

Gastroesophageal junction adenocarcinoma metastasizing to gingiva

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ABSTRACT

Malignant tumors rarely metastasize to the oral cavity and account for about 1% of all oral tumors. Metastasis to soft-tissue of the oral cavity is much rarer than jaw bones. Because of the rarity, metastatic tumors in the oral region are challenging for diagnosis. Primary tumors which metastasize to mouth are the most commonly lung, breast, and kidney. Oral cavity metastases represent distant spread and are associated with poor prognosis with short survival. We present a case of the gastroesophageal junction adenocarcinoma with metastasis to the oral soft-tissue.

Key words: Adenocarcinoma, metastases, oral cavity

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INTRODUCTION

Metastatic tumors in the oral cavity are uncommon. Review of the literature revealed only 63 cases of gingival soft-tissue metastases. They represent approximately 1% of all malignant tumors affecting the oral cavity.^[1] Metastasis to the jaw bones is much more common than oral soft-tissue, most common site being the mandible (82-90%) followed by the maxilla (10-20%) and the metastases to perioral soft-tissue accounts for only 15% of cases.^[2] Molar and pre-molar regions are most commonly affected. Location in the soft-tissue is mainly gums.^[3] They usually occur as gingival hyperplastic or reactive lesions with the clinical appearance of pyogenic granuloma or epulis, lesions with which they can be confused, even with the possibility of being multiple and bilateral.^[2] Seventy percent of oral metastases are manifested after the primary tumor becomes evident while the remaining 30%

are the first clinical sign of the tumor. Fifty percent of all oral metastases are caused by tumors from breast, lung, and kidney. The remaining are located in prostate, thyroid, gastrointestinal tract (mainly seen in Japanese), suprarenal, uterus, bones etc.^[2,3] The mean age of occurrence is 5th and 6th decades though it may occur at any age.^[3] There are no significant differences regarding sex; however, it is clear that the primary tumors vary depending on the sex.^[2] The occurrence of a metastatic lesion to the oral cavity is generally associated with poor prognosis having median survival of 6 months.^[3] Lower than 10% of patients survive 4 years after diagnosis.^[2]

CASE REPORT

A 60-year-old male patient, known case of adenocarcinoma of gastroesophageal (GE) junction on chemotherapy, presented with a soft exophytic swelling over gingiva of right lower jaw in the first and second pre-molar space. It gradually increased in size with loosening of the overlying teeth. Oral examination showed a soft swelling measuring 3 cm × 2 cm covered by necrotic tag. The swelling resembled periodontal pyogenic abscess or granuloma like hyperplastic lesion [Figure 1]. There was no evidence of lymphadenopathy. Patient was a chronic tobacco chewer and alcoholic. He had a significant dysphagia 1 year back. During his work-up, computed tomography scan revealed a growth at GE junction [Figure 2], which was reported as adenocarcinoma on histopathology [Figure 3]. Patient was put on chemotherapy.

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Biopsy from the gingival mass was taken. Grossly, we received three tiny pieces of grey white tissue, which were submitted entirely. Microscopy revealed a tumor composed of cuboidal to columnar cells having pleomorphic vesicular nuclei with prominent nucleoli and a moderate amount of eosinophilic to clear cytoplasm arranged predominantly in papillary and focally in acinar pattern. Few cells with bizarre nuclei and signet ring configuration were noted. Areas of tumor necrosis were seen. A diagnosis of papillary adenocarcinoma was offered [Figure 4]. Slides of the primary tumor were reviewed. Microscopy of the primary tumor and gingival mass showed a similar histological feature. Hence the diagnosis of metastatic adenocarcinoma was confirmed. Following this, the patient received one cycle of local radiotherapy.

DISCUSSION

Metastasis to the oral cavity is very rare accounting for about 1% of all oral tumors and metastasis to oral soft-tissue is still rarer.^[1,3] In the oral cavity though any site may be affected,

the most common primary site for oral soft-tissue metastases is gingiva (55%) because of its fine capillary bed followed by tongue (30%).

Metastasizing is a complex process, the biological basis of which requires tumor cells to breach a sequence of barriers. First, they have to detach from the primary tumor; then they must spread in the tissue, invade the blood or lymphatic vessels and survive travel in the circulation. After this, they have to settle in the microvasculature of the organ, extravasate through the vessel wall, invade the target organ and proliferate within the target tissue. For a micro metastasis to grow beyond the size of 2-3 mm, tumor cells have to induce the formation of new blood vessels (angiogenesis) for adequate supply of oxygen and nutrients.^[3] Chronically swollen gums with rich vascular neoformation must be considered in the attraction of metastatic cells since fragmented basal membrane of newly formed vessels (neocapillaries) can be more easily penetrated by tumor cells than mature vessels.^[2] One possible explanation for blood borne metastases to



Figure 1: Clinical photograph of gingival growth

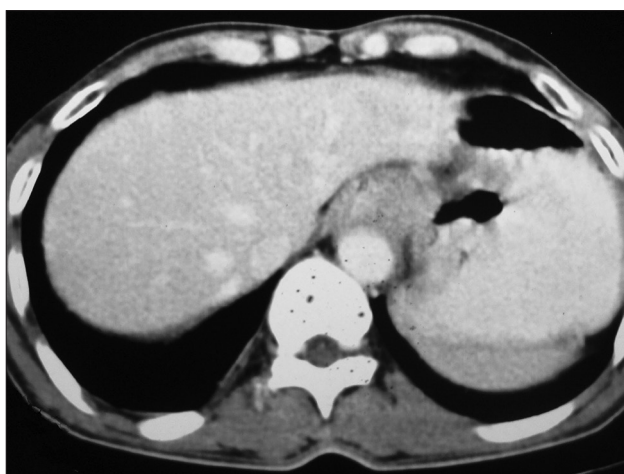


Figure 2: Computed tomography scan showing gastroesophageal junction growth

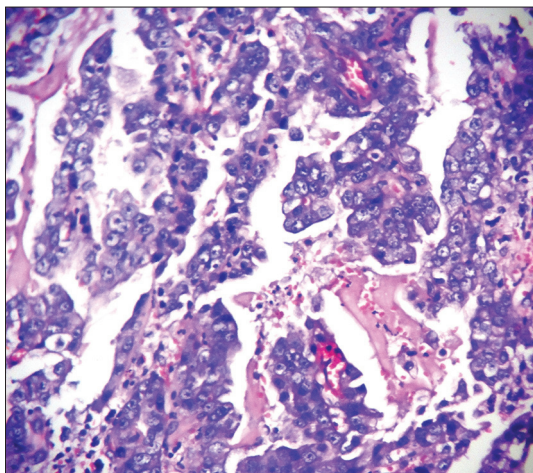


Figure 3: Biopsy from the gastroesophageal junction growth, showing adenocarcinoma (H and E, x400)

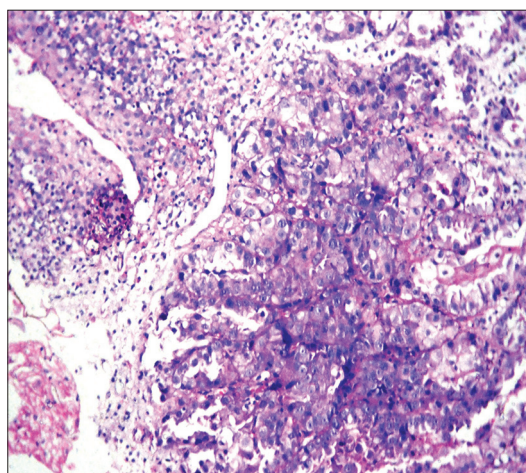


Figure 4: Biopsy from gingival growth with similar histomorphological features as Figure 3 (H and E, x400)

the head and neck, especially, in the absence of pulmonary metastases is Batson's plexus, a valveless vertebral venous plexus that might allow retrograde spread of tumor cells bypassing filtration through the lungs.^[4]

Adenocarcinoma of GE junction remains a significant clinical problem that is increasing in incidence and is associated with a poor prognosis. Siewert and Stain have proposed a clinical classification system, Type I to III for GE junction cancer to aid the clinicians in developing treatment strategies for this heterogenous clinical entity.^[5]

Type I: Adenocarcinoma of the distal esophagus arising from intestinal metaplasia, i.e., Barrett's esophagus and may infiltrate the GE junction from above.

Type II: True carcinoma of the cardia arising from the cardiac epithelium or short segments with metaplasia at the GE junction.

Type III: Subcardial gastric carcinoma infiltrating the GE junction and distal esophagus from below.

According to this classification, our case is Type III. Patients with Type I tumors are more likely to have a hiatal hernia and a long history of gastro esophageal reflux disease than patients with Type II or III tumor. The so called Barrett's esophagus with subsequent development of progressively severe dysplastic changes is the main precursor lesion for adenocarcinoma in the distal esophagus (found in more than 80% of patients). Type II and III tumors may also arise from short segments of intestinal metaplasia at or below the cardia; however, it happens in less than 40% of patients with carcinoma of the cardia and in less than 10% of those with subcardial carcinoma. There is a strong association between *Helicobacter pylori* and intestinal metaplasia at or below the gastric cardia. On the contrary, the specialized intestinal metaplasia in the distal esophagus is reflux related. The prevalence of undifferentiated tumor and tumor with a non-intestinal growth pattern is rather low in Type I tumors and increases significantly from Type II to III tumors. Our patient had kept regular follow-up for 2 months and he expired 3 months after the diagnosis. The overall survival rate is poor in most patients with adenocarcinoma of GE junction because lymph node or

visceral metastases are frequently present at the time patients become symptomatic.^[6]

Inflammatory and reactive lesions are more common in the oral soft tissue and include lesions such as pyogenic granuloma, periodontal abscess or inflammatory hyperplastic lesion making it imperative that metastases could be included in the differential diagnosis of gingival masses. Most patients complain of swelling, pain or paresthesia that develop in a relatively short time.^[7] The main symptoms are bleeding and a rapidly growing ulcerated mass. Diagnosis requires careful history taking particularly of previous surgeries. Oral metastasis is usually an evidence of widespread disease. Because the prognosis is poor, treatment is palliative in most cases. Surgery is usually undertaken when the primary tumor is controlled and there is no evidence of other metastases. Most patients die within 1 year after the diagnosis of metastasis. Therefore, the clinicians should be aware of clinical and prognostic implications of oral metastatic lesions.^[8]

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