International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor (2018): 7.426

Diagnostic Dilemma–Oral Squamous Cell Carcinoma

Neha Singh[1], Ritika Sharma[2], Deepak Bhargava[3], Mithilesh Mishra[4], Vidya Devichandavarkar[5], Syed Mustansir Ul Hassnain[6]

[1]Post Graduate Resident, Sharda University, Greater Noida

[2, 4, 5]Reader, Sharda University, Greater Noida

[3]HOD & Professor, Sharda University, Greater Noida

Abstract: ***Incidence of oral cavity cancer is 2.7% and age-standardized rates (ASR, world standard population) is 5.5. The mortality rate is 2.1% and ASR (W) is 2.7 and 5-year prevalence is 3.1% according to International Agency for Research on Cancer, WHO. In India and Asian countries, oral cancer is most common type of malignancy and account for more than 50% of all cancer cases. This finding is generally linked to the high prevalence of a unique smokeless tobacco habit. This article reports a case of osteomyelitis of mandibular jaw which represented as a diagnostic dilemma in regards to the oral cancer.***

Keywords: Oral squamous cell carcinoma, Osteomyelitis

1. Introduction

Cancer is the end product of an unregulated proliferation resulting from accumulation of sequential genetic mutations in precursor cell. Oral squamous cell carcinoma is misnaming because cancer-generating cells are those within the basal cell layer of the mucosa and known as squamous cell carcinomas only because the malignant squamous cell precursors (basal cells) undergo partial squamous differentiation, making them look like squamous cells under the light microscope. [1]

Osteomyelitisis defined as the inflammation of bone and marrow. Itis possibly complication of any systemic infection but frequently manifests as a primary solitary focus of disease. Most common organisms like certain pyogenic bacteria and mycobacteria can cause osteomyelitis. [2]

When patient presents with an area of exposed bone with abnormal surrounding soft tissue changes, the diagnostic differentiation between ulcerative and malignant disease canbe challenging, and even the histopathological comparison may prove difficult. Here we describe a case,initially diagnosed as osteomyelitis but after biopsy it was diagnosed as carcinoma.

2. Case Report

A 60 year male patient was referred to the department of oral pathology withpainless ulcers inlower left side of mandible accompanying closed sinus tract on chin since 6 months (Figure 1).

Figure 1: No abnormality detected on extraoral examination

He was smoking 10 bidis/day since 25 years. Intraoral examination revealed ulcerated lesionin left body of mandible with exposed bone and surrounding ulcerated hypergranulation tissue with everted margins. It was tender on palpation with nopus discharge (Figure 2).

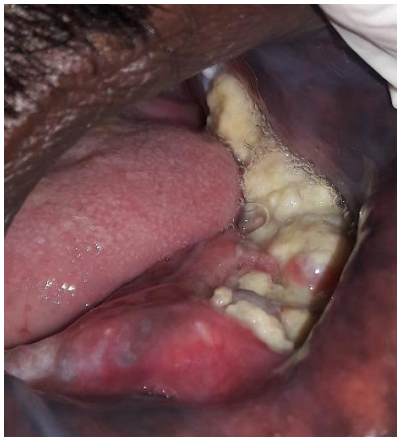


Figure 2: Intraoral examination of left posterior side of mandible

Orthopantomograph (OPG) showed an osteolytic lesion with irregular margins in left posterior mandibular region (Figure 3).

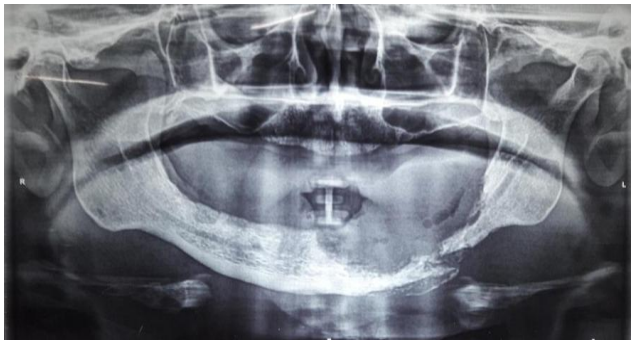


Figure 3: Orthopantomograph showing an osteolytic lesion in the left posterior mandible

Contrast-enhanced computed tomography (CECT) neck showed destruction of the left ramus of mandible with adjacent periosteal reaction suggestive of questionable osteomyelitis. Adjacent soft tissues showed swelling and inflammatory changes. Multiple bilateral levels I and II lymph nodes were enlarged. (Figure 4).

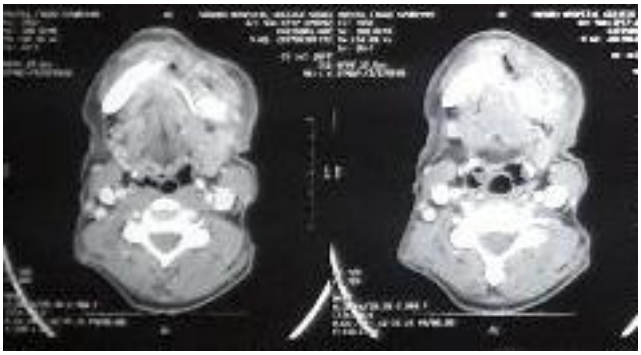


Figure 4: Computed tomography after injection of contrast in soft tissue window showed an osteolytic lesion in left posterior mandible associated with cortical erosion

Complete blood count (CBC) indicated that total leucocyte count (TLC), urea, uric acidand SGOT (AST) was raised and chloride was decreased.Incisional biopsy was done and tissue was sent for histopathological diagnosis.

Grossing of multiple soft tissuebits was done and histopathological finding stated as 'well differentiated squamous cell carcinoma'.Patient was operated& multiple tissues were again sent for histopathological diagnosis.

Segmental mandibulectomy specimen was received along with submandibular salivary gland and lymph node (Figure 5).

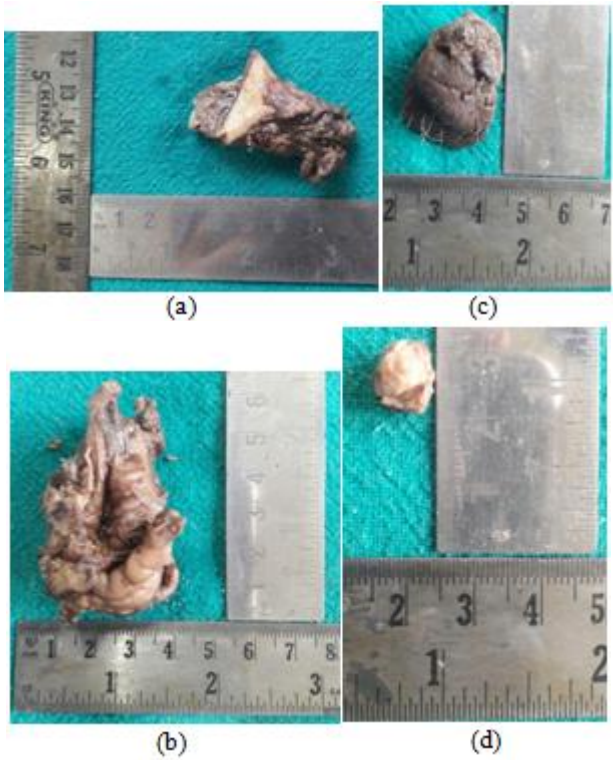
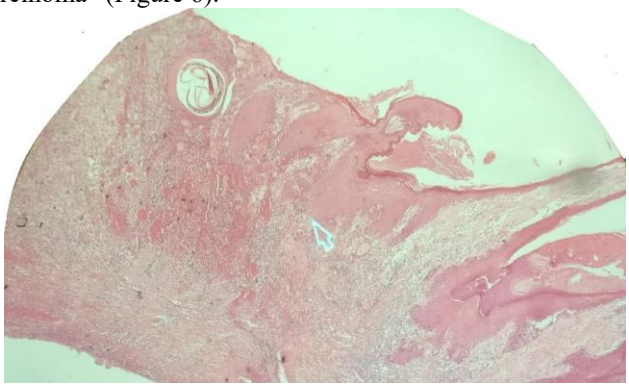
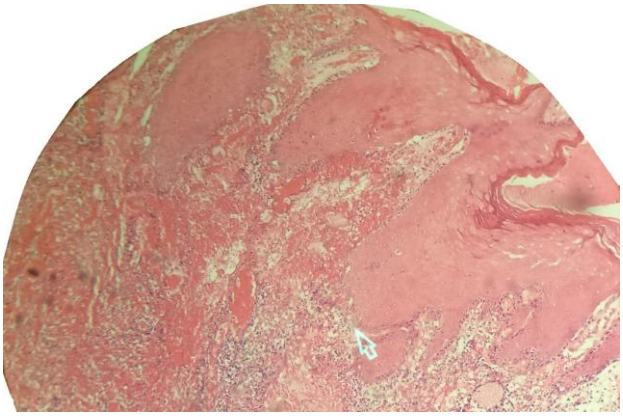


Figure 5: Grossing of multiple bits of hard and soft tissue specimen, (a) Tumor mass, (b) Submandibular salivary gland, (c) Surface lesion with sinus tract, and (d) Single submandibular lymph node (level 1B)

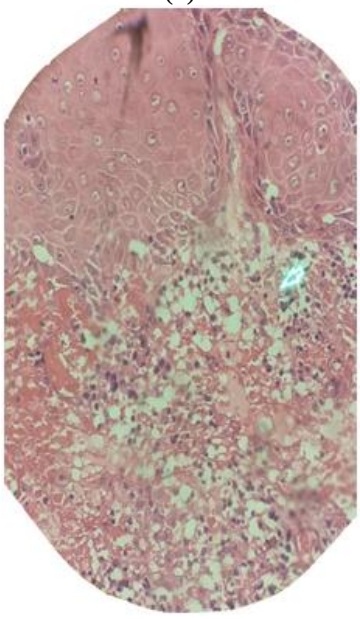
Histopathological examination showed hyperplastic parakeratinized stratified squamous epithelium with islands of atypical squamous cells invading into the connective tissue stroma. Dysplastic features like cellular and nuclear pleomorphism, nuclear hyperchromatism, increased nucleo-cytoplasmic ratio, increased mitotic figures and keratin pearls were seen. Connective tissue stroma showed dense collagen bundles interspersed with fibroblast, red blood cells, adipose tissue, chronic lymphocytic infiltrate. Invasion of squamous cells was seen in the lymph node level 1B. Anterior and posterior tumor margin were free from tumor infiltration. Itwas diagnosed as "well differentiated squamous cell carcinoma" (Figure 6).



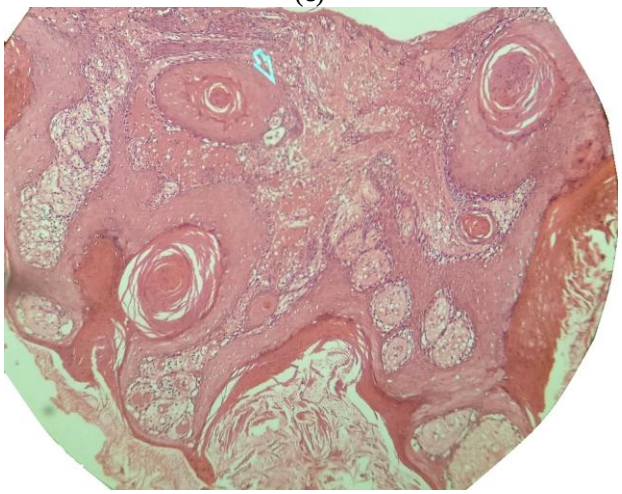
(a)



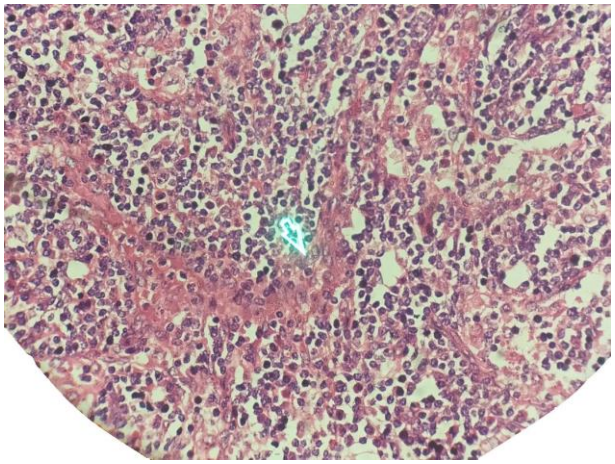
(b)



(c)



(d)



(e)

Figure 6:- (a) Photomicrograph seen in scanner (5X) showed hyperplastic parakeratinized stratified squamous epithelium &breach in the basement membrane (arrow),(b) Photomicrograph seen in 10X shows breach in the basement membrane (arrow) & Connective tissue stroma showed dense collagen bundles & chronic lymphocytic infiltrate, (c) Photomicrograph seen in 40X showed islands of atypical squamous cells invading into the connective tissue stroma (arrow) & dysplastic features of squamous cells, (d) Photomicrograph seen in 10X showed keratin pearls formation & individual cell keratinization (arrow), (e) Photomicrograph seen in 40X showed atypical squamous cells (arrow) in lymph node level 1B (Hematoxylin-Eosin stain).

Treatment

Segmental mandibulectomy along with titanium mandibular reconstruction plates were placed within mandible alongside radiotherapy.

Follow Up

After 10 months, patient complained of pain inthe same region. Radiographic finding revealedinfection of reconstructed plate. Plate was removed, lesion showed poor wound healing, re-occurrence of ulcerated areas and asymmetry of the face.

Figure 7: Asymmetrical face with deviation towards the left side on extraoral examination

Intraoral examination revealed multiple ulcerative growth with rolled out, everted margins. It wastender on palpation and no pus dischargein anterior front mandibular region.

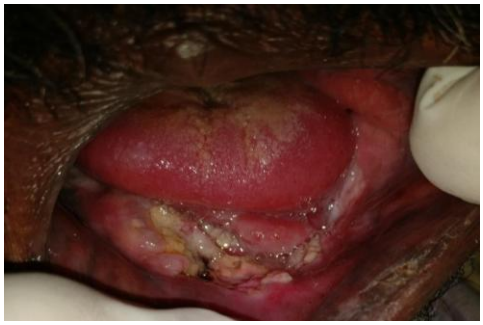


Figure 8: Intraoral examination of mandibular anterior region

3. Discussion

Squamous cell carcinomais the commonest malignant neoplasm of mouth. Tumors arise on lower lip and at different intraoral sites like lateral margin of the tongue and floor of the mouth.[3]

The cause of oral squamous cell carcinoma is multifactorial (extrinsic and intrinsic factors).Extrinsicfactors includeexternal agents like tobacco smoke, alcohol, syphilis and sunlight. Intrinsicfactors include systemic or generalized states, suchlike general malnutrition or iron-deficiency anemia. The proportion of smokers (80%) among patients with oral carcinoma is two to three times greater than the general population.[4]

In osteomyelitis, changes in calcified tissue are secondary to inflammation of the soft tissue component of the bone. It commonly occurs as a complication of dental sepsis and predisposing factors include fractures due to trauma& radiation. The disease maybe acute, subacute, or chronic and presents a different clinical course, depending upon its nature. [5]Osteomyelitis is facile because ofcharacteristic clinical and roentgenographic features. Acute osteomyelitis can simulate neoplasms such as Ewing's tumor and malignant lymphoma on roentgenograms. Chronic osteomyelitis can produce areas of destruction simulating neoplasm such as giant cell tumor, or may form a focal abscess simulating osteoid osteoma. Development of malignant disease in osteomyelitis of long duration with draining sinus, is rare but well-known complication. [6]

Oral cancer arises from the accumulation of a number of discrete genetic events that lead to invasive cancer. These changes will occur in genes that encode for proteins that control the cell cycle, cell survival, cell motility, and angiogenesis.[7]

A change in surface texture of the lesion or presence of mass or ulceration should be assessed. Lesion may be flat or elevated and ulcerated or nonulcerated. Lymphatic spread involves the submandibular, digastric nodes and upper cervical nodes.[8]

The diagnosis of squamous cell carcinoma essentially confirmed by biopsy, which is taken from the clinically most suspicious area, avoiding central necrotic area. Well-differentiated squamous cell carcinomas must be distinguished from verrucous carcinomas and papillary squamous cell carcinomas, and furthermore from benign conditions, suchlike pseudoepitheliomatous hyperplasia.[9]

The principle objective of treatment is to cure the patient which depends on factors like cell type and degree of differentiation; site, size, and location of the primary lesion; lymph node status; presence of bone involvement; ability to achieve ample surgical margins. Chemotherapy& radiotherapy is an adjunct to the principal therapeutic modalities. [8]

Despite such approach, the majority of patients develop local recurrences and distant metastases in 20%–30% of patients. Treatment options comprises supportive care only, or with addition single-agent chemotherapy, combination chemotherapy or targeted therapies either alone or in combination with cytotoxic agents. [10]

It is the responsibility of every dental practitioner to educate, detect, diagnose and refer the case of oral cancer so as to reduce the morbidity and mortality rates. Meeting these responsibilities will generate a much higher cure rate as well as survival rate.

References

[1] Marx RE, Stern D. Oral and maxillofacial pathology- A Rationale for Diagnosis and Treatment. 1st ed.Quintessence: Chicago; 2003.p.295.
[2] Kumar V, Abbas AK, Fausto N. Robbins and Cotran Pathologic Basis of Disease. 7th ed. Noordanesh Medical Publications. Elsevier Health Sciences: China; 2017.p1290.
[3] Fletcher CD.M. Diagnostic histopathology of tumors. 4th ed/Vol 1. Elsevier Health Sciences: China; 2007. p. 246-247.
[4] Neville BW, Damm DD, Allen CM, Bouquot JE. Oral and maxillofacial pathology. 2nd ed. Elsevier Health Sciences: Philadelphia; 2015. p. 356-357.
[5] Rajendran R, Sivapathasundharam B. Shafer's textbook of oral pathology. 5th ed. Elsevier India:Noida; 2006. p. 682-684.
[6] Sternberg SS, Antonioli DA, Carter D,Mills SE, Oberman HA. Sternberg's diagnostic surgical pathology. 3rd ed. Lippincott Williams & Wilkins: Philadelphia; 2004. p. 305.
[7] Regezi JA, Sciubba JJ, Jordan RCK. Oral pathology: clinical pathologic correlations. 4th ed. Elsevier Health Sciences: United States of America; 2016 .p. 53.
[8] Burket LW, Greenberg MS, Glick M. Burket's oral medicine: diagnosis & treatment. 10th ed. BC Decker Inc: Spain; 2003. p. 203, 206-207.
[9] Cardesa A, Slootweg PJ, Gale N, Franchi A. Pathology of the Head and Neck. Springer: New York; 2017. p. 15.
[10] Vermorken JB, Specenier P. Optimal treatment for recurrent/metastatic head and neck cancer. Annals of oncology. 2010 Oct 1;21(7):252-61.

Author Profile

Dr Neha Singh, Post Graduate Resident, Sharda University, Greater Noida

Dr Ritika Sharma, Reader, Sharda University, Greater Noida

Dr Deepak Bhargava, HOD & Professor, Sharda University, Greater Noida

Dr Mithilesh Mishra, Reader, Sharda University, Greater Noida

Dr Vidya Devichandavarkar, Reader, Sharda University, Greater Noida

Syed Mustansir Ul Hassnain