Available online at http://www.journalcra.com

International Journal of Current Research
Vol. 9, Issue, 12, pp.62854-62859, December, 2017

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

ISSN: 0975-833X

CASE STUDY

INFARCTION OF MAXILLA : A RARE CASE REPORT

***,[1]Dr. Poulomi Bhakta, [2]Prof. (Dr.) Prashant Nahar, [2]Prof. (Dr.) Mohit Pal Singh, [2]Dr. Bhuvaneshwari, S., [1]Dr. Saurabh Goel and [3]Dr. Rudra Prasad Chatterjee**

[1]Department of Oral Medicine and Radiology, Pacific Dental College and Research Centre, Udaipur, Rajasthan
[2]Department of Oral Medicine and Radiology, Pacific Dental College and Hospital, Udaipur, Rajasthan
[3]Department of Oral & Maxillo-facial Pathology and Oral & Dental Anatomy and Histology, Guru of Nanak Institute Dental Sciences and Research, Panihati, Kolkata

ARTICLE INFO

Article History:
Received 12th September, 2017
Received in revised form
19th October, 2017
Accepted 22nd November, 2017
Published online 27th December, 2017

Key words:
Osteonecrosis,
Maxilla,
Ischemia.

ABSTRACT

Osteonecrosis of the jaw is a disease of bone where symptomatic exposure of nonhealing areas over jaws are seen. We report our experience in the management of a patient with history of trauma to nose, osteoarthritis, diabetes mellitus & hypertension, who presented with osteonecrosis of the jaw and was under medications but was unaware of the name of the medications. The patient was found to be of an average built, had normal gait with no physical handicap and was alert, conscious and cooperative and responsive to verbal commands with vital parameters within normal range. Intraoral clinical examination revealed poor oral hygiene with bony exposure & necrotic appearance of the maxillary alveolus in the edentulous 11, 12, 13, 14, 15 & 21, 22, 23, 24, 25 region. To confirm the diagnosis, patient was subjected to radiographic, laboratory and histopathological investigations and was advised to take Clindamycin capsules, supplementation of Antioxidant, Calcium capsules and topical application of Metronidazole gel & oral rinse with Povidone iodine 2% Gargles. Based on this diagnosis, patient underwent surgical debridement and finally resection of bony segment of maxillary alveolus extending from 15 to 25 region in the Department of Oral & Maxillofacial Surgery. After the surgery, patient had an acceptable maxillary alveolus and was put on periodic recall for six months. But *there was recurrence after 19*th months.

Citation: Dr. Poulomi Bhakta, Prof. (Dr.) Prashant Nahar, Prof. (Dr.) Mohit Pal Singh, Dr. Bhuvaneshwari, S., Dr. Saurabh Goel and Dr. Rudra Prasad Chatterjee, 2017. "Infarction of maxilla : A rare case report", *International Journal of Current Research*, 9, (12), 62854-62859.

INTRODUCTION

A 60 –year old male reported to Department of Oral medicine, Diagnosis & Radiology, Pacific Dental College & Hospital, Airport road, Debari, Udaipur with a chief complaint of absence of few teeth in relation to upper jaw since 7 months. Medical history revealed that he was suffering from Hypertension, Hyperglycemia and Osteoarthritis and was under medication but was unaware of the name of medications. Patient also had a history of trauma to the nose one year back. The patientwas found to be of an average built, had normal gait with no physical handicap and was alert, conscious and cooperative and responsive to verbal commands with vital parameters within normal range. Extraoral examination of the patient revealed no facial asymmetry (Table/Fig-1). Intraoral clinical examination revealed poor oral hygiene, a complete set of permanent dentition with missing maxillary right & left central incisors, lateral incisors, canines, premolars, right maxillary third molar, left maxillary second and third molars, mandibular right and left third molars. Bony exposure with necrotic appearance, yellowish-brown in colour and porous surface on the labial side of the maxillary alveolus was revealed in the edentulous 11, 12, 13, 14, 15 & 21, 22, 23, 24, 25, 26 region and surrounding soft tissues were erythematous and edematous (Table/Fig-2a, 2b). In addition, the upper right & left maxillary first molars exhibited advanced mobility. On examination, we also found carious 17, 36, 37, 46, 47 and class I malocclusion.Based on history and clinical findings, the provisional diagnosis of suspected Osteonecrosis of Maxillary arch was made. Patient was subjected to radiographic, laboratory and histopathological investigations. OPG (Table/ Fig-3) revealed maxillary alveolar bony resorption with loss of bony trabeculae evident in relation to edentulous 11, 12, 13, 14, 15, 21, 22, 23, 24, 25, 26 region, Distoangularly impacted 18, 28 & Mesioangularly impacted 38&Horizontally impacted 48 and Dental caries with pulpal involvement in relation to 17. Maxillary occlusal cross sectional view (Table/Fig-4) revealed maxillary alveolar bony resorption with loss of bony trabeculae evident in relation to edentulous 11, 12, 13, 14, 15, 21, 22, 23, 24, 25 region. Complete Blood Count (Table/Fig-5a, 5b) revealed leukocytosis (15.01 thou/mm^3), neutrophilia (11.87 thou/mm^3), raised ESR values (23mm/hr) with other normal parameters. Bone biopsy (Table/Fig-6) showed trabeculae of bones intermingling with each other, admixed with marrow tissues and empty osteocytic lacunae.

Corresponding author:* **Dr. Poulomi Bhakta,
Department of Oral Medicine and Radiology, Pacific Dental College and Research Centre, Udaipur, Rajasthan, India.

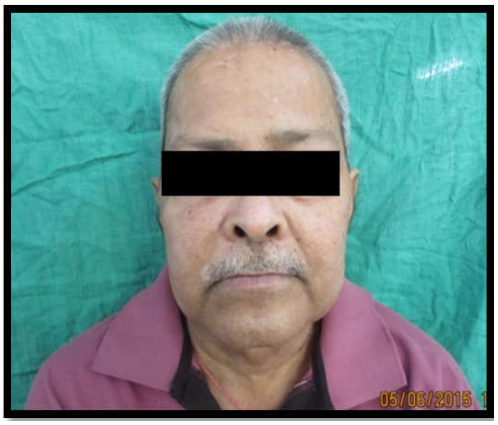

Fig.1. Frontal Profile View showing no facial asymmetry

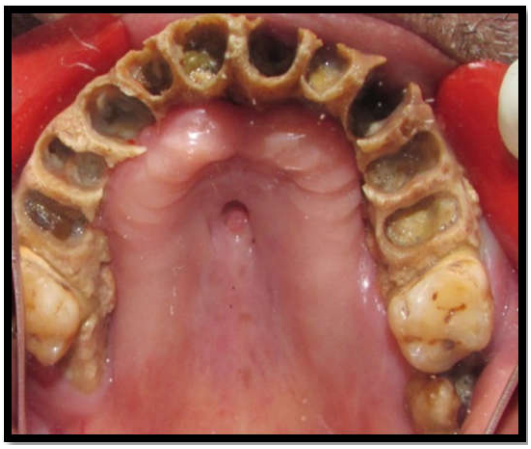

Fig.2a. Intraoral picture showing Palatal view of maxillary arch

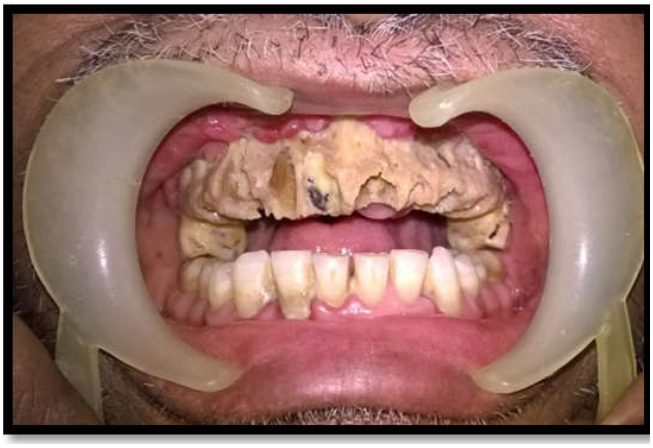

Fig.2b. Intraoral picture showing Labial view of maxillary & mandibular arch

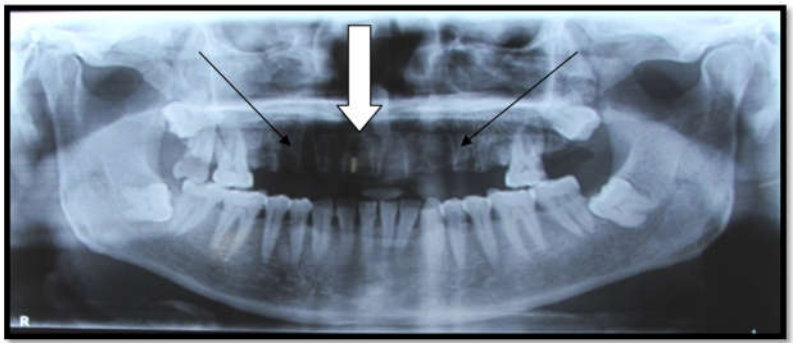

Fig.3. Pre-operative OPG view showing (black & white arrows) maxillary alveolar bony resorption with loss of bony trabeculae

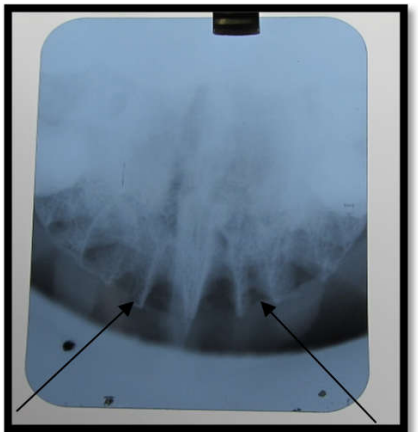

Fig.4. Maxillary Occlusal Cross-sectional view Showing maxillary alveolar bony resorption with loss of bony trabeculae

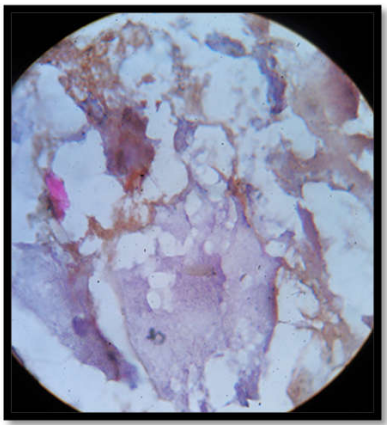

Fig.6. Bones intermingling with one trabeculae of another, admixed with marrow tissues and empty osteocytic lacunae

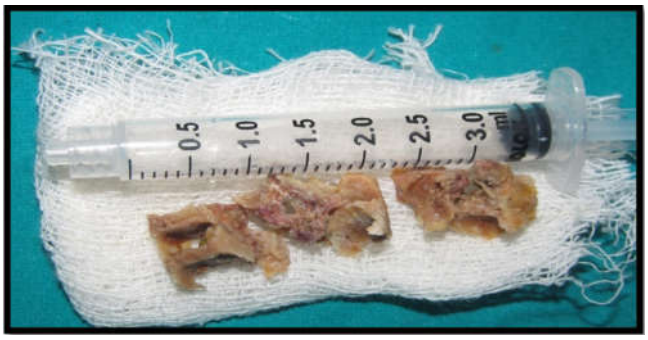

Fig.7. Necrotic bones

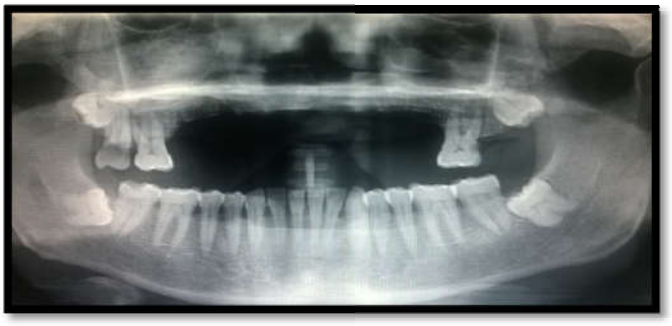

Fig.8. Post-operative OPG view

Fig.9. Dislodged Premaxilla

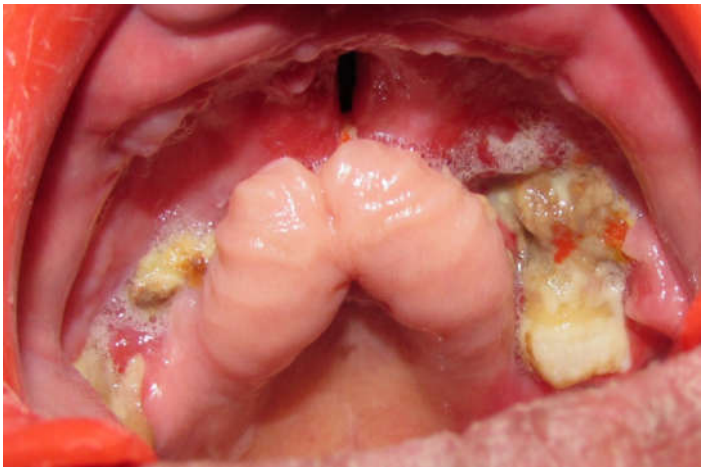

Fig.10. Intraoral clinical view showing oro-nasal communication

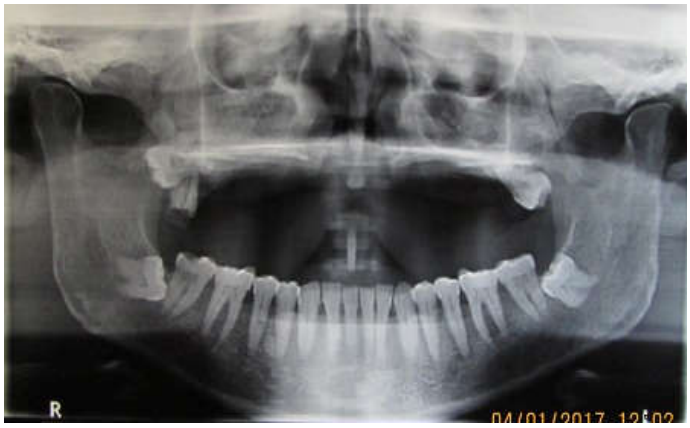

Fig.11. OPG View after 19 months follow-up

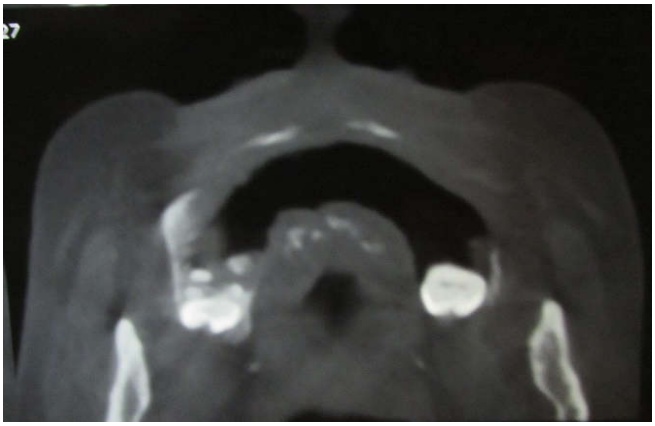

Fig.12a. Axial view of CBCT Maxilla

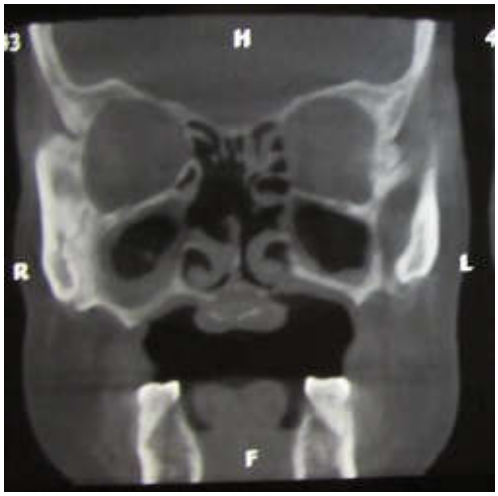

Fig.12b. Coronal view of CBCT Maxilla

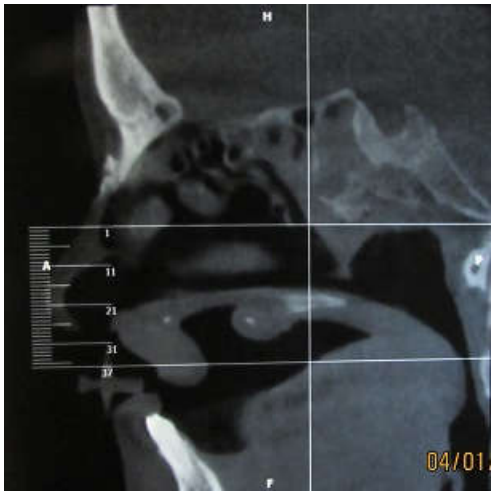

Fig.12c. Sagittal view of CBCT Maxilla

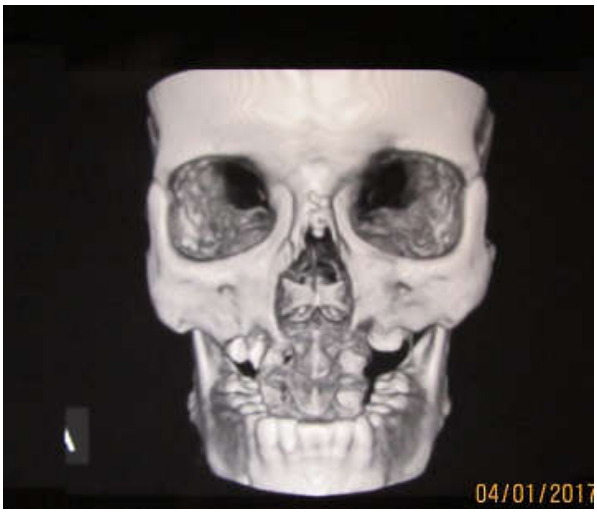

Fig.12d. 3D view of CBCT Maxilla

[Table/Fig-5a]

S. No.	Investigation	Result	Unit
1.	Hemoglobin	14.70	g/dL
2.	Blood sugar(R)	98.0	mg/dL
3.	Bleeding Time (B.T.)	1'30"	Minutes
4.	Clotting Time (C.T.)	5'35"	Minutes
5.	Packed Cell Volume (PCV)	43.50	%
6.	RBC count	4.74	mill/mm^3
7.	MCV	91.80	fL
8.	MCH	31.00	Pg
9.	MCHC	33.80	g/dL
10.	Red Cell Distribution Width (RDW)	15.50	%
11.	Total Leukocyte Count (TLC)	15.01	thou/mm^3
12.	Differential Leukocyte Count (DLC)		
	Segmented Neutrophils	79.10	%
	Lymphocytes	15.90	%
	Monocytes	4.00	%
	Eosinophils	0.70	%
	Basophils	0.30	%

[Table/Fig-5b]

S.No.	Investigation	Result	Unit
1.	Absolute Leukocyte Count		
	Neutrophils	11.87	thou/mm^3
	Lymphocytes	2.39	thou/mm^3
	Monocytes	0.60	thou/mm^3
	Eosinophils	0.11	thou/mm^3
	Basophils	0.05	thou/mm^3
2.	Platelet Count	416.0	thou/mm^3
3.	ESR	23	mm/hr
4.	HIV (I&II)	Negative	
5.	HBsAg	Negative	

The overall clinical, radiological and histopathological picture was suggestive of Osteonecrosis of Maxillary arch. Patient was advised to take Clindamycin capsules-300 mg (thrice daily for 5 days), supplementation of Antioxidant capsules (twice daily for 15 days), Calcium capsules (once daily for 15 days) along with Topical application of Metronidazole Gel and oral rinse with Povidone iodine 2% Gargles. Based on this diagnosis, patient underwent surgical debridement and finally resection of bony segment of maxillary alveolus extending from 15 to 25 region in the Department of Oral & Maxillofacial Surgery. The necrotic bone (Table/Fig-7) and overlying granulation tissue were removed and sent for histopathological evaluation, which confirmed the early histopathological diagnosis. After the surgery, patient had an acceptable maxillary alveolus (Table/Fig-8) and was put on periodic recall for six months.

On the first 6 months follow-up, there was no change in clinical appearance of maxillary alveolar ridge & palate. Following this, the patient did not come for next schedule follow-up (1 year). But the patient reported after 19th months with recurrence. On clinical examination, it was found that the complete premaxilla (necrosed bone) had got dislodged (Table/Fig-9). On detailed intraoral examination, oronasal communication was evident in premaxillary region (Table/Fig-10). Patient was subjected to radiographic & hematological investigations. OPG (Table/Fig-11) revealed maxillary alveolar bony resorption evident in relation to edentulous 11, 12, 13, 14, 15, 16, 21, 22, 23, 24, 25, 26, 27 region, Distoangularly impacted 18, 28 & Mesioangularly impacted with dilacerated root 38 & Horizontally impacted 48. CBCT of complete maxilla (Table/Fig-12a, 12b, 12c, 12d) was done which

revealed oro-antral & oro-nasal communication associated with associated with Pan Sinusitis, malunion fracture nasal bone and suspected chronic osteomyelitis of maxilla. Complete Blood Count revealed leukocytosis (11.67 thou/mm^3), neutrophilia (8.54 thou/mm^3), with other normal parameters.

DISCUSSION

The most common cause of osteonecrosis of the jaws is either exposure to bisphosphonate or radiation. In addition, osteonecrosis of the jaws is also caused by dentoalveolar trauma. Signs & symptoms of osteonecrosis of the jaws are exposure of jaw bones with or without pain, swelling and fistula formation. To prevent the osteonecrosis of the jaws nonrestorable or questionable teeth, root tips and periodontally compromised teeth should be extracted prior radiotherapy. Analgesics, topical antibiotic (ie, tetracycline), antiseptic (chlorhexidine) rinses, topical & systemic antimicrobials should be prescribed for the management of osteonecrosis of the jaws. Hyperbaric oxygen (HBO) therapy is another best treatment approach because it increases the oxygenation of tissue, angiogenesis and promotes functional activity of osteoblast and fibroblast (Joel Epstein 11th edition). Studies have shown that other than osteoradionecrosis, bisphosphonate induced osteonecrosis is systemic which is associated with decreased vascularity. So, hyperbaric oxygen therapy may not be helpful in treating patients with bisphosphonate induced osteonecrosis except osteoradionecrosis (Michael *et al*., 2005). According to studies, the occurrence of osteonecrosis in patients taking bisphosphonates is ranging between 0.8-12%, the most of which is associated with intravenous infusion of Zolidronic acid (Zometa) and Pamidronate (Amber Kiyani *et al*., 2009). According to some authors, the most common site of osteonecrosis are jaws because the jaws are the only bone structures which are attributed to continuous trauma and masticatory stress with exposure to environment and to oral microorganisms. Dental extractions, spontaneous exposures and necrosis of the alveolar bone have been reported as predisposing factor of osteonecrosis. Hellstein *et al*. reported the term bisphosphonate osteochemonecrosis or bis-phossy jaw because the features of bisphosphonate associated osteonecrosis and phossy jaw are similar (Rajendran and Sivapathasundharam, 7th edition). Bisphosphonates i.e synthetic analogues of inorganic pyrophosphates get incorporated in skeletal bone and bind to Ca^{2++} in regions of high bone turnover and remain incorporated for more than 10 years. After incorporation, they start a cascade of biochemical processes resulting in disability of osteoclasts to resorb bone, or even in apoptosis. The precondition for easy invasion of microorganisms is the presence of teeth and the development of bony infections are caused by dental caries complications and periodontal disease (Petia Pechalova *et al*., 2011). Authors suggested that Panoramic radiographs, dental cone beam or spiral computed tomographies are the useful imaging techniques for the detection of BRONJ. When there is no clinical exposure of bone, scintigraphy, PET scan, or MRI are indicated for detection of early areas of bony involvement. Serum C-telopeptide crosslink oftype 1 collagen (CTX) marker is used for identifying the risk of BRONJ (Ilke Coskun Benlidayi and Rengin Guzel, 2013). Narongroeknawin *et al*. suggested that dental and periodontal disease, dental surgery, oral trauma, and poor oral hygiene are the oral predisposing factors and dose, duration & type of bisphosphonate therapy, concomitant therapies (chemotherapy, corticosteroids), alcohol use, smoking, advanced age, and other underlying medical conditions (diabetes mellitus or peripheral vascular disease) are the systemic predisposing factors of osteonecrosis of the jaw (Narongroeknawin *et al*., 2010). Studies have shown that whites are more prone to osteonecrosis of the jaw (ONJ) than blacks (Badros *et al*., 2006). Authors suggested that incidence rate of osteonecrosis of the jaw is higher in case of malignancy and chronic renal insufficiency (Yeo-Gab Kim *et al*., 2010). Studies have shown, Non – bisphosphonate drugs like Denosumab, Bevacizumab and Sunitinib are used as chemotherapeutic drugs and they could be the causative factors of osteonecrosis of jaw (Ramirez *et al*., 2015). Authors suggested that Single nucleotide polymorphisms in the cytochrome P450-2C gene and IGF 1 genes may play role in the pathogenesis of BRONJ (Sarasquete *et al*., 2008; Nicoletti *et al*., 2012). Authors suggested that the microorganisms which are most commonly seen in exposed bone in case of bisphosphonate - related osteonecrosis of the jaw are Actinomyces,Veillonella, Eikenella, Moraxella, Fusobacterium, Bacillus, Staphylococcus, Streptococcus, and Selenomonas.All of them are sensitive to penicillin. So, penicillin is the drug of choice for non-surgical treatment of BRONJ (Sedghizadeh *et al*., 2008; Sawatari and Marx, 2007; Woo *et al*., 2005).

Conclusion

We conclude that the occurrence of osteonecrosis of the jaw is multifactorial. Generally, mandible is more commonly affected than maxilla (2:1) in osteonecrosis of the jaw but in our case maxilla is affected. In our case, the presence of exposed maxillary jaw bone inside the mouth is the only clinical feature of jaw necrosis which leads to a diagnosis. After analysing every risk factor, appropriate treatment planning may avoid the development of osteonecrosis of the jaw.

Acknowledgements

The authors gratefully acknowledge the contribution made by Professor (Dr.) Prashant Nahar, Department of Oral Medicine & Radiology, Pacific Dental College & Hospital, Udaipur, Rajasthan, for his valuable guidance, support and help in every aspect.

REFERENCES

Amber Kiyani, Azhar Sheikh and Qandeel Musharaf, 2009. Bisphosphonate therapy related osteonecrosis of jaw bones : a case report. *Pakistan Oral & Dental Journal*, 29 (2):225-227.

Badros A, Weikel D, Salama A, Goloubeva O, Schneider A, Rapoport A. *et al*. 2006. Osteonecrosis of the jaw in multiple myeloma patients: clinical features and risk factors. *J Clin Oncol*., 24:945-52.

Ilke Coskun Benlidayi and Rengin Guzel, 2013. Oral Bisphosphonate Related Osteonecrosis of the Jaw: A Challenging Adverse Effect: Review Article. *ISRN Rheumatology*, 1-6.

Joel Epstein and Isaäc Van Der Waal. Oral Cancer : Burket's Oral Medicine : 11th edition: 177- 185.

Michael R. Markiewicz, B.S., Joseph E. Margarone, John H. Campbell and Alfredo Aguirre, 2005. Bisphosphonate associated osteonecrosis of the jaws : A review of current knowledge. *JADA*, 136 : 1669-1674.

Narongroeknawin *et al*. 2010. Bisphosphonate-associated osteonecrosis of the jaw, withhealing after teriparatide: a review of the literature and a case report. *Spec Care Dentist*., 30(2): 77–82.

Nicoletti, P., V. M. Cartsos, P. K. Palaska *et al*. 2012. "Genomewide pharmacogenetics of bisphosphonate-induced osteonecrosis of the jaw: the role of RBMS3", *Oncologist*, vol. 17, no. 2, pp. 279–287.

Petia Pechalova *et al*. 2011. Bisphosphonate-associated osteonecrosis of the jaws – report of three cases in bulgaria and review of the literature. *Acta Clin Croat*, 50 (2) : 273-279

Rajendran, R. and B. Sivapathasundharam. Shafer's Textbook of Oral Pathology : Seventh Edition : 2208 -2209.

Ramirez L, Lopez-Pintor RM, Casanas E, Arriba Ld and Hernandez G. 2015. New Non Bisphosphonate Drugs that Produce Osteonecrosis of the Jaws. *Oral Health Prev Dent*., 13(5):385-93.

Sarasquete, M. E., R. Garc´ıa-Sanz, L. Mar´ın *et al*., 2008. "Bisphosphonate-related osteonecrosis of the jaw is associated withpolymorphisms of the cytoehrome P450 CYP2C8 in multiple myeloma: a genome-wide single nucleotide polymorphism analysis", *Blood*, vol. 112, no. 7, pp. 2709–2712.

Sawatari Y. and Marx RE. 2007. Bisphosphonates and bisphosphonate induced osteonecrosis. *Oral Maxillofac Surg Clin North Am*., 19(4):487–98.

Sedghizadeh PP, Kumar SK, Gorur A, Schaudinn C, Shuler CF. and Costerton JW. 2008. Identifi cation of microbial biofilms in osteonecrosis of the jaws secondary to bisphosphonate therapy. *J Oral Maxillofac Surg*., 66(4): 767–75.

Woo SB, Hande K. and Richardson PG. 2005. Osteonecrosis of the jaw and bisphosphonates. *N Engl J Med*., 353(1):99–102.

Yeo-Gab Kim, Baek-Soo Lee1, Yong-Dae Kwon, Joon-Ho Suh and Sang-Mi Jeen, 2010. Study on bisphosphonate-related osteonecrosis of the jaw (BRONJ): case report and literature review. *J Korean Assoc Oral Maxillofac Surg*., 36:291-302.

****