

## Mesenchymal Translocation- Haemosiderotic Fibrolipomatous Tumour

Anubha Bajaj\*

Histopathologist in A.B. Diagnostics, New Delhi, India

### ABSTRACT

Haemosiderotic fibrohistiocytic lipomatous tumour was initially described by Marshall-Taylor and Fanburg-Smith in 2000 and is cogitated as an exceptional, biologically benign tumour emerging as a circumscribed lesion comprised of an adipose tissue and spindle-shaped cellular component with abundant haemosiderin. Haemosiderotic fibrolipomatous tumour is also designated as haemosiderotic fibrohistiocytic lipomatous tumour and is contemplated as an equivalent to a preliminary lesion of pleomorphic hyalinising angiectatic tumour.

Keywords: Haemosiderotic fibrohistiocytic; Antioxidants; Silver nitrate; Genetic; chromosomal

### DISEASE CHARACTERISTICS

Haemosiderotic fibrolipomatous tumour denominates a slight female preponderance. Majority instances appear within the distal extremities, especially the dorsal surface, foot or ankle. An estimated 87% of instances appear on foot and ankle, preceding trauma can be discerned in around 43% subjects whereas tumour reoccurrence or residual tumour is detected in nearly 41% individuals [1, 2].

The exceptional tumefaction delineates an incidence of below <0.2% of benign lipomatous neoplasia. Age of tumour incrimination varies from 8 months to 74 years whereas mean age of disease emergence is 51 years. The neoplasm is non-destructive, can reoccur although distant metastasis are absent. Cogent tumour localization is associated with unique histological features and characteristic disease attributes as discerned on magnetic resonance imaging (MRI) [3].

A clonal, reciprocal, chromosomal translocation betwixt chromosome 1 and 10, in association with additional genomic rearrangements, amidst chromosome 1 and 3 is observed. Typically, chromosomal translocation is enunciated within haemosiderotic fibrolipomatous tumour. Preponderant fibrohistiocytic lesions can be localized singularly within the dermis, concurrently within dermis and subcutaneous or purely within the subcutaneous tissue. Additionally, fibroblastic component

can be reactive, as cogitated with dermatofibromas, or neoplastic. However, on account of aforesaid chromosomal translocations, a neoplastic instead a reactive origin of the tumefaction is hypothesized [4].

### CLINICAL ELUCIDATION

Commonly, a gradually progressive, mildly atrophic, bluish-green plaque with moderate superimposed telangiectasia of variable magnitude is situated upon dorsal extremities, foot or ankle and arises preponderantly within middle aged adults.

Haemosiderotic fibrolipomatous tumour can be associated with venous stasis and anatomic distortion of incriminated organs. Preceding, localised soft tissue trauma may or may not be implicated in the aetiology [5]. On examination, the tumour can simulate a haematoma of extended duration or angiomatoid fibrous histiocytoma. The clinical course is benign although the neoplasm can be locally aggressive [6]. Venous stasis can incur an exaggerated tissue reaction with proliferation of spindle-shaped, fibroblastic cells, red cell extravasation and haemosiderin aggregation within the adipose tissue. The neoplasm can be concurrent to or represent a precursor lesion of pleomorphic hyalinising angiectatic tumour of soft tissue, on account of discernible vascular hyalinization within the tumour [7]. The tumour denominates a characteristic proliferation of fibroblast-like, spindle-shaped cells and adipocytes accompanied by

**Correspondence to:** Anubha Bajaj, Histopathologist in A.B. Diagnostics, New Delhi, India, Tel: 2458923146; E-mail: anubha.bajaj@gmail.com

**Received:** February 04, 2021; **Accepted:** February 18, 2021; **Published:** February 25, 2021

**Citation:** Bajaj A (2021) Mesenchymal Translocation- Haemosiderotic Fibrolipomatous Tumour. J Stem Cell Res Ther. 11:474.

**Copyright:** © 2021 Bajaj A. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

deposition of haemosiderin. Spindle-shaped cellular component demonstrates a potential for malignant metamorphoses, a feature which is absent within the adipose tissue. An aggressive sarcoma or a myxo-inflammatory fibroblastic sarcoma can thus be engendered. Mature, well-differentiated adipocytes are a prominent component of haemosiderotic fibrolipomatous tumour although appear as an innocent bystander. Cogent clinical information and immune histochemical evaluation can aid neoplastic categorization. Appropriate assessment of architectural configuration within an enlarged tissue specimen is necessitated for adequate classification [8].

#### HISTOLOGICAL ELUCIDATION

Characteristically, haemosiderotic fibrolipomatous tumour delineates well-defined tissue components discerned as adipose tissue and spindle-shaped cells. Spindle-shaped cells depict a dissecting pattern amidst lobules of adipocytes and along fibrotic septa. Foci of minimal inflammatory infiltrate are observed. Abundant quantities of intracytoplasmic hemosiderin pigment are discerned within spindle-shaped cells and macrophages circumscribing the vasculature. Hemosiderin pigment can be suitably stained by Prussian-blue iron stain. Miniature blood vessels are hyalinised. Immune reactive smooth muscle actin (SMA) highlights vascular prominence although spindle-shaped cells are non-reactive.

#### IMMUNE HISTOCHEMICAL EVALUATION

Haemosiderotic fibrolipomatous tumour is immune reactive to Calponin and immune non-reactive to CD68, smooth muscle actin(SMA), muscle specific actin (MSA), desmin, S100 protein, human melanoma black antigen 45(HMB45), keratin and epithelial membrane antigen(EMA).

Upon molecular genetic analysis, haemosiderotic fibrolipomatous tumour and myxoinflammatory fibroblastic sarcoma demonstrate an identical chromosomal translocation along with genomic fusion of TGFBR3-MGEA5 genes. Currently, genomic rearrangements of TGFBR3 and /or MGEA5 are denominated within pleomorphic hyalinising angiectatic tumour, thereby delineating histological features concurrent with haemosiderotic fibrolipomatous tumour [9].

#### DIFFERENTIAL DIAGNOSIS

Haemosiderotic fibrolipomatous tumour requires segregation from several cutaneous and subcutaneous pigmented lesions of fibrohistiocytic derivation such as

- Pigmented aneurysmal dermatofibroma appearing as a nodular tumour with well-circumscribed, storiform pattern
- Pigmented variant of dermatofibrosarcoma protuberans, also denominated as Bednar tumour, demonstrating a prominent cartwheel pattern and melanin pigment
- Kaposi's sarcoma, especially the plaque stage, depicting red cell extravasation, intracytoplasmic hyaline globules reactive to periodic acid Schiff's stain and a few cells demonstrating nuclear immune reactivity to human herpes virus 8 (HHV-8) antigens.
- Plexiform fibrous histiocytoma delineating a multinodular tumefaction with storiform configuration, predominant

multinucleated giant cells and scant amount of haemosiderin. Haemosiderotic fibrolipomatous tumour necessitates a demarcation from various neoplasia delineating a combination of bland, spindle-shaped cells and adipose tissue such as dermatofibrosarcoma protuberans, fibromatosis, atypical lipomatous tumour, spindle cell lipoma or a concurrence of spindle-shaped cells, histiocytes and multinucleated cells as discerned in plexiform fibrohistiocytic tumour. Subcutaneous lesions of haemosiderotic fibrolipomatous tumour, as discerned with miniature tissue samples, require a distinction from dermatofibrosarcoma protuberans, plexiform fibro-histiocytic tumour, spindle cell lipoma or well differentiated liposarcoma [10].

#### PROGNOSTIC OUTCOMES

Extended follow up demonstrates frequent, delayed, localized tumour reoccurrences in around 50% subjects; however occurrence of distant metastasis is absent. Extensive monitoring of the neoplasm can be done with computerized tomography (CT) and magnetic resonance imaging (MRI). Follow up fails to demonstrate a significant progression of the neoplasm or metastatic dissemination. Extensive follow up of the neoplasm is recommended in instances unamenable to comprehensive surgical extermination. Irrespective of superior immune histochemical techniques and molecular genetic assay, a competent histological elucidation, a tumour architectural pattern along with a cogent clinical representation is mandatory for definitive discernment of haemosiderotic fibrolipomatous tumour.

#### REFERENCES

1. Wu F, Zhao S, Yu B, Chen YM, Wang W, Song Z, et al. A new coronavirus associated with human respiratory disease in China. *Nat.* 2020;579(7798):265-269.
2. Zhang C, Zheng W, Huang X, Bell EW, Zhou X, Zhang Y. Protein Structure and Sequence Reanalysis of 2019-nCoV genome refutes snakes as its intermediate host and the unique similarity between its spike protein insertions and HIV1. *J Proteome Res.* 2020;19(4):1351-1360.
3. Andersen KG, Rambaut A, Lipkin WI, Holmes EC, Garry RF. The proximal origin of SARS-CoV-2. *Nat Med.* 2020;26(8):450-455.
4. Bojkova D, McCreig JE, McLaughlin KM, Masterson SG, Widera M, Kraehling V, et al. SARS-CoV-2 and SARS-CoV differ in their cell tropism and drug sensitivity profiles. *BioRxiv.* 2020.
5. Zhang XW, Yap YL. Yap. Structural similarity between HIV-1 gp41 and SARS-Cov S2 proteins suggests an analogous membrane fusion mechanism. *J Molec Struct.* 2004;677(66):73-76.
6. George A. Cytokine storm, an overreaction of the body's immune system *New Scientist.* 2020.
7. Caly L, Druce JD, Catton MG, Jans DA, Wagstaff KM. The FDA-approved drug ivermectin inhibits the replication of SARS-CoV-2 in vitro. *Antiviral Res.* 2020;178(6):104787.
8. Omura S, Crump A. The life and times of ivermectin success story. *Trop Med Infect Dis.* 2004;2(5):984-989.
9. Sajid MS, Iqbal Z, Muhammad G, Iqbal MU. Immunomodulatory effect of various anti-parasitics: a review. *Parasitol.* 2006;132(3):301-313.
10. Wassermann S, Stumpf M, Schmidt K, Schieffer B, Ohm BB, Nickenig G. Interleukin-6 induces oxidative stress and endothelial

dysfunction by overexpression of the angiotensin 2 type 1 receptor.  
Circ Res. 2004;5(5):534-541.