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Editorial

The Blended Benevolence-Osteochondromyxoma

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Osteochondromyxoma manifests as an exceptionally discerned, congenital bone neoplasm. Neoplasm may infrequently concur as a diagnostic component of Carney complex constituted of familial lentinginous or multi-organ tumour syndrome.

Generally, the painless osteochondromyxoma is confined to facial bones, nasal sinuses, nasal bones, skull, sellar region, ribs, diaphysis of long bones as the tibia or radius, thoracic region and vertebral column. Tumefaction appears concurrent with inactivating mutations of tumour suppressor gene PRKAR1A situated upon chromosome 17q22-24. Osteochondromyxoma is posited to arise from deranged mesenchymal stem cells which presumably engender osteoblasts [1,2].

Pertinent genetic mutations are concordant to hyper-stimulation of protein kinase A (PKA) associated with elevated cellular levels of cyclic AMP (cAMP) [1,2].

Tumefaction typically represents as a painless nodule. Alternatively, tumefaction may be discovered incidentally secondary to skeletal survey employed for determining the occurrence of Carney complex [1,2].

Osteochondromyxoma represents with clinical symptoms contingent to tumour magnitude and location which occur as swelling, discomfort and localized inflammation of soft tissues. Besides, non specific clinical symptoms may appear. Additionally, osteochondromyxoma may occur as an isolated disease entity [2,3].

Grossly, neoplasm displays an amalgamation of a gelatinous, cartilaginous and bony tissue aggregates [2,3].

Upon microscopy, neoplasm is minimally, moderately or variably cellular. Tumefaction is comprised of sheets and lobules of bland mesenchymal cells embedded within an abundant myxomatous, cartilaginous, partially osseous tissue and hyaline fibrous matrix. Tumefaction may erode circumscribing bone and frequently extends into adjacent soft tissue.

Tumour configures lobular sheets constituted of chondroid tissue, osteoid and a polymorphic cellular component admixed with hyaline bands. The benign, locally aggressive neoplasm appears to lack fragments of bluish, immature bony trabeculae [3,4].

Tumefaction is composed of an admixture of mesenchymal cells, basophilic myxoid foci and mucopolysaccharide ground substance. Tumour cellularity is contingent to quantifiable myxoid matrix. Osteoid, bone, collagen fibres, immature and mature cartilage or bands and nodules of hyaline fibrous tissue configure the neoplasm [3,4].

Tumour cells appear organized and configure well defined sheets or occasional poorly defined cellular whorls. Alternatively, neoplasm may be disorganized and demonstrate micro-lobular or macro-lobular tumour pattern [3,4].

Osteochondromyxoma cells are designated as chondroblast-like and osteoblast-like cells. Tumour is confined within a capsule composed of parallel, 10 cells to 15 cells thick layers of spindle shaped mesenchymal cells [3,4].

Tumour cells are imbued with eosinophilic cytoplasm and display columns of nuclei. Non encapsulated neoplasms lack distinc-

tive demarcation and appear to commingle with circumscribing soft tissue. Inflammation of surrounding soft tissue is exceptional.

Mitotic figures are occasional. Nuclear atypia is infrequently discerned [3,4].

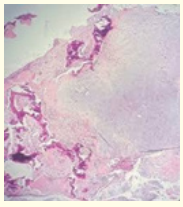

Figure 1: Osteochondromyxoma demonstrating a lobular pattern composed of osteoblast-like and chondroblast-like mesenchymal cells commingled within a myxoid matrix [6].

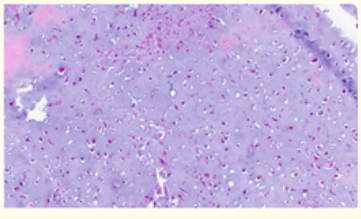

Figure 2: Osteochondromyxoma delineating mesenchymal cells imbued with eosinophilic cytoplasm and columns of nuclei commingled with a myxomatous matrix [7].

Tumour cell cytoplasm may be appropriately stained by periodic acid-Schiff's (PAS) stain wherein staining of matrix may be minimal. Also, cellular cytoplasm and matrix may be stained by colloidal iron.

Movat's pentachrome stain may be beneficially employed in order to segregate tumour components as myxomatous (faint blue), cartilaginous (blue, green or olive), hyaline (yellow) and osseous (yellow, scarlet or brick red) [3,4].

Tumour cells appear immune reactive to vimentin and occasionally to S-100 protein. Immune reactivity to collagen II is focal and moderate whereas immune reactivity to collagen IV is minimal [3,4].

Osteochondromyxoma requires segregation from neoplasms as chondromyxoid fibroma, myxoid chondrosarcoma, chondromesenchymal hamartoma or fibrous dysplasia [4,5].

Upon plain radiography, osteochondromyxoma demonstrates a well circumscribed, destructive neoplasm with focal mineralization and diverse countenance contingent to tumour location. Tumefaction delineates expansion of bony cortex with admixed lucent and sclerotic zones [4,5].

Plain radiography appears beneficial for assessment of parameters as tumour configuration, localization, tumour perimeter, transition zone and breach within superimposed bone cortex [4,5].

Upon imaging, osteochondromyxoma requires demarcation from neoplasms such as chondromyxoid fibroma, mesenchymal hamartoma, myxoma, chondrosarcoma with myxoid change and fibrocartilaginous mesenchymoma [4,5].

Computerized tomography (CT) demonstrates calcified density of soft tissue commingled with an osteoid matrix. Erosion of superimposed bony cortex may be occasionally discerned [4,5].

Magnetic resonance imaging (MRI) is a precise methodology which may be advantageously adopted for assessing symptomatic bone tumours. T1 weighted magnetic resonance imaging (MRI) displays a heterogeneous, mixed-signal intensity which is isointense or hypo-intense to adjoining skeletal muscle [4,5].

Upon T2 weighted magnetic resonance imaging, a heterogeneous tumefaction with predominantly enhanced signal intensity is observed.

Upon administration of gadolinium contrast, a heterogeneous signal image enhancement is exemplified [4,5].

Osteochondromyxoma may be aptly subjected to comprehensive surgical resection. Localized tumour reoccurrence ensues secondary to incomplete tumour excision [4,5].

Comprehensive surgical extermination of the neoplasm ensures appropriate alleviation and superior prognostic outcomes. As localized tumour reoccurrence is frequently discerned following inadequate surgical resection, neoplastic relapse is common within sites where comprehensive tumour resection is challenging to attain. Distant tumour metastasis remains undocumented [4,5].

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6. Image 1 Courtesy: Cureus.com.

7. Image 2 Courtesy: Science direct.