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Opinion

Wiry and Stringy - Fibroma Ovary

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Fibroma of the ovary emerges as a benign stromal tumour comprised of spindle shaped, ovoid to spherical, fibroblastic cells enmeshed within a variably collagenous stroma. Tumefaction is constituted of variants as fibroma with minor sex cord elements, cellular fibroma and mitotically active cellular fibroma. Cellular fibroma necessitates distinction from diffuse adult type of granulosa cell tumour and fibrosarcoma. Gorlin's syndrome may concur in young females demonstrating bilateral ovarian fibromas.

Cellular fibromas associated with tumour rupture or adhesions appear associated with enhanced possible tumour reoccurrence and mandate extensive monitoring.

Fibroma is a commonly encountered neoplasm of the ovarian stroma and expounds ~4% of ovarian tumours. Cellular fibroma represents ~10% of ovarian fibromas. Tumefaction is uncommonly encountered <30 years wherein mean age of disease emergence is 48 years. An estimated ~25% of subjects with nevoid basal cell carcinoma syndrome (Gorlin's syndrome) appear to concur with ovarian fibromas [1,2]. Tumefaction expresses an autosomal dominant mode of disease inheritance on account of chromosomal mutations of human homologue of patched Drosophila gene (PTCH) [1,2].

Commonly arising within the ovary, tumour depicts neoplastic transformation of ovarian stromal cells concurrent to hereditary or sporadic genetic anomalies [1,2].

Ovarian fibroma is associated with trisomy or tetrasomy 12. Exceptionally, genetic mutations within IDH1 may be observed.

Tumour cells of cellular fibroma display loss of heterozygosity at chromosome 9q22.3 (PTCH1) and chromosome 19p13.3 (STK11). Genomic mutations within FOXL2 are minimal to absent [1,2].

Commonly, clinical symptoms concordant with an ovarian mass as abdominal pain, abdominal distension and enhanced frequency of urination may be encountered. Alternatively, tumour may be incidentally discovered. Torsion of the ovary may occur. Hormonal manifestations are exceptional. Enlarged neoplasms >10 centimetre magnitude are associated with ascites in ~10% instances [1,2].

Syndromic ovarian fibromas are observed <30 years or within the paediatric population. Commonly bilateral in ~75% instances, neoplasm may appear nodular and calcified with frequent medial overlapping. Tumefaction may secrete renin with consequent occurrence of gestational hypertension. Tumour reoccurrence may ensue [1,2].

Nearly 1% subjects depict Meig's syndrome characterized by a triad of benign adnexal mass, ascites and pleural effusion which occurs on account of seepage of fluid from tumour borne ovary into peritoneal cavity along with singular or dual pleural cavities through lymphatic channels or a communication between abdominal and pleural cavity within the designated as lumbocostal triangle [2,3].

Upon frozen section examination, a variably cellular neoplasm composed of spindle shaped cells is encountered. Tumefaction demonstrates a fascicular or storiform pattern of evolution along with or devoid of hyaline plaques or calcification [2,3].

Grossly, a firm, unilateral, well circumscribed, chalky, solid, grey/white, yellow/white or tan/yellow tumefaction with smooth, lobulated surface is observed. Tumour magnitude varies from one centimetre to 21.5 centimetres with a mean of 6 centimetres. Roughly < 10% lesions are bilateral. Cut surface appears whorled. Soft tissue oedema may be observed with consequently emerging soft consistency. Up to ~25% neoplasms depict cystic degeneration

whereas ~10% neoplasms delineate calcification. Roughly ~20% tumours emerge as pedunculated or polypoid tumours confined to the ovarian surface. Haemorrhage or necrosis may or may not be represented.

Cellular fibroma varies from one centimetre to 19 centimetres in diameter with mean magnitude of 8 centimetres to 9 centimetres. Cut surface is tan to yellow, soft and fleshy. Adhesions to superimposed ovarian surface may or may not be discerned.

Ovarian fibroma associated with Gorlin's syndrome configures a bilateral, multinodular and calcified neoplasm [2,3].

Upon microscopy, conventional fibroma recapitulates ovarian cortex and represents as a well circumscribed, non encapsulated, variably cellular tumefaction delineating fascicular or infrequently a storiform pattern of evolution. Tumour cells are entangled within a variably collagenous stroma with occasional hyaline plaques [3,4].

Neoplasm is composed of bland spindle shaped cells imbued with scanty, eosinophilic cytoplasm and ovoid nuclei with tapering ends. Tumour cell cytoplasm appears to merge with circumscribing stroma. Mitotic figures are occasional at ~3 mitoses per 10 high power fields. Tumour cells may display Verocay-like areas wherein cells delineate parallel arrays and are impregnated with minimally wavy spindle shaped nuclei. Focal calcification, oedema, haemorrhage, infarct subtype of necrosis and infrequent aggregates of luteinized cells may be discerned. Exceptionally, intracytoplasmic lipid, eosinophilic hyaline globules, melanin pigment or bizarre nuclei may be discerned [3,4].

Cellular fibroma configures ~10% of ovarian lesions and simulates diffuse subtype of adult granulosa cell tumour. Neoplasm is intensely cellular with minimal intercellular collagen and demonstrates cells with bland, spindle shaped nuclei and ~3 mitoses per 10 high power fields. The terminology of "mitotically active cellular fibroma" represents cellular fibromas with ≥ 4 mitoses per 10 high power fields [3,4].

Fibroma with minor sex cord elements is comprised of sex cord component accounting for < 10% of comprehensive tumour volume. However, the nomenclature is devoid of specific prognostic significance [3,4].

Upon ultrastructural examination, tumour is composed of innumerable attenuated, elongated cells demonstrating intercommunicating cytoplasmic processes intermingled with abundant collagen fibrils. Aforesaid fibrils configure bundles delineating a distinct 'cross banding' pattern comprised of light band circumscribed by attenuated, dark, intensely stained bands reappearing at an interval of 600 Å. Tumour cells are pervaded with scanty cytoplasm, few mitochondria, minimal endoplasmic reticulum and an indistinct cellular perimeter. Tumour cell nuclei are elongated, attenuated and exemplify significant peripheral heterochromatin condensation. Tumour cells are encompassed by a collagenous stroma [3,4].

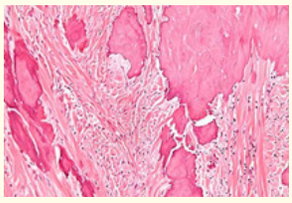

Figure 1: Fibroma ovary demonstrating fascicles of spindle shaped cells imbued with scanty cytoplasm and wavy, elliptical nuclei with pointed ends. The cellular component is surrounded by collagenous stroma [7].

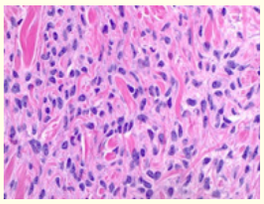

Figure 2: Fibroma ovary delineating spindle shaped cells pervaded with minimal cytoplasm and wavy, ovoid nuclei with tapering ends. Circumscribing stroma is collagenous [8].

Ovarian fibroma appears immune reactive to Wilm's tumour 1 (WT1) antigen, steroidogenic factor 1 (SF1), FOXL2, inhibin, vimentin, CD56, β oestrogen receptor (ER), progesterone receptor (PR) or smooth muscle actin (SMA) [5,6].

Reticulin stain demonstrates an individual pericellular pattern of collagen fibre distribution and differentiates fibroma from diffuse subtype of adult granulosa cell tumour [5,6].

Tumour cells appear immune non reactive to CD10, desmin, caldesmon, CD99, calretinin, S100 protein and CD34 [5,6].

Fibroma ovary requires segregation from neoplasms as diffuse adult granulosa cell tumour, ovarian thecoma, sclerosing stromal

Stage I	T1	N0	M0
Stage IA	T1a	N0	M0
Stage IB	T1b	N0	M0
Stage IC	T1c	N0	M0
Stage II	T2	N0	M0
Stage IIA	T2a	N0	M0
Stage IIB	T2b	N0	M0
Stage IIIA1	T1/2	N1	M0
Stage IIIA2	T3a	Any N	M0
Stage IIIB	T3b	Any N	M0
Stage IIIC	T3c	Any N	M0
Stage IV	Any T	Any N	M1
Stage IVA	Any T	Any N	M1a
Stage IVB	Any T	Any N	M1b

Table 1

tumour, leiomyoma, endometrial stromal tumour, low grade metastatic gastrointestinal stromal tumour, ovarian stromal hyperplasia, massive oedema of the ovary and ovarian fibromatosis or fibrosarcoma.

Cogent morphological assessment is necessitated for definitive neoplastic discernment. Precise macroscopic and frozen section examination appears beneficial in tentative evaluation of the tumour. Ovarian fibroma may induce nonspecific augmentation of serum CA125 levels [5,6].

Upon ultrasonography, a solid, homogeneous, hypoechoic tumefaction with posterior acoustic shadows is observed, recapitulating the features of a pedunculated sub-serosal uterine leiomyoma. Neoplasm expounds a heterogeneous echogenicity, especially within tumefaction demonstrating necrosis, haemorrhage or cystic degeneration [5,6].

Computerized tomography (CT) delineates a solid tumefaction with minimal hypo-attenuation and minimal, delayed contrast enhancement. Tumour calcification may exceptionally ensue.

T1 weighted magnetic resonance imaging (MRI) expresses a homogeneous signal with minimal intensity [5,6].

T2 weighted imaging expounds a well circumscribed tumour mass with minimal signal intensity. Besides, hyper-intense areas may appear due to oedema or cystic degeneration. Characteristically upon T2 weighted imaging, a hypo-intense band appears to demarcate the tumour from uterine cavity.

Images with administration of gadolinium contrast demonstrates heterogeneous enhancement. Neoplasm may simulate a malignant tumour [5,6].

Ovarian fibroma may suitably be subjected to surgical extermination procedures as salpingo-oophorectomy, oophorectomy or ovarian sparing procedures along with or in the absence of hysterectomy. Aforesaid manoeuvers are contingent to age of incriminated subject.

Cellular fibroma necessitates extensive monitoring, especially lesions with involvement of ovarian surface, intraoperative rupture or extra-ovarian tumour dissemination [5,6].

Ovarian fibroma is associated with superior prognostic outcomes. Around 6% of cellular fibromas and ~10% cellular fibromas with significant mitosis implicate the ovarian surface or demonstrate extra-ovarian adhesions. Nearly 11% of cellular fibromas and 13% of cellular fibromas with significant mitosis delineate significant extra-ovarian neoplastic dissemination. Cellular fibromas may reoccur following a significant duration, thereby warranting meticulous monitoring [5,6].

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7. Image 1 Courtesy: Libre Pathology.

8. Image 2 Courtesy: Pathology outlines.