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VARIOUS FORMS OF OSTEOSARCOMA OF JAWS WITH HISTOMORPHOLOGY AND IMMUNOHISTOCHEMISTRY: A CASE SERIES AND REVIEW

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ABSTRACT

Background: Osteosarcomas are highly malignant bone tumours characterised by the presence of malignant mesenchymal cells producing osteoid or immature bone. Osteosarcoma of the jaws (OSJ) is making up 6% to 9% of all cases of OS and 1% of all head and neck malignancies with mandibular predilection. OSJ tend to occur in third to fourth decade of life with slight male predilection. Although the exact cause of OS is still unknown, A strong association with the adolescent growth spurt and hormonal factors may play a role. Additional risk factors include radiation exposure, alkylating agents, chronic osteomyelitis, trauma, viral infection, metallic implants, fibrous dysplasia, Paget disease of bone. Defects in RB, p53 and chromosome 21q, INK4a, DNA helicase genes play an important role in the process. The diagnosis involves clinico-radiological and histological correlation. The aim of this study {Systematic review and case series} to see various forms of osteosarcoma affecting jaw bone with histomorphology and immunohistochemistry. **Method:** Total 6 cases were included in this study visited to GDC and H, Ahmedabad and case series with a systematic review was performed. Pubmed, Cochrane and Google Scholar were used as search engine. The literature research was done by using clearly defined terms and their links. Total 26 article selected and evaluated for this review. **Conclusion:** Osteosarcoma is an uncommon condition with unusual presentation and various histopathological patterns. The process of diagnosing Osteosarcoma was based on an integrated study of clinical, radiologic, histopathologic and immunohistochemistry findings. Though histological examination is gold standard, Immunohistochemical analysis plays an important role in establishing the histogenetic origin of many entities thus helps in accurate diagnosis and prompt treatment. So, clinician should have thorough knowledge of relationship between IHC and bone tumors.

KEYWORDS

Osteosarcoma, Immunohistochemistry, Malignant bone tumours

INTRODUCTION

Osteosarcoma (OS) is a rare malignant tumour of osseous origin which arises from primitive bone forming mesenchyme. It is commonest among all primary malignant bone tumour accounts 27.5% of all malignant tumours and 19.2% of all bone tumours. Most commonly it involves metaphysis of long bones of the extremities.[1,2] Osteosarcoma of the jaws (OSJ) is making up 6% to 9% of all cases of OS and 1% of all head and neck malignancies with mandibular predilection.[2-5] OSJ tend to occur in third to fourth decade of life with slight male predilection.[2,5,6] Although the exact cause of OS is still unknown, A strong association with the adolescent growth spurt and hormonal factors may play a role. Additional risk factors include radiation exposure, alkylating agents, chronic osteomyelitis, trauma, viral infection, metallic implants, fibrous dysplasia, Paget disease of bone.[2,5,7] Defects in RB, p53 and chromosome 21q, INK4a, DNA helicase genes play an important role in the process.[2,3,5]

The most common presenting symptoms of OSJs are swelling/mass and pain at the site of disease including numbness and facial dysesthesia/paraesthesia, teeth mobility, trismus, nasal obstruction (in maxillary tumors) and pathologic fracture. Rarely systemic symptoms of fever, asthenia or weight loss seen.[1-3,5] Radiographic features differ as per the stage of the lesion, may be entirely radiolucent, entirely radiopaque or mixed. Osteolytic stage shows moth-eaten appearance. Mixed stage shows a small amount of new bone, forming strands or giving honey comb appearance. Osteoblastic type lesion has granular- or sclerotic-appearing bone, cotton wisps of bone with adjacent destruction of the normal osseous architecture. Codman triangle and "sunray" appearance may be seen. Periodontal ligament space widening and lamina dura attenuation may occur.[2,6,7,8,9]

As per WHO classification of bone tumours in 2013[7] and updated classification in 2020[10], conventional OSs are histologically classified as osteoblastic, chondroblastic, fibroblastic and other less common variants. The tumor cells may vary from relatively uniform, round or spindle-shaped cells to highly pleomorphic cells with bizarre nuclear and cytoplasmic shapes. The amount of matrix produced by the tumor may vary considerably.[2]

Histological Classification of Osteosarcoma (WHO)[7]

DISCUSSION

Osteosarcoma is a malignant neoplasm of mesenchymal origin that can produce an immature osteoid or disorganized bone. OSJ accounts up to 1/10th of all OSs and arise later than their extragnathic counterparts, with a median age of 28 years. They occur approximately twice in men than in women and mandible affected more frequently than maxilla as seen in present study.[4,6,7]

OSJs clinically present with swelling, pain with facial dysesthesia. In present study, various forms of OSJs have clinical presentations as per literature.[5,10,11] Osteoblastic, Chondroblastic, Small cell, MFH-type and Epithelioid variants of OSJ showed aggressive clinical behaviour while Parosteal OSJ was less aggressive. Conventional osteosarcoma is a high-grade malignant tumor.[1] As reported by Rinaggio et al [6] and Naka et al [12], Epithelioid and MFH-type has more aggressive clinical behaviour compared to conventional types of OSJ. Huang et al [13] reported that Parosteal OSJ is non-aggressive. Small cell OSJ has similar clinical presentation to conventional OSJ.[14] Radiographically, present case series of OSJs showed radiolucent, radiopaque, or mixed appearance depending on behaviour of tumour as per literature.[4,6,7,8,10,12,15,16] Out of six cases, only one case [No. 1] showed osteolytic while five cases [No. 2,3,4,5 & 6] showed mixed appearance. Radiographically, Parosteal OSJ demonstrate a radiolucent line ("string sign") that corresponds to the periosteum between the sclerotic tumor and the underlying cortex, seen only in 30% of cases was absent in present case [No. 4][10,16] Biologic behavior of MFH-like OSJ is very aggressive and most cases showed purely lytic pattern similar to present case [No. 5] [12] Epithelioid OSJ [No. 6] was osteolytic similar to case reported by ALQahtani.[4]

Histological and Immunohistochemical Features of Various Types of OSJ

Osteoblastic Osteosarcoma

Among various histologic varients of OSJs, chondroblastic variant is predominant, followed by osteoblastic. Osteoblastic OS shows Predominant osteoid formation similar to present case [No.1]. Considering IHC features, Bone matrix proteins such as osteocalcin [OC], osteonectin and alkaline phosphatase expressed in OSs. OC is specific for osteoblastic cell lines and can be useful to confirm the diagnosis of OS. OC is consistently expressed in conventional OS, either in the extracellular matrix or cell cytoplasm. [17]

Chondroblastic Osteosarcoma

Chondroblastic osteosarcoma [COS] is defined as a histological entity characterized by predominant presence of chondroid matrix, which tends to exhibit a high degree of hyaline cartilage and is intimately associated with the non-chondroid element (osteoid or bone matrix). [18] Present case [No. 2] showed extensive cartilaginous matrix. Almeida et al [18] reported COS having irregular bone trabeculae amidst discretely atypical chondroblastic cell proliferation, hyperchromatic and pleomorphic fusiform cells at periphery of mineralised tissue. OS can be positive for S-100, particularly in areas of chondroid differentiation and there are reports of S-100 positivity not only in chondroblastic but also in osteoblastic and fibroblastic variant. COS will be positive for Vimentin, EMA, S100 and rarely Cytokeratin While Chondrosarcoma shows positivity for S100 and Vimentin while it is negative for cytokeratin and EMA. Galactin-1 was further seen to be largely expressed in osteosarcoma but not in chondrosarcoma. COS on the other hand is positive for Vimentin, EMA, S100 and rarely cytokeratin. [19]

Small Cell Osteosarcoma

Small cell OS constitutes between 1% and 2% of all osteosarcomas.[20] Histologically, it consists of sheets of round cells that produce an osteoid matrix as seen in present case [No. 3]. IHC of small cell tumours shows positivity for CD 99. IHC of present case [no. 3] shows CD99 positivity and vimentin negativity. Sethi et al [20] reported case of small cell OS in mandible showing round to ovoid tumours arranged in form of sheets or lobules and CD 99 positivity in IHC. Presence of round cells and CD99 positivity may be mistaken for Ewing sarcoma but the production of osteoid is not a characteristic feature of Ewing sarcoma. [14,20]

Parosteal Osteosarcoma

Geschicter and Copeland first described Parosteal (or juxtacortical) osteosarcoma (5% of all OS). As seen in present case [No. 4], It shows spindle cell fibroblast-like proliferation with well-developed, elongated bony trabeculae, often arranged in a parallel streaming or "pulled steel wool" pattern. [3,7,10,20] Huang et al [13] reported parosteal OS of maxilla having abnormally mineralized bony matrix, presenting as irregular, complex interlacing trabeculae of woven bone merged with the bony trabeculae of the adjacent bone with no well-defined capsule.

MFH -type Osteosarcoma

Mirra first used the term "MFH-like osteosarcoma" in 1980 and thereby gave a more detailed histologic criteria to this subtype.[12] In 2002, the World Health Organization declassified MFH as a formal diagnostic entity and renamed it as an undifferentiated pleomorphic sarcoma as no true cell of origin has ever been identified. [21-23] Malignant fibrous histiocytoma-like osteosarcoma shows spindle anaplastic cells and storiform-pleomorphic type areas with osteoid formation.[12,15] IHC of MFH type OS [No. 5] showed vimentin positivity and negativity for actin and desmin. It is important to differentiate MFH type OS from MFH because MFH also shows strong positivity for vimentin and other markers like histiocyte marker – CD68 and no reaction to the epithelial membrane antigen and cytokeratin. Tdashi et al[24] reported four cases of MFH like osteosarcoma which showed vimentin positivity and negativity for epithelial membrane antigen and cytokeratin. No overexpression of p53 protein was found in MFH-like osteosarcoma as in osteoblastic OS.[12]

Epithelioid Osteosarcoma

Osteosarcoma with epithelioid morphology was first reported by Scranton et al in 1975.the term epithelioid is used to denote a variant of various tumours formed predominantly by cells with epithelial like morphology. [4,25] Microscopically EOS consists of enlarged osteoblasts with epithelioid features that are organised in sheets, cords, trabeculae, glandular or even rossete structures with little osteoid. [20] Present case [No.6] Showed nests and acini of pleomorphic tumour cells. H. kaveri[26] reported a case of EOS affecting the maxilla showed pleomorphic, round to ovoid tumor cells with hyperchromatic and vesicular nuclei, focal chondroblastic areas and proliferation of pleomorphic spindle cells. EOS [No. 6] showed vimentin and SATB2 positivity and negativity for cytokeratin 7, EMA, OCT ¾, PLAP and S-100. In case report of Rinaggio et al [6], IHC studies showed a strong diffuse positivity for vimentin, whereas cytokeratin, epithelial membrane protein (EMA) and S-100 were negative in EOS. SATB2 is very sensitive marker [90%] for OS expressed independently of malignant osteoid quantity as reported by Machado et al. [27]

Further detection of MDM2 and CDK4 is a useful diagnostic tool in separating low grade osteosarcoma from benign lesion. The expression of IDH1 in formalin fixed paraffin tissue correlated with p53 and thus may serve as a candidate biomarker for OSs. [3,25]

Because of the many histological variants of osteosarcoma, a wide range of tumor types needs to be considered in its differential diagnosis.[4] angiosarcoma, chondrosarcoma, fibrosarcoma, leiomyosarcoma, synovial sarcoma, epithelioid sarcoma and malignant peripheral nerve sheath tumor. In children, rhabdomyosarcoma, rhabdoid tumor, angiosarcoma and malignant peripheral nerve sheath tumor should be considered in the differential diagnosis. [9,10,11]

When histological features are causing diagnostic dilema, correlation of histological and immunohistochemical findings are important to give diagnosis of OS. Thus, the IHC profile and morphological characteristics found in OSJs are quite variable and Focal expression of a variety of markers found occasionally in otherwise typical osteosarcomas. This variability in marker expression might be explained by the multipotentiality of the mesenchymal cells in OSs.[4]

The treatment and management of osteosarcoma consists of a combination of surgery, chemotherapy and radiotherapy. Clean surgical margins are associated with a considerably higher 5year survival rate and lower risk of local recurrence. [2,5,6,11,28,26] JOS has better prognosis compared with long-bone osteosarcomas due to lesser incidence of distant metastasis and histologically better differentiation of jaw osteosarcomas than that of long bone osteosarcomas. Many factors affect the prognosis of osteosarcoma. The most studied are histological subtype, grade, tumor size, patient age and response to chemotherapy and positive resection margins Complete resection of tumors involving the maxilla can be technically challenging, so local recurrence is more frequent in maxillary than mandibular osteosarcomas[2,3,5,8,15] In present case series, Osteoblastic, Chondroblastic and Parosteal types showed good prognosis while small cell, MFH-type and Epithelioid variant showed poor prognosis. As per literature Osteoblastic, chondroblastic, fibroblastic types do not have any great bearing on the prognosis. Small cell OS has poor prognosis than conventional types as reported by Sethi et al. [14]. Parosteal osteosarcomas are low-grade tumors, with a low risk for recurrence and metastasis following radical excision. [10] EOS has been shown to have aggressive behavior and a poor prognosis [4,6]

CONCLUSION

Osteosarcoma is an uncommon condition with unusual presentation and various histopathological patterns. The process of diagnosing Osteosarcoma was based on an integrated study of clinical, radiologic, histopathologic and immunohistochemistry findings. Though histological examination is gold standard, Immunohistochemical analysis plays an important role in establishing the histogenetic origin of many entities thus helps in accurate diagnosis and prompt treatment. So, clinician should have thorough knowledge of relationship between IHC and bone tumors.

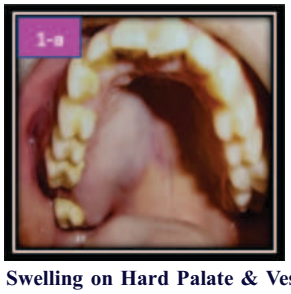

Figure 1a: Firm Swelling on Hard Palate & Vestibule on Right Side

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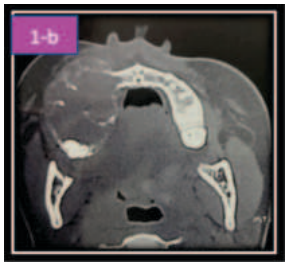

Figure 1b: CBCT Showes Mixed Radiolucent-radioopaque Lesion with Thin Septa and Erosion of Maxilla on Right Side

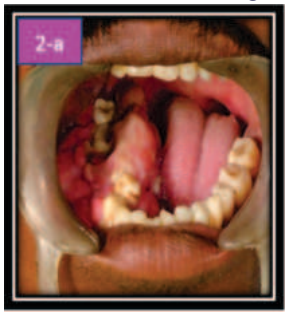

Figure 2a: Firm Swelling on Alveolar Ridge and Vestibule with Superficial Ulceration, Lingually Displaced Teeth i.r.t. 45,47.

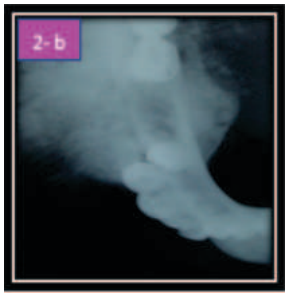

Figure 2b: Occlusal (Cross Sectional) Radiograph Shows Mixed Radiolucent-radioopaque Lesion with Sunray Appearance, Expansion of Buccal and Lingual Cortical Plates

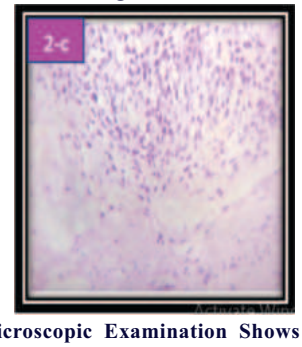

Figure 2c: Microscopic Examination Shows Spindle Cells, Atypical Osteoblasts with Cartilaginous Matrix in Chondroblastic Osteosarcoma (x10, H & E)

Figure 3a: Firm Swelling on Middle and Lower Third of Face on Right Side

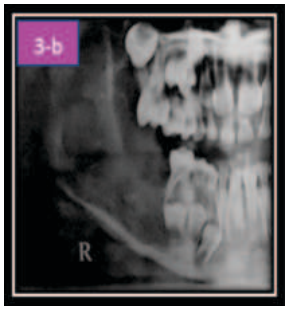

Figure 3b: OPG Shows Mixed Radiolucent-radioopaque Lesion Involving Entire Ramus with Granular and Sunray Appearance From Anterior Border of Ramus

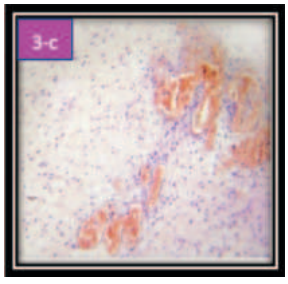

Figure 3c: Microscopic Examination Shows Spindle and Round Cells with Osteoid in Small Cell Osteosarcoma (x10, H & E)

Figure 4a: Firm Growth on Alveolar Ridge and Vestibule on Lower Left Side with Superficial Ulceration

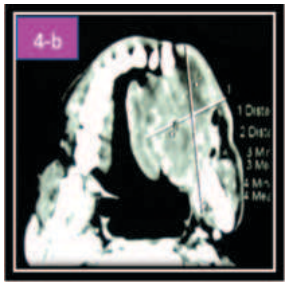

Figure 4b: CT Shows Mixed Radiolucent-radioopaque Lesion with Erosion of Buccal and Lingual Cortical Plates

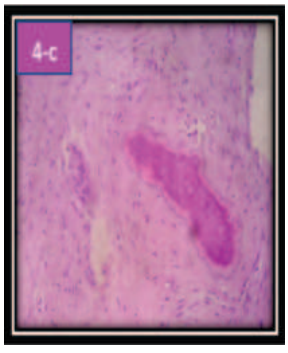

Figure 4c: Microscopic Examination Shows Well Differentiated Fibroblastic Cells with Osteoid in Parosteal Osteosarcoma (x10, H & E)

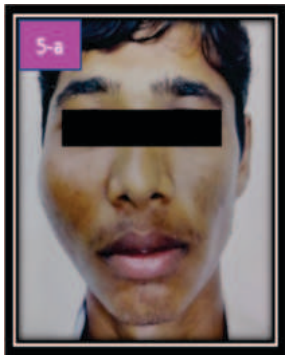

Figure 5a: Hard Swelling on Midface Region on Right Side

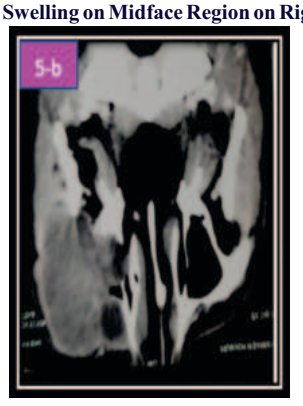

Figure 5b: CT Shows Mixed Radiolucent-radioopaque Lesion with Erosion of Maxilla, all Walls of Maxillary Sinus

Figure 6a: Firm Growth on Alveolar Ridge, Buccal and Lingual Vestibule & Floor of Mouth With Brown Necrotic Tissue on Overlying Mucosa

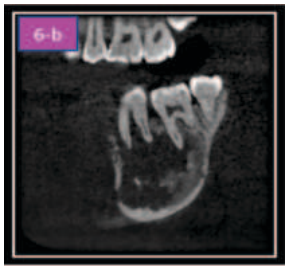

Figure 6b: CBCT Shows Mixed Radiolucent-radioopaque Lesion Having Areas of Radiopacity with Erosion and Perforation Cortex, Floating Teeth i.r.t 36,37.

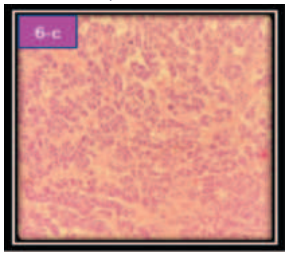

Figure 6c: Microscopic Examination Exhibiting Nests and Acini of Pleomorphic Tumour Cells with Eosinophilic Cytoplasm and Prominent Nucleoli in Epithelioid Osteosarcoma (x10, H & E)

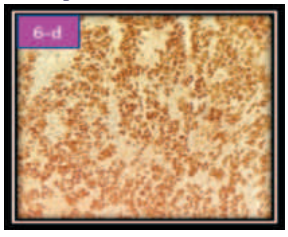

Figure 6d: IHC Shows Vimentin Positivity in Epithelioid Osteosarcoma (x10)

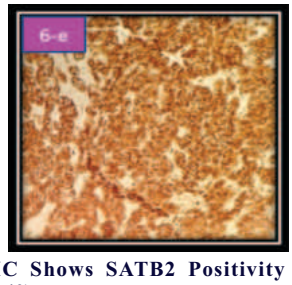

Figure 6e: IHC Shows SATB2 Positivity in Epithelioid Osteosarcoma (x40)

Case series of OSJ

Case No.	Age/ Sex	Anatomic Location	Clinical Findings & behaviour	Radiographic Findings	Histologic Findings	IHC Findings	Final Diagnosis	Treatment	Follow Up (mo.)
1	15/F	R Maxilla	- Ill-defined firm swelling of 5x3 cm in hard palate & vestibule, mobility of teeth i.r.t. 14,15,16,18. [Fig. 1-a] - Rapidly growing (15 days)	Mixed radiolucent-radioopaque lesion with thin septa, erosion of maxilla and all walls of maxillary sinus, loss of lamina dura & floating teeth i.r.t 14,15,16,18. [Fig. 1-b]	Atypical osteoblast with abundant extracellular osteoid	NP	Osteoblastic OS	Maxillectomy	12, NED
2	25/M	R mandible	- Ill-defined firm swelling of 5x4 cm in alveolar ridge and vestibule with superficial ulceration, mobility of teeth i.r.t. 44,45,47,48, lingually displaced teeth i.r.t. 45,47. [Fig. 2-a] - Rapidly growing (3 months)	Mixed radiolucent-radioopaque lesion showing sunray appearance, Thinning and expansion of buccal and lingual cortical plates, loss of lamina dura i.r.t 44,45,47. [Fig. 2-b]	Spindle cells and atypical osteoblasts with extensive cartilaginous matrix. [Fig. 2-c]	NP	Chondroblastic OS	Hemimandibulectomy with SOHND	6, NED
3	8/F	R mandible	- Ill-defined firm swelling of 4x3 cm in alveolar ridge and vestibule with superficial ulceration, mobility of teeth i.r.t. 85. [Fig. 3-a] - Rapidly growing (2 months)	Mixed radiolucent-radioopaque lesion showing granular and sunray appearance, erosion of body, angle and ramus of mandible, loss of lamina dura & floating teeth i.r.t 85. [Fig. 3-b]	Malignant spindle and round cells with osteoid. [Fig. 3-c]	P- CD 99 N- Vimentin	Small cell OS	Palliative care	1, DOD
4	35/M	L mandible	-Ill-defined firm growth of 6x4 cm in alveolar ridge and vestibule with superficial ulceration, mobility of teeth i.r.t. 23,33,34, unable to close mouth due to overgrowth. [Fig. 4-a] - Slowly growing (9 months)	Mixed radiolucent-radioopaque lesion with pathologic fracture of body, erosion of the upper and lower alveolus, posterior wall of right maxillary sinus, loss of lamina dura i.r.t 23,33,34. [Fig. 4-b]	Well differentiated fibroblastic cells with osteoid. [Fig. 4-c]	NP	Parosteal OS	hemimandibulectomy	18, NED
5	20/M	R Maxilla	- Ill-defined hard swelling of 4x2 cm in vestibule, no teeth mobility & displacement. [Fig. 5-a] - Rapidly growing (2 months)	Mixed radiolucent-radioopaque lesion with erosion of maxilla, all walls of maxillary sinus, lateral wall of orbit and zygomatic arch. [Fig. 5-b]	Spindle anaplastic cells with osteoid	P- Vimentin N- Actin, Desmin	MFH type OS	Maxillectomy	3, DOD

6	16M	L mandible	- Ill-defined firm growth of 7x5 cm in alveolar ridge, buccal and lingual vestibule & floor of mouth, yellow, red, black colours at different areas with brown necrotic tissue on overlying mucosa, mobility of teeth not accessible & unable to close mouth due to overgrowth. [Fig. 6-a] - Rapidly growing (25 days)	Mixed radiolucent-radioopaque lesion having areas of radiopacity with erosion and perforation of buccal and lingual cortex, loss of lamina dura & floating teeth i.r.t 33,34,36,37. [Fig. 6-b]	Nests and acini of pleomorphic tumour cells with eosinophilic cytoplasm and prominent nucleoli. [Fig. 6-c]	P- Vimentin SATB2 [Fig. 6-d & 6-e] N- EMA, CK7 PLAP, S 100 OCT3/4	Epithelioid OS	Palliative care	4, DOD

F- Female, M- Male, R- Right, L- Left, NP- Not Performed, P-Positive, N-Negative, Mo.- Months, NED- No Evidence of Disease, DOD- Died Of Disease

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