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Mutable and Slimy-Myxoid Pleomorphic Liposarcoma

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Editorial

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Abbreviations: MRI: Magnetic Resonance Imaging; ACGH: Array Comparative Genomic Hybridization.

Editorial

Myxoid pleomorphic liposarcoma is an exceptionally discerned, aggressive variant of liposarcoma predominantly incriminating young individuals. The preponderantly hybrid tumefaction demonstrates an amalgamation of morphological features of myxoid liposarcoma and pleomorphic liposarcoma. Additionally designated as pleomorphic myxoid liposarcoma, myxoid pleomorphic liposarcoma is a soft tissue tumour delineating an aggressive clinical course and inferior overall survival. Commonly, tumefaction is devoid of FUS/EWSR1-DDIT3 genetic fusions or MDM2 genomic amplification. Commonly, myxoid pleomorphic liposarcoma incriminates young subjects < 30 years. However, few lesions may occur within older adults. A specific gender predilection is absent. Nevertheless, a mild female preponderance or an equivalent gender predisposition may be encountered [1,2].

Myxoid pleomorphic liposarcoma predominantly arises within the mediastinum. Infrequently, neoplasm may emerge within head and neck, trunk or extremities [1,2]. Myxoid pleomorphic liposarcoma exhibits genomic losses within chromosome 13, especially the RB1 loci, a pathognomonic feature encountered within tumorigenesis of myxoid pleomorphic liposarcoma [1,2]. Myxoid pleomorphic liposarcoma manifests as a sarcoma demonstrating complex genomics with characteristic structural chromosomal aberrations in association with mono-allelic deletion of RB1 gene. Adoption of array comparative genomic hybridization (aCGH) frequently delineates a distinctive genomic profile with whole chromosome gains within chromosomes 1, 6, 7, 8, 19, 20, 21 and X. Besides, losses within chromosomes 2, 3,4, 5, 10, 11, 12,13, 14, 15, 16, 17 and 22 are encountered [1,2]. Methylation profiling exhibits an overlapping genetic profile between myxoid pleomorphic liposarcoma and classic pleomorphic liposarcoma, thus indicating a close concurrence within the dual neoplasms. Besides, KMT2D genetic mutations may occur. Also, FUS/EWSR1-DDIT3 genomic fusions or MDM2 genetic amplification is absent [1,2]. Frequently, an enlarged neoplasm is discerned upon initial disease representation, possibly on account of tumour localization within the mediastinum and consequently delayed emergence of clinical symptoms. Generally, a rapidly enlarging tumour mass or pain may manifest as a preliminary symptom [1,2]. Myxoid pleomorphic liposarcoma may be associated with Li-Fraumeni syndrome [2,3]. Upon gross examination, neoplasm appears as an enlarged, grossly infiltrative lesion. Alternatively, a nodular tumefaction may be encountered. Cut surface is variably myxoid [2,3]. Upon microscopy, majority of instances demonstrate a variable admixture of myxoid liposarcoma an pleomorphic liposarcoma-like a Myxoid liposarcoma-like areas are minimally to moderately cellular and comprised of spindle shaped cells permeated with inconspicuous cytoplasm and spherical to elliptical, hyperchromatic nuclei. Circumscribing stroma is myxoid and manifests abundant, enmeshed, ramified capillaries and vascular articulations [2,3]. Although minimal, nuclear atypia and nuclear pleomorphism appears in excess of, as represented in classic myxoid liposarcoma. Pools of myxoid substance are frequently encountered [2,3]. Pleomorphic liposarcoma-like areas are extensively cellular and preponderantly constituted of pleomorphic cells pervaded with irregular, hyperchromatic nuclei. Foci of adipocytic differentiation are variable and lipoblasts are frequently discerned. Mitotic activity is significantly elevated and tumour necrosis is common [2,3].

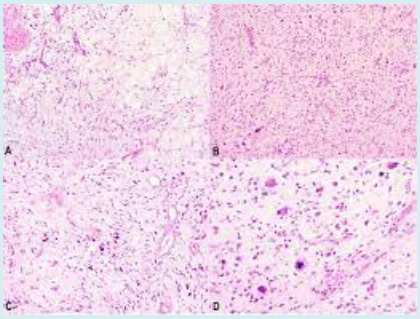

Figure 1: Myxoid pleomorphic liposarcoma delineating aggregates of spindle shaped cells permeated with inconspicuous cytoplasm and ovoid hyperchromatic nuclei. Surrounding pool of myxoid substance is imbued with abundant, ramified capillaries [6].

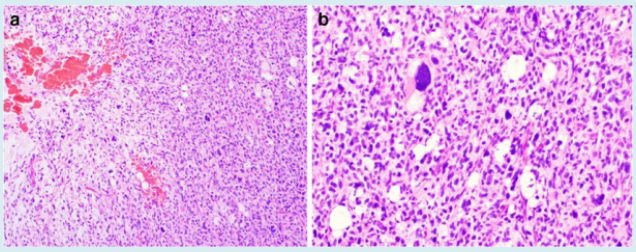

Figure 2: Myxoid pleomorphic liposarcoma demonstrating clusters of spindle shaped cells incorporated with inconspicuous cytoplasm and oval, hyperchromatic nuclei. Circumscribing pools of myxoid substance are pervaded with focal haemorrhage and abundant, branching capillaries [7].

Myxoid pleomorphic liposarcoma is immune reactive to CD34, p16 or p53. Myxoid pleomorphic liposarcoma is immune non reactive to MDM2 and CDK4. Majority of instances depict loss of expression of Rb gene. Myxoid pleomorphic liposarcoma requires segregation from neoplasms such as myxoid liposarcoma, dedifferentiated liposarcoma, pleomorphic liposarcoma or atypical spindle cell/ pleomorphic lipomatous tumour.

Upon imaging, diagnostic features are insufficiently characterized. Cogent disease discernment is contingent to evaluation of pertinent histological features and molecular assessment [4,5]. Magnetic resonance imaging (MRI) exhibits an enlarged, deep seated tumefaction demonstrating minimal signal intensity upon T1 weighted imaging and an enhanced signal intensity upon T2 weighted imaging, thereby indicating the presence of a myxoid component [4,5]. Myxoid pleomorphic liposarcoma can be appropriately alleviated with comprehensive surgical eradication. A tumour free surgical perimeter is necessitated and appears potentially curative. Specific, applicable target therapies appear currently unavailable. Cogent adoption of adjuvant chemotherapy or radiotherapy remains unestablished [4,5]. Tumefaction is exceptionally associated with Li-Fraumeni syndrome, a feature which warrants prudence during employment of radiotherapy. Myxoid pleomorphic liposarcoma is a soft tissue tumefaction accompanied by an aggressive biological course and inferior overall survival. Enhanced proportionate localized reoccurrence may occur within 50% instances and metastatic neoplastic dissemination may be discerned comprehensively within 40% to 100% tumours [4,5].

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6. Image 1 Courtesy: Nature.com

7. Image 2 Courtesy: Research gate