lower left side of the face. The swelling was oval shaped and was extending horizontally from mid-portion of left body of mandible to left angle of mandible and vertically from mid-ramus region to inferior border of mandible. It was tender, about 4.5 cm \times 4.5 cm in size with bony hard consistency. The patient also presented with bilateral submandibular lymphadenopathy.

Intraoral examination revealed sessile growth [Figure 1] arising from extraction socket of lower left first, second, and third molars. It was about $3.5 \text{ cm} \times 2 \text{ cm} \times 2.5 \text{ cm}$ in size with soft consistency and corrugated surface.

As the growth was arising from an extraction socket and the patient was a chronic tobacco chewer, a provisional diagnosis of malignant tumor of mandible was considered. The patient was then referred for radiographic examination, computed tomography (CT) scan imaging, complete blood count, and incisional biopsy of the intraoral growth.

OPG [Figure 2] revealed an osteolytic lesion in the lower left first, second, and third molars, which was ill defined, uncorticated. A pathologic fracture of the inferior border of the mandible was also noticed. Bone and soft tissue algorithm of CT scan [Figures 3 and 4]examination revealed destructive lesion involving posterior region of body and ramus of the left mandible. On careful examination, soft tissue window revealed an expansile lesion with hyperintense periphery involving thyroid gland.

Incisional biopsy of the intraoral growth revealed a metastatic follicular thyroid carcinoma.

The patient was then referred to higher center for further management.

Discussion

Follicular thyroid cancer tends to be a malignancy of older persons, with the mean age of patients in most studies being more than 50 years. Although papillary thyroid carcinomas are generally more common than follicular cancers, the latter are



Figure 1: Intraoral photograph showing granulomatous growth arising from extraction socket

more prone to spread hematogenously, especially to lung and bones, with a rate of 5%–20%. Conversely, follicular cancers exhibit a relatively small propensity for lymphatic spread.^[6]

Metastasis is a consequence of complex biologic cascade that begins with the detachment of tumor cell from primary tumor spreading into the tissues, invading the lymphovascular structures followed by their survival in the circulation.^[7] The microvasculature of the target organ provides room for the metastatic tumor cells to harbor, from where they can extravagate, proliferate, and invade within this target tissue. Angiogenesis is mandatory for the tumor cell load beyond 2-3 mm for adequate supply of oxygen and nutrients.^[8] Recent studies on the mechanism by which cancer metastasizes to bone have shown that cancer cells alter the physiologic balance between bone resorption and bone formation. Breast cancer metastases are frequently osteolytic and this has been attributed to overexpression of osteoclasts, inducing factors such as parathyroid hormone-related protein, interleukin (IL)-8, and IL-11.^[9] Predominantly osteoblastic metastasis is mediated by osteoblast-mediating factors, such as bone morphogenetic proteins, Wnt family ligands, endothelin 1, and platelet derived growth factors (PGDF). Furthermore, the release of matrix embedded growth factors, such as insulin-like growth factors



Figure 2: Orthopantomograph showing ill-defined osteolytic lesion with islands of remaining bone within the interior and pathologic fracture of lower border of mandible left side. (Digitally enhanced image)



Figure 3: Coronal section at bone window level showing osteolytic lesion causing erosion of lower border of left mandible

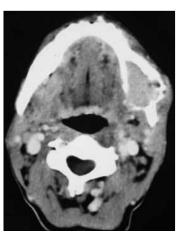


Figure 4: Axial section at soft tissue window level showing destructive lesion involving posterior region of body and ramus of left mandible

and transforming growth factor-beta upon osteolysis promotes the induction of osteoclast-promoting factors.^[10]

Reports from different parts of the world show a variable incidence of metastasis to jaw bone from different primary sites, ranging from 1–4 cases per year.^[1,7] Most of the metastatic tumors occur in 5th, 6th, and 7th decades^[3,7,11]; however, in an Indian study the metastatic tumors were found to occur at an early age between the 3rd and 7th decade. In the younger age group (first to second decade) the metastasis was found to occur from adrenal neuroblastoma, medulloblastoma, and osteogenic sarcoma. ^[12] The clinical presentations of the metastatic lesions include pain, swelling, loosening of tooth, paresthesia, and pathologic fracture. ^[1,3] Less frequently the lesion can present as pain in the temporomandibular joint region or as an osteomyelitis in the jaw or as trigeminal neuralgia.^[13] Studies have shown that chronic trauma to the oral tissue favors metastatic spread to the oral cavity. ^[14] In another study it was found that in 55 cases tooth extraction preceded the discovery of metastasis.^[15] In the majority of the cases, a latency period of 2 months between the extraction and the development of the metastasis was reported. In our case also we observed a similar finding. Thus the role of trauma to the oral mucosa in the causation of oral metastasis needs further investigation.

Emre and Ehab studied various cases and observed that the most frequent location for metastasis among jaw bones is mandible. In the mandible, ramus and angle are more commonly involved. They concluded that the propensity of posterior mandible for metastasis is due to its better vascularity.^[16] A few investigators believe that metastasis to jaw bone through hematogenous route requires the presence of hematopoietically active bone marrow well connected with the sinusoidal vascular spaces at the site of deposition of malignant cells.^[17,18]

The posterior mandible and the focal osteoporotic bone marrow defects in the edentulous mandible have been shown to be the hematopoietically active sites that may attract the metastatic tumor cells.^[19,20] Still some other investigators believe that the high bone turnover in this region may be the cause.^[21]

Although the incidence of metastatic tumors in oral cavity is considered low, a significant number of such lesions have a high tendency to go undetected, this is because of the following:

- A. Micrometastasis is rarely detected by radiographic survey.^[22]
- B. Cases with poor prognosis and terminal stage of the disease lose or are dead before presenting to a clinician.^[12]
- C. Earlier jaw bones were not included in the radiographic survey for metastatic workup.^[3]

The exact incidence of metastatic diseases that affect the mandible is still unknown. Hence all medical and dental clinicians must include malignant disease, primary or metastatic, alongside the more common benign pathologies when considering the differential diagnosis of oral complaints. This is particularly important in the primary care setting, especially when dealing with elderly patients, or those with a history of malignancy.

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