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Short Communication

The Sinewy Assumption-Gardner-Type Fibroma

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Gardner-type fibroma emerges as an exceptionally encountered, benign soft tissue lesion demonstrating morphological features akin to nuchal-type fibroma. Majority (~90%) of lesions are concurrent with familial adenomatous polyposis (FAP) or Gardner's syndrome delineating specific germline mutations within the APC gene.

Tumefaction is comprised of haphazardly disseminated thickened bands of collagen commingled with a population of bland fibroblasts wherein the cellular component appears to entrap adjacent soft tissue.

Neoplasm commonly occurs within infants, paediatric population or adolescents. An estimated 45% subjects may demonstrate desmoid-type fibromatosis.

Gardner-type fibroma may be confined to superficial or deep seated soft tissue. No site of disease emergence is exempt [1,2].

Gardner-type fibroma may preliminarily indicate the occurrence of Gardner's syndrome with concurrent genetic mutations within the APC gene. Chromosomal mutation confined to chromosome 5q21 is exemplified [2,3].

Although Gardner's syndrome is comprised of colonic polyps, osteomas and anomalies within the retinal epithelium, cutaneous manifestations commonly emerge as epidermoid cysts layered by stratified squamous epithelium [2,3].

Grossly, neoplasm appears as an inadequately circumscribed, firm, rubbery, plaque-like, white to tan or pink lesion of magnitude varying from one centimetre to 10 centimetres. Cut surface appears grey/white and yellowish with fibrotic areas demonstrating entrapped mature adipose tissue [3,4].

Upon microscopy, tumefaction expounds haphazard dissemination of thickened collagen bundles commingled with hypo-cellular areas comprised of bland fibroblasts. Miniature vascular articulations appear admixed with the cellular component.

Neoplasm depicts a plaque-like pattern of tumour evolution along with infiltration of adjacent anatomic structures [4,5].

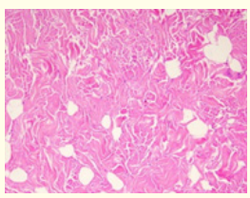

Figure 1: Gardner-type fibroma depicting haphazard dissemination of thick collagen bundles admixed with bland fibroblasts and innumerable vascular articulations [8].

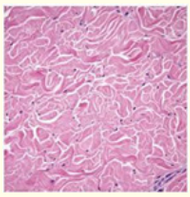

Figure 2: Gardner-type fibroma delineating haphazard dissemination of thick collagen bundles commingled with bland fibroblasts and innumerable vascular articulations [9].

Morphological features	Infectious mononucleosis	Classic Hodgkin's lymphoma	Diffuse large B cell lymphomas EBV+
Nodal compartment	Paracortical	Diffusely effaced	Diffusely effaced
Tumour cells	Absent	Scattered large cells, confluent in nodular sclerosis, syncytial variant	Diffuse, abundant, large cells
Intervening lymphocytes			
B cells	Small to intermediate	Small	Small
T cells	CD8+, small to intermediate	CD4+, small	CD8+, small
Monocytoid B cell hyper-plasia	Frequently present	Absent	Absent
Plasma cells	Variable	Few	Infrequent
Histiocytes	Variable	Variable	Variable
Eosinophils	Infrequent	Abundant	May occur
Necrosis	Focal	Nodular sclerosis subtype+	May occur
Immuno-architecture			
EBV latency type	Type III	Type II	Type I
EBER	Majority cells+	HRS cells+	Large cells+
LMP1	Few small/large cells+	HRS cells+	Large tumour cells+
EBNA2	Predominant +	-	-
EBNA3	+	-	-
CD20	Large cells+	HRS cells faintly+ in 20% lesions	Large cells +
CD3	Small lymphocytes +	HRS cells-	Large cells-
CD30	HRS-like cells+, dim	HRS cells+, intense	Tumour cells+/-
CD45	Majority cells+	HRS cells-	Tumour cells+
CD4, PD-1, ICOS, CXCL13	Immunoblasts-	HRS cells-	Tumour cells-
CD21	GCs+	Residual GCs+	Absent
Molecular assay	Polyclonal B/T cells	Polyclonal B/T cells	Monoclonal IGH rearrangements

Table 1: Differentiation between nodal B cell immunoblastic proliferation and lymphoma [4,5].

EBV: Epstein Barr virus, EBER: EBV encoded RNA, EBNA: Epstein Barr virus nuclear antigen, HRS: Hodgkin's Reed Sternberg cells, GC : Germinal centre.

Gardner-type fibroma appears immune reactive to CD34, cyclin D1, vimentin and nuclear β catenin.

Tumour cells appear immune non reactive to muscle specific actin (MSA), smooth muscle actin (SMA), desmin, oestrogen receptors (ER) and progesterone receptors (PR) [5,6].

Gardner-type fibroma requires segregation from neoplasms as familial adenomatous polyposis, Gardner's syndrome, Turcot's syndrome and attenuated variants of familial polyposis associated with chromosomal mutation of APC gene [6,7].

Neoplasm may be appropriately alleviated by surgical extermination, pre-eminently adopted for superior cosmetic outcomes [6,7].

Bibliography

1. Rabai NA., *et al*. "A Sporadic Giant Mesenteric Desmoid-Type Fibromatosis in a Teenage Female: A Report of a Rare Case". *Cureus* 15.9 (2023): e46087.

2. Asaad SK., *et al*. "Extra abdominal recurrent aggressive fibromatosis: A case series and a literature review". *Molecular and Clinical Oncology* 19.4 (2023): 84.

3. Alba-Pavón P., *et al*. "Analysis of germline variants in paediatric patients diagnosed with desmoid tumors and nuchal-type fibromas". *Translational Pediatrics* 12.9 (2023): 1715-1724.

4. Charifa A., *et al*. "Gardner Syndrome". Stat Pearls International. Treasure Island, Florida (2024).

5. Blackwell MC., *et al*. "Extracolonic manifestations of Gardner syndrome: A case report". *Imaging Science in Dentistry* 53.2 (2023): 169-174.

6. Thomaidis V., *et al*. "A case of early FAP diagnosis with extraintestinal manifestations on the face". *Acta Medica Academica* 48 (2019): 217-224.

7. Huang K., *et al*. "Efficacy and safety of different thermal ablative therapies for desmoid-type fibromatosis: a systematic review and meta-analysis". *Quantitative Imaging in Medicine and Surgery* 13.10 (2023): 6683-6697.

8. Image 1 Courtesy: Science direct.

9. Image 2 Courtesy: Basic medical key.