REVIEW ARTICLE

Miscellaneous tumour-like lesions of the ovary: cross-sectional imaging review

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ABSTRACT. Miscellaneous tumour-like ovarian lesions are histobiologically diverse, and are often mistaken for the more common ovarian cancers, leading to aggressive management. Knowledge of characteristic clinical, laboratory and imaging findings of these select non-neoplastic ovarian entities allows correct diagnoses and permits optimal management.

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There is a wide spectrum of uncommon, tumour-like, ovarian lesions that exhibit varied clinical manifestations and characteristic histomorphology. Because of their rarity and atypical clinical presentation, these lesions are commonly misdiagnosed as cancers, with resultant radical surgery. Select non-neoplastic entities demonstrate characteristic clinical, laboratory and imaging findings to allow correct diagnoses that permit optimal management. In this article, we review clinical and imaging manifestations of select uncommon tumour-like lesions of the ovary, including infective and inflammatory lesions, pregnancy-related ovarian lesions, ovarian torsion, massive ovarian oedema and ovarian fibromatosis.

Infective and inflammatory causes of ovarian masses

Acute oophoritis

Approximately 1 million females are afflicted with pelvic inflammatory disease (PID) and nearly 275 000 are hospitalised every year in the USA [1]. *Neisseria gonorrhoeae* or *Chlamydia trachomatis* account for the majority of ascending infections; 30–40% of infections are polymicrobial [1]. Secondary infertility due to tubal occlusion is a known complication of PID. Ovarian involvement is secondary to salpingitis and is often associated with signs and symptoms of PID. Clinical presentation includes nausea, vomiting, fever, pelvic pain, vaginal discharge, dyspareunia, and adnexal or cervical tenderness. Pathologically, the ovaries are enlarged and show inflammatory infiltrates at histology.

On imaging, the ovaries are enlarged and may show variable enhancement (Figure 1). Other associated findings of PID include soft-tissue stranding and infiltration of pelvic floor fascial planes, thickening of the uterosacral ligaments, and thickened/dilated fallopian tubes. With chronicity, the disease progresses to development of tubo-ovarian or pelvic abscesses (Figure 2). Enhancing perihepatic peritoneum with other evidence of PID suggests Fitz-Hugh–Curtis syndrome [2]. Most (60–80%) tubo-ovarian abscesses may resolve with antibiotics; surgery is reserved for refractory cases.

Xanthogranulomatous oophoritis

The xanthogranulomatous inflammatory process commonly involves the kidneys and the gallbladder; female genital tract involvement is extremely rare. In the female genital tract, xanthogranulomatous inflammation is often confined to the endometrial cavity; very few ovarian cases are reported. The average age of occurrence is 31 years [3]. It is thought that recurrent PID contributes to xanthogranulomatous oophoritis (XGO). Contributing factors include chronic bacterial infection (*Escherichia coli*, *Proteus* and *Staphylococcus aureus*), inadequate treatment, coexisting endometriosis and intra-uterine contraceptive devices [4]. Patients with XGO clinically manifest with fever and pelvic pain. Microscopically, the hallmarks of XGO include tissue destruction and conspicuous inflammatory cellular infiltrates (foamy histiocytes, multinucleated giant cells, plasma cells, fibroblasts and neutrophils) [4].

Contrast-enhanced CT shows complex solid–cystic lesions with thick enhancing walls and variably enhancing solid intramural nodules (Figure 3). The solid intramural nodules appear hypointense on T_1 weighted MRI

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Figure 1. A 32-year-old female with acute oophoritis. Coronal contrast-enhanced CT scan through the pelvis shows an enlarged right ovary with a complex cyst (white arrows) showing thin internal septations (black arrowhead) and heterogeneously enhancing peripheral solid areas (black arrow) with neighbouring inflammatory stranding (white arrowhead). Given the complex appearance of the right ovarian cyst on imaging, a fertility-preserving cystectomy was performed; the histopathology suggested a diagnosis of acute oophoritis rather than a neoplasm.

[3]. The ovarian lesion may appear T_1 hyperintense, and may show enhancing solid areas [5]. Oophorectomy is the treatment of choice for XGO.

Ovarian tuberculosis

Tuberculosis (TB) may spread to the genital tract through the haematogenous or lymphatic route. Genital

Figure 3. A 42-year-old female with xanthogranulomatous oophoritis. Axial contrast-enhanced CT scan through the pelvis demonstrates bilateral complex solid-cystic lesions with markedly thick enhancing walls and internal septations (arrows). The complex appearance of the masses and the possibility of underlying malignancy prompted a surgical exploration. However, the post-operative histopathology suggested a diagnosis of xanthogranulomatous oophoritis. Star indicates the uterus.

tuberculosis (GTB) is infrequent, usually asymptomatic and incidentally detected during infertility work-up; this constitutes about 3% of cases of infertility in the developing world [6]. In the descending order of frequency, TB involves endometrium (72%), fallopian tubes (34%), ovary (12.9%) and cervix (2.4%) [7]. 40% of GTB patients also have tubercular peritonitis and raised CA 125 levels. Erroneous diagnosis of metastatic ovarian carcinoma is not uncommon; an enzyme-linked immuno-spot and polymerase chain reaction may be contributory to a pre-operative diagnosis of peritoneal TB [8]. The presence of chronic inflammatory cells with

(a)

(b)

Figure 2. Ovarian abscesses in two different patients. (a) A 36-year-old female with pelvic inflammatory disease. Axial contrast-enhanced CT scan through the pelvis shows a unilocular right ovarian cyst with a thick and enhancing wall (arrow); the mesosalpinx is thickened (arrowhead). UB, urinary bladder; U, uterus. (b) A 28-year-old female with pelvic pain. Axial contrast-enhanced CT scan demonstrates a complex multiloculated left adnexal cystic lesion with a thick and enhancing wall and septations (black arrows); the left ovary and left mesosalpinx were not seen separately. A small loculated fluid collection was also noted anterior to the lesion (white arrowhead).

Figure 4. A 31-year-old female with ovarian tuberculosis who presented clinically with an adnexal mass, increasing abdominal girth and elevated serum cancer antigen 125. (a) Axial contrast-enhanced CT scan through the pelvis demonstrates bilateral, complex, enhancing solid–cystic adnexal lesions (arrows); bilateral ovaries and mesosalpinx were not seen separately. U, uterus. (b) Axial contrast-enhanced CT scan through the mid-abdomen shows moderate ascites (star) and thickened peritoneal reflections (arrowhead). Clinical and imaging findings were indistinguishable from ovarian malignancy and peritoneal carcinomatosis. Surgical exploration, hysterectomy and bilateral salpingo-oophorectomy and post-operative histopathology established the diagnosis of ovarian tuberculosis.

caseating granulomas, the hallmark of TB, clinches the diagnosis.

Imaging often demonstrates an adnexal mass or a tubo-ovarian abscess with ascites. Contrast-enhanced CT may show complex solid–cystic adnexal masses, ascites, omental or mesenteric infiltrations, and peritoneal thickening (Figure 4). Calcification and lymphadenopathy may also be present. MR may demonstrate a T_2 hypointense, irregular-walled, tubo-ovarian mass along with nodular thickening of the peritoneum [5]. A multidrug anti-tubercular regimen for a total of 6–9 months cures tuberculosis in most compliant cases.

Inflammatory pseudotumour

Also referred to as inflammatory myofibroblastic tumour or myofibroblastoma, inflammatory pseudotumour (IPT) is a rare idiopathic proliferative lesion, commonly seen in young adults and children. IPT may involve any organ but most commonly occurs in the lung or orbit. The most accepted theory of pathogenesis supports immunological factors and a post-inflammatory reparative process due to surgery, infection or trauma [9]. Patients may show fever, weight loss and pelvic pain. IPT histologically consists of vimentin-positive myofibroblastic spindle cells admixed with lymphocytes/plasma cells and variable fibrosis. IPT may show tissue invasion and disease recurrence. Imaging findings of IPT are non-specific. IPT appears as complex cystic–solid adnexal masses that may be indistinguishable from the more common ovarian neoplasms (Figure 5). The diagnosis is based on histopathology following biopsy. The prognosis is favourable following complete surgical resection. The role of non-steroidal anti-inflammatory drugs is under evaluation [9].

Autoimmune oophoritis

Autoreactive T-cells, antibodies to adrenal cortex and/or ovarian steroidogenic enzymes and coexisting primary ovarian insufficiency constitute the entity of autoimmune oophoritis. Autoimmune oophoritis is often associated with other autoimmune disorders of the thyroid and adrenals; it can also be a component of autoimmune polyendocrine syndrome in adolescents [10, 11]. Patients present with signs of premature ovarian failure such as oligomenorrhoea and amenorrhoea. Patients are aged 17–48 years, with a mean age of 31 years. This condition uniquely affects steroid-producing cells (oestradiol

Figure 5. A 38-year-old female with an ovarian inflammatory pseudotumour. Axial contrast-enhanced CT scan through the pelvis demonstrates a large multiloculated complex cystic lesion in the left adnexae (arrows); enhancing septations (arrowhead) and solid component (star) are also seen; the imaging appearance is indistinguishable from an ovarian malignancy. U, uterus. The post-operative histopathological diagnosis was an inflammatory pseudotumour of the ovary.

production) but spares the primordial follicles and the granulosa cells (secreting inhibin B). Elevated follicle-stimulating hormone (FSH) levels (in response to reduced oestradiol levels) stimulate ovarian follicle growth and cyst formation [10]. The end point may be ovarian failure and atrophy. Pathologically, the ovaries may be normal or enlarged with multiple cysts in the early stage. Late-stage disease is characterised by small fibrocystic ovaries [11]. At histology, the condition is characterised by chronic inflammatory cell infiltration among the theca cells of the follicles.

Imaging may show bilaterally enlarged and multicystic ovaries (cysts measuring up to 3 cm). On MR, the ovaries may demonstrate a “gyriform” appearance with a peripheral rind of low T_2 signal (similar to autoimmune pancreatitis) and preserved ovarian follicles (Figure 6a,b). Coexistent retroperitoneal and mesenteric fibrosis may also be seen (Figure 6c). Primary treatment is hormone replacement therapy.

Pregnancy-related ovarian masses

Ovarian pregnancy

Ovarian pregnancy is a rare (3.3%) form of ectopic implantation, with an incidence of 1:6000 to 1:40 000 pregnancies [12]. Use of ovulatory stimulation drugs, contraceptive devices and reproductive assistive techniques predispose to ectopic pregnancies. Patients may manifest abdominal pain, vaginal bleeding and adnexal mass with or without hypotension. Spiegelberg criteria to diagnose ovarian pregnancy include intact fallopian tubes with their fimbriae, demonstration of an ovarian gestational sac and the presence of ovarian tissue within the specimen attached to the gestation sac. Histopathology reveals the presence of chorionic villi and tissue from the corpus luteum.

Transvaginal ultrasound may show a gestational sac within the ovary (Figure 7) with detectable embryonic heart motion and surrounding hypervascularity [13]. Most (>90%) ovarian pregnancies get terminated in the first trimester; only one case has been reported to progress up to full term [14]. Chronic, unruptured, non-viable ovarian pregnancy may appear as a cystic mass with haemorrhage with or without solid components (Figure 8). Laparoscopy is considered the gold standard for diagnosis and treatment. Currently, fertility-sparing laparoscopic surgery is the treatment of choice. Medical treatment consists of the use of methotrexate and prostaglandins.

Luteoma of pregnancy

Pregnancy luteoma (PL) is an uncommon non-neoplastic, hormone-dependent, tumour-like lesion of pregnancy that disappears in the puerperium. First described in 1963, fewer than 200 cases have been reported to date in the English literature [15]. PL commonly occurs in the third to fourth decade; African American females are commonly affected. PLs occur as a result of overproliferation or atypical response of luteinised stromal cells under the influence of beta-human chorionic gonadotrophin (β -hCG). Corpus luteum of pregnancy may coexist with PL. The majority of patients with PL are asymptomatic, being incidentally detected during antenatal or post-partum imaging, or intra-operatively during Caesarean section. Up to 25% of these patients may however manifest with endocrine abnormality, causing maternal and foetal hirsutism and virilisation [16]. Luteomas of pregnancy frequently are large, measuring 6–20 cm in diameter. Up to 50% of PLs are multiple, with one-third being bilateral in distribution. At histology, luteoma shows diffuse proliferation of polygonal eosinophilic cells with little or no lipid [17].

Figure 6. A 36-year-old female with autoimmune oophoritis. (a) Coronal T_2 weighted image through the pelvis shows an enlarged right ovary (arrows) with T_2 hypointense peripheral rind (arrowhead) and “gyriform” outline. The imaging appearance is reminiscent of T_2 hypointense rind of the pancreas in autoimmune pancreatitis. (b) Sagittal T_1 weighted post-contrast fat-suppressed image shows the presence of preserved ovarian follicles (arrow) and enhancement of peripheral rind (arrowhead). (c) Coronal contrast-enhanced CT scan through the mesenteric root shows enhancing haziness of the mesentery (circled) consistent with sclerosing mesenteritis. Clinical and imaging findings were concerning for an autoimmune process. Surgical excision of the ovary was performed, which revealed dense lympho-plasmacytic infiltration with areas of fibrosis within the ovary consistent with the diagnosis of autoimmune oophoritis.

Figure 7. A 26-year-old female with ovarian pregnancy who presented with 6 weeks' amenorrhoea, pelvic pain and elevated serum beta-human chorionic gonadotrophin. Transvaginal ultrasound demonstrated an empty uterine cavity (not shown) and a well-defined cystic structure consistent with a gestational sac (large arrows) and a yolk sac (star) within the left ovary; the ovarian parenchyma and follicle (small arrow) are visible anterior to the gestational sac. A diagnosis of probable ovarian pregnancy was established on imaging and confirmed on surgery.

Figure 8. A 39-year-old female with chronic ectopic ovarian pregnancy. Axial contrast-enhanced CT scan through the pelvis shows a complex cystic mass (arrows) with heterogeneous enhancement of the solid component (arrowhead); the findings are non-specific. The uterus (U) is normal in size and shows normal enhancement. Surgical exploration and post-surgical histopathological analysis established the diagnosis of chronic right ovarian pregnancy.

On imaging, PLs manifest as solid or solid-cystic masses (Figure 9). PLs appear as variable T_1 signal intensity and T_2 hypo- or hyperintense signal masses with variable enhancement [18]. Misdiagnosis may lead to unnecessary oophorectomy. Follow-up scans show serial regression in size, with complete resolution in the post-partum state, usually within 3 months of delivery.

Hyperreactio luteinalis

Hyperreactio luteinalis (HL) is a gonadotrophin-induced benign condition characterised by the presence of numerous thin-walled luteinised follicular cysts with hypertrophy of the theca interna and marked bilateral ovarian enlargement (up to 35 cm). HL is commonly described in conditions with high β -hCG levels such as gestational trophoblastic disease (hydatidiform mole and choriocarcinoma) and multiple gestations. Approximately 10–50% of patients with gestational trophoblastic disease may have HL [19]. Usually asymptomatic, HL may present with acute pelvic pain secondary to haemorrhage, rupture or torsion. Virilisation may be seen in a small subset of patients [18]. More than half of patients with HL are seen in the third trimester, with 16% in the first trimester and peripartum period [20]. Pathologically, HL is characterised by moderate to massive ovarian enlargement with multiple large (1–3 cm) thin-walled follicles and oedematous and vascularised gonadal tissue centrally. Histological hallmarks consist of luteinisation and hypertrophy of the theca interna layer.

On imaging, multiple ovarian cysts are seen in an enlarged ovary (Figure 10). Some cysts may have T_1 hyperintensity due to haemorrhage; the oedematous and vascularised parenchymal centre may reveal T_1/T_2 isointensity [21]. Ascites is uncommonly seen [21]. Management of HL primarily involves recognition of the underlying cause of excessive β -hCG levels. Rarely, HL may be seen with a normal singleton pregnancy without elevated β -hCG levels [22]. Typically, cysts of HL gradually involute and the condition resolves spontaneously following termination of pregnancy or treatment for gestational trophoblastic disease. Spontaneous regression during pregnancy is extremely rare. Surgical intervention is indicated in cases complicated by infarction, haemorrhage or virilisation.

Ovarian hyperstimulation syndrome

Ovarian hyperstimulation syndrome (OHSS) is an iatrogenic complication of fertility medications, presenting as bilaterally enlarged ovaries (up to 25 cm) and multiple cysts [23]. Increased ovarian angiogenesis in response to gonadotrophins and amplified responses to normal luteinising hormone (LH) leading to activation of the cascade of vascular permeability in ovarian vessels may explain its pathogenesis. The patients commonly manifest abdominal pain or bloating. Mild OHSS is characterised by uncomplicated ovarian enlargement with multiple cysts, moderate disease is characterised by ascites, and severe disease is associated with pleural effusion, haemoconcentration, thrombosis and oliguria [24].

Figure 9. A 35-year-old pregnant female with luteoma of pregnancy. Transvaginal ultrasound demonstrates (a,b) a well-circumscribed complex cystic lesion in the right ovary (arrows) with multiple echogenic internal septations (arrowhead) and a non-vascular solid component (star). The ultrasound appearance was non-specific, and sequential scans revealed gradual regression and complete resolution within a few weeks.

Up to 65% of patients may have sonographically detectable mild OHSS during infertility treatment. OHSS is self-limiting and typically resolves within 1–2 weeks; complete recovery is expected at the onset of the next menstrual cycle [23]. OHSS may persist and become more severe during pregnancy. OHSS has also been reported to occur spontaneously without ovulation induction [25]. Imaging depicts bilaterally enlarged multicystic ovaries with ascites or pleural effusion (Figure 11). Ovarian torsion or rupture may complicate OHSS. Treatment is supportive, including basic cardiovascular and respiratory support, alleviating pain and anxiety, and prevention of complications.

Polycystic ovarian syndrome

Polycystic ovarian syndrome (PCOS) is the most common cause of secondary amenorrhoea associated

with a hyperandrogenic state. PCOS may involve 5–10% of reproductive age-group females and usually presents in the 15–30 years age group. The diagnosis of PCOS is primarily made on clinical and biochemical grounds. Imaging plays a corroborative role in supporting the diagnosis and excluding other ovarian neoplasms, which may cause hyperandrogenism [26]. Common clinical presentations of PCOS include menstrual irregularity (80%), hirsutism (62%) and coexisting obesity (31%) [27]. Endometrial hyperplasia, and rarely endometrial carcinoma (1%), may be seen in association with PCOS. An estimated 25% of patients with endometrial cancer who are under 40 years old may have PCOS [28]. Characteristic laboratory abnormalities include an elevated LH:FSH ratio (exceeding 2:1) and serum androgen level.

Ultrasonography is the imaging investigative modality of choice and shows enlarged ovaries (usually bilateral, 2–5 times normal) with multiple small peripheral cysts. The presence of multiple (>10–12) small (<10 mm)

Figure 10. A 34-year-old female with hyperreactio luteinalis. (a) Axial contrast-enhanced CT scan through the pelvis demonstrates an enlarged normally enhancing uterus (arrows) showing heterogeneous myometrial enhancement with hypoattenuating material within the endometrial cavity (star), consistent with choriocarcinoma. (b) Axial contrast-enhanced CT scan through the lower abdomen, cephalic to (a), at the level of the uterine fundus shows a markedly enlarged left ovary with multiloculated cystic appearance (arrow). The septations are thin (arrowhead) and the loculi (star) are small (<3 cm). A similar appearance was seen in the right ovary (not shown).

Figure 11. A 33-year-old female with ovarian hyperstimulation and a history of secondary infertility and induction with gonadotrophins. Transvaginal ultrasound shows the bilaterally enlarged ovaries (arrows) with multicystic appearance; cysts are devoid of haemorrhage or internal echoes (star).

peripheral cysts suggests this diagnosis (Figure 12). Increased stromal echogenicity, one of the most specific findings, is seen in up to 67% of cases. Up to 94% of cases have either an ovarian volume exceeding 6.2 ml or >10 follicles (2–8 mm) [29]. Imaging plays a vital role in excluding functional ovarian neoplasms such as sex cord stromal tumours and Sertoli–Leydig cell tumours, which may be associated with hyperandrogenism [26]. Lifestyle changes, including regular exercise, may reduce both insulin and androgen levels, and restore ovulation, particularly in obese patients. Treatment paradigms

include use of oral contraceptive pills to regulate menstruation and clomiphene citrate to induce ovulation.

Stromal hyperthecosis

Stromal hyperthecosis (SH) refers to stromal proliferation without steroid cell hyperplasia and is characterised by hyperandrogenism [30]. Clinical manifestations include virilisation, obesity, hypertension and hyperinsulinaemia. SH is usually seen in females of reproductive age but is rarely encountered in post-menopausal females [31]. Hyperoestrogenic symptoms may predominate in post-menopausal females, and may be associated with endometrial hyperplasia or carcinoma [32]. The pathogenesis of stromal hyperthecosis is uncertain. Pathologically, the ovaries may be normal or enlarged (up to 8 cm) and show diffuse luteinised stromal cell proliferation involving both the cortex and the medulla [33]. Stromal luteomas and thecomas may be seen in association with this condition [34, 35].

On imaging, the ovaries in stromal hyperthecosis may appear normal or enlarged (bilateral>unilateral), without cysts. A solid mass may also be seen infrequently, due to a “nodular” form or a coexisting fibrothecoma [30]. The periphery of the ovarian masses appears T_1 and T_2 isointense (to myometrium), which is described as characteristic of stromal hyperthecosis [36]. In contrast to PCOS, SH is not accompanied by altered serum LH:FSH levels and rarely responds to an oral contraceptive regimen alone. SH is efficiently managed with a combination of long-term gonadotrophin-releasing hormone (GnRH) agonist and oestrogen replacement therapy. Bilateral oophorectomy is the treatment of choice in symptomatic patients, and results in reversal of hyperandrogenic syndrome, hypertension and glucose intolerance.

Figure 12. A 21-year-old female with polycystic ovarian syndrome. Transvaginal ultrasound shows classical findings of polycystic ovarian syndrome: enlarged right ovary (arrows) with multiple (>10) small (4–6 mm) follicles (arrowhead) arranged at the periphery with echogenic stroma (S). The other ovary also had a similar appearance (not shown). Laboratory evaluation confirmed an elevated ratio of luteinising hormone to follicle-stimulating hormone and raised levels of androgens.

Figure 13. A 31-year-old female with ovarian infarction who presented clinically with acute left lower quadrant pain and left adnexal mass. (a,b) Sagittal T_2 weighted and axial T_2 weighted fat-suppressed MR images through the left pelvis show a diffusely hyperintense enlarged left ovary (arrows) with complete loss of normal architecture. Normal follicles and ovarian stroma are not appreciated. An incidental large para-ovarian cyst was also noted (star). (c) Axial T_1 weighted fat-suppressed post-contrast MR image through the left pelvis demonstrates the lack of ovarian enhancement (arrows). Pre-operatively, a diagnosis of primary ovarian malignancy with enhancing mural nodules/papillary excrescences was suggested. However, surgical pathology revealed a large para-ovarian cyst with total infarction of the left ovary.

Other miscellaneous ovarian lesions

Ovarian torsion

Ovarian torsion is an important cause of acute pelvic pain; early and accurate diagnosis permits timely surgery to salvage the ovary. The median age of presentation of ovarian torsion is 33.5 years [37]. Approximately 50–60% of cases are due to underlying ovarian masses or cysts (most commonly mature cystic teratomas), frequently exceeding 4–6 cm in diameter. Patients may present with nausea, vomiting, acute pelvic pain and adnexal mass. The diagnosis is challenging and needs a high level of clinical suspicion.

Complete “twisting” along the vascular pedicle leads to infarction; partial or intermittent torsion causes venous and lymphatic obstruction leading to diffuse ovarian oedema. An enlarged (>4 cm) unilateral ovary with multiple peripherally arranged small cysts (“string of pearls” sign) suggests ovarian torsion. The enlarged ovary is frequently located in the midline, superior to the uterine fundus. Ascites and a combination of twisted pedicle and whirlpool sign are reliable ultrasound features [38]. the presence of blood flow in the artery but lack of flow in the twisted pedicle is predictive of non-viability of the ovary [39]. MR shows T_2 hyperintensity due to oedema and lack of contrast enhancement; MR helps to assess pre-operative viability. The infarcted ovary may erroneously

Figure 14. A 25-year-old female with massive ovarian oedema. (a) Transvaginal ultrasound with colour Doppler demonstrates a diffusely enlarged and hypoechoic right ovary with poor parenchymal vascularity (arrows) and preserved follicles (arrowhead). The preservation of follicles differentiates massive ovarian oedema from infarction seen with complete torsion. (b) Axial contrast-enhanced CT scan through the pelvis demonstrates a solid hypodense mass-like appearance of the right ovary (star) with subtle enhancement of the periphery (arrow). U, uterus.

Figure 15. A 44-year-old female with ovarian fibromatosis. (a) Axial contrast-enhanced CT scan through the pelvis shows a large heterogeneously enhancing solid right ovarian mass (arrow) with focal internal areas of hypoattenuation (star). (b) Sagittal T_2 weighted fat-suppressed image through the right pelvis shows diffuse iso- to slightly hypointense signal of the ovarian parenchyma (arrows) with preserved follicles (arrowhead) distributed in random fashion. A thick hypointense rind attributed to the low intensity of the fibrous tissue surrounds the ovary (small arrows). (c) Coronal contrast-enhanced T_1 weighted fat-suppressed image through the right pelvis shows enhancing ovarian parenchyma (arrows), preserved follicles (arrowhead) and thick peripheral hypointense rind around the ovary (small arrow). Post-operative histopathology confirmed the diagnosis of ovarian fibromatosis.

be recognised as a solid component of coexisting cyst (primary cause of torsion) on imaging (Figure 13).

Massive ovarian oedema

Massive ovarian oedema (MOO) refers to marked, tumour-like, ovarian enlargement due to intermittent ovarian torsion, and resultant partial venous and lymphatic obstruction. MOO is commonly seen in young females (6–33 years) with a mean age of presentation of 21 years [17]. Most patients with MOO present with intermittent pelvic pain of several months' duration and adnexal mass. Abnormal uterine bleeding, elevated androgens and abdominal distension are infrequently seen. Precocious puberty in infants due to MOO has been described in the literature [40]. Most cases are unilateral, with predominant (75%) involvement of the right ovary [17]. Grossly, ovaries measure 5–35 cm in diameter (average 11 cm). Histologically, the ovarian architecture is preserved with a thick and fibrotic outer cortex.

The imaging features of MOO correspond to the degree of torsion and oedema. On ultrasonography, MOO appears like a hypoechoic hypovascular solid ovarian "mass" and can be confused with a pedunculated leiomyoma or an ovarian neoplasm (Figure 14). MRI may demonstrate T_1 hypointense and T_2 hyperintense signal intensity, as well as poor contrast enhancement of the involved ovary. After the exclusion of a neoplasm with a wedge biopsy, the treatment consists of oophorectomy, but oophorectomy is not infrequent.

Ovarian fibromatosis

Ovarian fibromatosis (OF) is a rare fibrosing condition that is thought to be a sequela of long-standing massive ovarian oedema. Ovarian fibromatosis commonly occurs in young females (13–39 years) with a mean age of presentation of 25 years [17]. Clinical presentations include menstrual irregularity, abdominal pain, and infrequently hirsutism and virilisation [17]. Pathologically, ovarian

fibromatosis is characterised by homogeneous ovarian enlargements, which measure between 8 and 14 cm, appear predominantly solid on the cut surface and show predominantly fibrous component on histopathological examination [17]. Thickening of the ovarian cortex is seen and the ovarian follicles are spared.

OF manifests as a well-circumscribed, solid ovarian mass without a significant increase in vascularity. The echogenicity of OF on ultrasound is variable; preserved ovarian follicles are seen along the periphery of the mass. CT scan shows a relatively hyperdense (to myometrium), homogenous, solid and variably enhancing solid ovarian mass. Presence of punctate calcification has also been described (Figure 15a). On MR, OF shows T_1 and T_2 hypointense signal intensity. A black garland-like appearance has been described on T_2 weighted images, possibly attributed to the low intensity of the fibrous tissue that surrounds the ovary (Figure 15b,c) [41]. OF needs to be differentiated from other masses with predominant fibrous content such as fibromas, thecomas, fibrothecomas, Brenner tumours and Krukenberg tumours [42]. Treatment of this benign entity is salpingo-oophorectomy.

Conclusion

Miscellaneous ovarian non-neoplastic entities comprise a wide spectrum. Select tumour-like lesions of the ovary demonstrate characteristic imaging findings that may permit correct diagnosis. Biopsy with histopathological evaluation is warranted in some cases to establish definitive diagnosis. While unilateral or bilateral oophorectomy cures some conditions, conservative management may be warranted in others. Accurate characterisation and diagnosis is essential to institute optimal management while avoiding radical surgery.

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