Pelvic incidentalomas

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Abstract

Recent advances in multi-detector computed tomography, magnetic resonance imaging, and ultrasound have led to the detection of incidental ovarian, uterine, vascular and pelvic nodal abnormalities in both the oncology and non-oncology patient population that in the past remained undiscovered. These incidental pelvic lesions have created a management dilemma for both clinicians and radiologists. Depending on the clinical setting, these lesions may require no further evaluation, additional immediate or serial follow-up imaging, or surgical intervention. In this review, guidelines concerning the diagnosis and management of some of the more common pelvic incidentalomas are presented.

Keywords: Incidentaloma; incidental ovarian mass; Krukenberg tumors; ovarian metastases; ovarian cyst; breast cancer metastases; colon cancer metastases; gastric cancer metastases; pelvic lymph nodes; pelvic veins.

Introduction

Recent advances in computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound have led to the detection of incidental ovarian, uterine, vascular and pelvic nodal lesions in both the oncology and non-oncology patient populations that in the past remained undiscovered. These incidentalomas are unexpected, asymptomatic abnormalities that are discovered serendipitously while searching for other pathology[1–9]. Incidental pelvic lesions have created a management dilemma for both clinicians and radiologists, particularly in the oncology patient in whom any mass, clinical or subclinical, warrants further evaluation. Strategies for optimizing patient management of these lesions are only beginning to emerge in terms of deciding which of these incidentalomas can be ignored, which can simply be monitored over time, and which lesions require more aggressive workup.

Subjecting the patient to unnecessary testing and treatment carries its own set of risks that can result in an injurious and expensive cascade of imaging and intervention. The exhaustive evaluation performed in some patients reflects the unwillingness of many physicians to accept uncertainty even in the case of a very rare diagnosis. This unwillingness is in part driven by a paucity of data on the topic, the lack of clear-cut algorithms with regard to diagnostic and treatment strategies, fear of potential malpractice liability, and/or the anxiety of the patient. In this review, guidelines concerning the approach to some of the more common pelvic incidentalomas are presented.

Ultimately the following three questions need to be answered in patients with pelvic incidentalomas: (1) does the incidentaloma put the patient at risk for an adverse outcome? (2) can primary or metastatic malignancy be accurately and confidently differentiated from a benign incidentaloma? and (3) if a lesion is benign, might it still require surgical intervention?

Ovarian incidentalomas

Prevalence

Some 5–10% of women in the United States will undergo surgery for a suspected adnexal tumor during their lifetime leading to 160,000 to 289,000 hospitalizations
annually\textsuperscript{10}. The number of incidentally discovered ovarian masses has significantly increased with technical improvements in and increased utilization of cross-sectional imaging\textsuperscript{11}.

For most of the twentieth century, the traditional management of a postmenopausal woman with a cystic adnexal lesion was surgical removal. The rationale for the surgical option was based on the fact that more than 85% of ovarian malignancies are epithelial in origin and most of these cancers are cystic. This approach was promulgated in the era before cross-sectional imaging when only masses sufficiently large to be palpable on physical examination were detected.

In the 1980s and 1990s, a number of studies were published based on ultrasound, CT (Fig. 1) and MRI, and concluded that simple cystic lesions are quite common in both pre- and postmenopausal women and that simple cysts less than 5 cm in diameter are not likely to be malignant\textsuperscript{12/C15116}. In a prospective review of 184 asymptomatic, non-oncologic, postmenopausal volunteers, transabdominal and transvaginal ultrasound were performed and cysts were found in 17% of the women, of which only one was palpable. On follow-up scans, 53% of cysts disappeared, 28% remained constant in size, 11% enlarged greater than 3 mm, 3% decreased in size and 6% both increased and decreased in size on repeated examination. There was no statistical relationship found between the presence of cysts or cyst activity with respect to the type of hormone replacement or length of time since menopause. The authors concluded that simple adnexal cysts <3 cm in size in greatest diameter that have a normal resistive index and normal CA-125 level are most likely benign and may be followed up safely with ultrasound\textsuperscript{12}.

In a retrospective review of 3448 CT scans performed in both pre- and postmenopausal women, Slanetz \textit{et al.}\textsuperscript{15} found incidental adnexal lesions in 5% of cases. Of the 168 patients who had incidental adnexal lesions, 72 had had extra-ovarian neoplasms. In both pre- and postmenopausal women, these lesions most often proved to be benign, even in the presence of a known malignancy (excluding ovarian carcinoma). In the 40% of patients with known non-gynecologic malignancies, no primary ovarian neoplasms were discovered, and only 3% of the lesions represented metastases, all of which were found in postmenopausal women. No primary ovarian malignancies were discovered incidentally in the non-oncology population either. The authors recommended that postmenopausal women should at least have follow-up imaging to characterize the lesion definitively, to document its resolution or at least to insure its stability especially if the lesion is >3 cm, has a heterogeneous and/or predominantly solid appearance on CT.

**Metastatic disease to the ovaries**

Some 5–20% of ovarian malignancies represent metastases and in up to 38% of cases, the discovery of the metastasis precedes the detection of the primary neoplasm\textsuperscript{16/C15123}. Metastases to the ovaries may occur hematogenously, via direct extension, or by peritoneal spread.

Although the term Krukenberg tumor is now generally used as a synonym for any metastatic lesion to the ovary, it actually refers to a metastasis consisting of mucin-filled signet ring cells in a cellular stroma, usually from cancer of the gastric antrum. By strict definition only 30–40% of ovarian metastases are Krukenberg tumors. Up to 85% of ovarian metastases originate from primary colon and gastric cancers. Other common primary neoplasms include cancers of the breast, lung, and pancreas\textsuperscript{16–23}.

Most studies have shown that differentiation between metastatic and primary tumors on the basis of imaging findings alone is difficult. Clinical and imaging context may be helpful in some cases. Metastases typically are solid, bilateral, and strongly enhancing. Cystic and necrotic areas are common, such that lesions may be predominantly cystic and resemble primary ovarian cancer. The overlap of radiologic appearances between primary ovarian cancer and metastases to the ovary is substantial and confident imaging distinction may be difficult\textsuperscript{24–29}.

**Breast cancer primaries**

Patients with breast cancer and ovarian masses deserve special consideration because of the increasing prevalence and incidence of breast cancer and because the distinction between a primary ovarian cancer and metastatic breast cancer is critical. Patients with primary

\textbf{Figure 1} Incidental ovarian cysts (white arrows) are identified during a staging CT scan obtained on this 67-year-old woman with lung cancer. Note the calcified fibroid (black arrow) within the uterus.

Goldstein \textit{et al.}\textsuperscript{14} also found no instances in cancer in 42 postmenopausal women with simple cysts.

Kroon and Andolf\textsuperscript{13} found no instances of ovarian cancer in 83 postmenopausal women with small (<5 cm), completely anechoic, thin-walled ovarian cysts. Similarly
ovarian cancer require extensive staging laparotomy, whereas patients with breast metastases may be treated with less invasive surgery\cite{30,31,32,33}.

Breast cancer metastasizes (Fig. 2) to the ovaries via a hematogeneous route and ovarian involvement is nodular and infiltrating and often bilateral and solid pathologically. Hematogeneous metastases differ from Krukenberg tumors and metastases from the gastrointestinal tract, which involve the ovary secondarily through peritoneal dissemination. Infiltrating lobular carcinoma is the most common histopathologic type of breast cancer to metastasize to the ovaries as well as the peritoneum and gastrointestinal tract\cite{33,34}.

Women with BRCA mutations have a 56–87% lifetime risk of developing breast cancer and a 27–44% lifetime risk of having ovarian cancer. Patients with Lynch syndrome (hereditary non-polyposis colon cancer) are also significantly more likely to develop ovarian cancer\cite{31}. In addition, metastatic ovarian disease is found at post-mortem in 50% of patients dying of breast cancer and 25% of oophorectomies performed for metastatic breast cancer\cite{17,18}. All patients with breast cancer have a two-fold increased risk of developing primary ovarian cancer\cite{30}.

Hann et al.\cite{33} found that in 54 patients with breast cancer and adnexal masses discovered on cross-sectional imaging, 74% were benign and 26% were malignant. Half of the patients with malignant masses had primary ovarian cancer and the other half had metastatic breast cancer to the ovaries. All breast metastases to the ovary were bilateral solid masses pathologically and occurred in patients with stage IV breast cancer. Eleven of 14 ovaries with breast metastases were solid on ultrasound; the remaining three had cystic components or coexistent benign ovarian cysts. Ovarian metastases were found in 58% of all patients with stage IV disease. Stage IV breast cancer and bilateral solid adnexal masses were the best predictors of ovarian metastases. Four of the seven patients with primary ovarian carcinoma had bilateral ovarian tumors and seven of 11 ovarian carcinomas were predominantly cystic sonographically. The incidence of ovarian malignancy was similar for premenopausal and postmenopausal patients with breast cancer.

In the group of patients with malignant ovarian neoplasms, primary ovarian carcinoma and breast metastases to the ovary were seen with equal frequency.

Weiner et al.\cite{35} prospectively screened 600 patients with breast cancer using endovaginal ultrasound to assess the frequency of synchronous ovarian neoplasms. Of 11 patients who consented to surgery, seven had benign ovarian abnormalities, three had primary ovarian carcinoma and one had metastases to the ovaries. Thus, the frequency of synchronous primary ovarian cancer in patients with breast cancer who underwent screening was 0.5%.

Curtin et al.\cite{30} found that 50% of patients with breast cancer and a preoperative diagnosis of a pelvic mass who underwent oophorectomy had malignant disease and the ratio of primary ovarian carcinoma to metastases was 3 to 1.

In a large study of 644 patients with early stage breast cancer with long-term follow-up, Rosen et al.\cite{22} observed that metachronous, non-mammary malignancies developed in 13% of patients and that ovarian carcinoma was the most common primary malignancy after treatment for breast cancer.

Gastrointestinal tract primaries

Most non-genital tract primary cancers that metastasize to the ovaries are of gastrointestinal origin\cite{28,29}. Some studies suggest that gastric cancer\cite{19,36} is the most frequent source of metastases but others have reported that colon cancer is the most common primary neoplasm\cite{37–39}.

In a study comparing the CT findings of ovarian metastases from colon cancer and primary ovarian cancer, Choi et al.\cite{29} found that a smooth tumor margin and a more cystic nature were found to be strong predictors of ovarian metastases from colon cancer.

Choi et al.\cite{28} in a different study, found that mass characteristics, laterality, enhancement patterns of the solid portion of the tumor, and lesion size were useful parameters for differentiating ovarian metastasis from primary gastric versus primary colon cancer. Ovarian metastases from gastric cancer (Fig. 3) were solid or mainly solid in appearance in 69.2% of cases, whereas only 9.7% of ovarian metastases from the colon cancer displayed this pattern. These differences may arise from differences in histologic type. In their series, signet ring cancers were the most common type of gastric cancer and they tended to be solid or mostly solid on CT. Tubular adenocarcinomas and mucinous adenocarcinomas were the most common types of colon cancers to metastasize to the ovary in their patient population and they displayed a predominantly cystic appearance.

**Figure 2** Axial CT in a 47-year-old woman with breast cancer shows metastases to the greater omentum (arrows), ovaries (o) and uterus (U).
Choi et al. [28] also found that colon cancer metastases to the ovary tended to be larger than those from gastric cancer (9.1 cm vs 5.6 cm, respectively) and the more cystic nature may be partly responsible for the larger size of colon cancer metastases. Dense enhancement of the solid portion of the tumor was found in 46.2% of gastric cancer metastases but only 10.3% of colon cancers. In addition, patients with ovarian metastases from colon cancer were older than those with metastatic gastric cancer. Nearly 38% of gastric metastases were found in patients who were younger than 40 years old but no ovarian metastases from colon cancer were found in these younger patients. This finding may relate to an increasing percentage of young patients in Asia with gastric cancer and the more aggressive nature of this disease in the young.

Other primary neoplasms

Lymphomatous involvement of the ovary manifests as large bilateral minimally enhancing solid ovarian masses with homogeneously low T1 and mildly high T2 signal intensity without necrosis, hemorrhage or calcification. Systemic lymphomatosis may suggest the diagnosis [40].

Leukemic involvement is rare but the ovaries may be a site of relapse [41]. Metastatic melanoma is uncommon or rarely arises in situ because of malignant transformation of melanocytes in a mature cystic teratoma [42]. Pancreatic adenocarcinoma may metastasize to the ovaries and the appearance can mimic primary mucinous ovarian cancer [43].

Mass characterization

A variety of excellent imaging tests including ultrasound, multi-detector computed tomography (MDCT), MRI, and positron emission tomography (PET)/CT are available for the detection, characterization, and staging of benign and malignant ovarian masses. Ultrasound is the primary imaging test used to evaluate gynecologic pathology in patients with known or suspected disease on the basis of clinical history or gynecologic examination. Contrast-enhanced MRI has been shown to be highly accurate in the detection and characterization of adnexal masses but is generally reserved for problem solving after inconclusive ultrasound findings. CT is the imaging test of choice in staging and preoperative planning of ovarian cancer, however CT is generally not used to characterize ovarian masses. Practically, however, a woman’s first imaging test is often a pelvic CT performed in conjunction with an abdominal CT. This is especially true in the following groups of patients: the diagnosis, staging, and follow-up of patients with extra-gynecologic malignancies; patients who present to the emergency department with acute or chronic abdominal pain; and those with suspected gastrointestinal and genitourinary sources of pathology. Newer studies show that CT is highly accurate in characterizing adnexal masses as malignant. Indeed, the increasing accuracy of MDCT may obviate the need for routine performance of ultrasound or MRI, thus providing a cost-effective and time-saving approach for the management of women with adnexal masses [44-49].

Benign imaging features

The most common benign ovarian masses are simple cysts, hemorrhagic cysts, corpus luteal cysts, endometriomas, and dermoids. Because they are so common and
usually have a typical appearance, the diagnosis can usually be made with confidence\[^{10}\].

**Simple cysts**

Follicular cysts are the most common well-defined adnexal masses. During the menstrual cycle, the dominant follicle can measure up to 19–25 mm in size and a surge in luteinizing hormone triggers ovulation and the conversion of the dominant follicle into the corpus luteum. The term cyst should be reserved for structures larger than 2.5–3 cm. Cysts less than this size are more appropriately termed follicles\[^{10}\].

Sonographically, follicular cysts are unilocular structures that contain anechoic fluid and posterior through transmission of sound. Because of the presence of mobile proteinaceous fluid or cellular debris, fine, low level echoes may be seen. These lesions typically involute and resolve within one to two menstrual cycles and follow-up ultrasound should be performed during the follicular phase (days 5–7 of a subsequent menstrual cycle)\[^{10}\].

When large, these cysts may persist beyond several menstrual cycles and there may be overlap between follicular cysts and cystadenomas. The latter appear larger and more persistent, and are typically found in older women.

On CT, follicular cysts appear as a sharply marginated round simple fluid collections (attenuation <20 HU) with thin non-enhancing wall. When found incidentally on CT, follow-up ultrasound should be done after one to two menstrual cycles.

Functional cysts have a low to intermediate signal intensity on T1-weighted images and very high signal intensity on T2-weighted images. The cyst walls are well defined and usually have decreased signal intensity on T2-weighted images and may show various degrees of contrast enhancement\[^{46,48}\].

**Hemorrhagic cysts**

Hemorrhage into a follicular cyst or corpus luteum may cause acute pain. The sonographic appearance depends upon the age of the hemorrhage and the degree of clot formation. Typically there is a complex mass with internal echoes and some degree of posterior through transmission. Although initially anechoic, fine low level echoes producing a find lace-like reticular pattern for the first 24 h is characteristic. With clot retraction, triangular or curvilinear echogenic regions may be seen at the cyst wall. Fluid debris levels may be found as the clot liquefies\[^{10}\].

On CT, hemorrhagic cysts before rupture appear unilocular with a density ranging from 25 to 100 HU. These are usually unilocular and have crenulated walls that may enhance. When these lesions are identified on CT they do not need immediate sonographic evaluation unless there is a significant hemoperitoneum. Follow-up ultrasound following several menstrual cycles will determine whether the cyst has resolved\[^{46}\].

On MR, hemorrhagic cysts have intermediate to high signal intensity on both T1- and T2-weighted sequences and can be confused with endometriomas\[^{46,48}\].

**Corpus luteal cysts**

Follicular cysts are usually asymptomatic and will reabsorb within 4–8 weeks. With failure of involution, corpus luteal cysts can develop and enlarge from 4 to 10 cm. The walls of luteal cysts are thicker than those of follicular cysts on both ultrasound and CT and may be irregular because of a recent rupture or an adherent blood clot.

Corpus luteal cysts on MR tend to have a thicker and more regular wall and show intense mural enhancement. They may also show hemorrhage, which alters their signal intensity. Because these features can also be seen in malignancy, follow-up imaging may be needed\[^{46,48,50,51}\].

**Endometriomas**

Nearly 10% of premenopausal women have endometriosis and as much as 80% of ectopic endometrial tissue is found in the ovaries. Sonographically these lesions have a uniform low level echogenicity or a ground glass appearance as a result of repeated episodes of cyclic bleeding. The appearance on CT is variable ranging from solid to a cystic heterogeneous adnexal mass. The margins may be irregular as a result of repeated episodes of bleeding. Because both CT and ultrasound have a low specificity for the diagnosis of endometriomas, follow-up imaging is needed to differentiate this lesion from hemorrhagic ovarian cysts as well as primary and secondary ovarian malignancies.

On T1-weighted MR images, endometriomas have high signal intensity that is more conspicuous on fat-suppressed images. On T2-weighted images, these lesions demonstrate a gradient of low signal intensity (shading) that results from repeated bleeding and the build-up of blood pool products, which shortens T2. Functional and hemorrhagic cysts do not demonstrate this profound T2 shortening. Variable mural enhancement following gadolinium administration may be seen\[^{46,48,52}\].

**Dermoids**

One of the most complex ovarian masses is the cystic teratoma or dermoid cyst. These lesions comprise 15% of all ovarian tumors and most of them are mature and benign, with 99% showing a cystic component. Approximately 85% are detected between the ages of 20 and 50 years. These lesions are slow growing and typically asymptomatic. Roughly 3% of these lesions will eventually undergo torsion.

The sonographic appearance is variable depending upon the presence of fat, teeth, hair and fluid within
the lesion. Predominantly solid and predominantly cystic lesions have been described with equal frequency. Rokitansky nodules or dermoid plugs, which often contain hair or calcification, are diagnostically specific. The most common appearance is a mass with highly reflective irregular solid components within a fluid-containing adnexal mass. The strong reflective echo pattern is caused by hair and sebum within the dermoid. Acoustic shadowing from the hair may totally obscure the back wall of a large dermoid, hence the term tip-of-the-iceberg sign [10].

CT demonstrable macroscopic fat is found in more than 90% of ovarian dermoids. Mural nodules, calcification, teeth are also depicted on MDCT [42,47]. On MR, dermoids typically show fat within the lesion, fat-fluid or fluid-fluid levels, layering debris, low signal intensity calcification (usually teeth), and Rokitansky nodules. Standard T1- and T2-weighted images can usually establish the diagnosis, however fat-saturated or opposed phase T1-weighted images improve diagnostic confidence [48,52].

Malignant imaging features

The risk of malignancy in a premenopausal woman with an indeterminate ovarian mass is approximately 8.75%. In postmenopausal women, this risk increases to 32.4%. Findings that suggest malignancy include: (1) size >4 cm; (2) solid mass or large solid component; (3) mural thickness >3 mm; (4) septal thickening >3 mm or the presence of nodularity or vegetations; (5) multilocularity (>3 locules); (6) necrosis; (7) involvement of adjacent organs or the pelvic side walls; (8) peritoneal, mesenteric, or omental disease; (9) ascites; (10) adenopathy; (11) bilateral; (12) inhomogeneity. These features are usually well depicted on contrast-enhanced CT, however gadolinium-enhanced MRI is slightly superior to CT and gray scale and Doppler ultrasound in characterizing adnexal masses. Contrast administration is important because it may reveal solid elements not appreciated on the non-contrast images [53–55].

Recommendations

Patel et al. [56] have suggested important guidelines for reimaging the female pelvis after a gynecologic mass is incidentally discovered on CT. Reimaging refers to further evaluation of a CT finding to be performed immediately or within a few days to characterize a mass or other CT findings. Follow-up imaging, in contradistinction, refers to ultrasound used to assess a potential change in CT findings because of the effects of time (involution or growth), typically performed 6 weeks to 6 months later. These authors indicate five situations in which reimaging of the pelvis is not indicated:

1. Gynecologic structures have normal CT appearance. Traditionally little attention has been given to gynecologic structures on CT so that normal structures often misinterpreted as abnormal. On contrast-enhanced CT, the myometrium typically enhances faster and more than the cervix with this differential enhancement persisting into the venous phase. When the uterus is tilted or retroverted, the less robustly enhancing cervix may be mistaken for a hypodense mass. The vaginal fornices often extend deep into the pelvis and can have a bulbous shape along the superior margin. In patients with previous hysterectomy, the vaginal cuff may be misinterpreted as a mass especially in patients who have had a supracervical hysterectomy or when there is a vaginal cuff cyst. When the uterus lies in its typical position superior to the bladder, it is often imaged coronally, which may simulate endometrial thickening on axial CT. In normal premenopausal women, the ovarian parenchyma enhances less than the myometrium on delayed contrast-enhanced scan. This differential enhancement is further highlighted by the existence of cysts and follicles. The normal corpus luteum shows a ring of hyperenhancement corresponding to the so-called ring of fire described on Doppler ultrasound.

2. CT demonstrates a gynecologic abnormality that has a reasonably limited differential diagnosis and temporal observation is needed to distinguish between possibilities. CT often will identify a smooth-walled ovarian cyst with fluid attenuation that does not require reimaging but may need follow-up imaging. Contemporaneous reimaging with ultrasound is of little benefit even if the cyst is hyperdense if a well-defined cyst wall is clearly depicted on CT. The differential diagnosis for this lesions would be an acute hemorrhagic cyst (high prevalence), endometrioma (medium to low prevalence), and benign neoplasm containing hemorrhage or high-density mucin (uncommon).

3. CT demonstrated a gynecologic malignancy with a characteristic diagnostic appearance.

4. CT finding has a clearly established origin within the myometrium.

5. Ultrasound is unable to yield additional useful information regarding the abnormality detected on CT.

Uterine and endometrial incidentalomas

Fibroids

Fibroids are present in nearly 40% of women older than 40 years and are particularly common in the black population. Most of these lesions are asymptomatic and found as incidental findings but can cause pain, bleeding, infertility, and clinically palpable masses. Their appearance is sufficiently characteristic on cross-sectional imaging to make the correct diagnosis [57–59].
Sonographically, fibroids have a variable appearance. A hypoechoic solid concentric mass is seen in about one-third of cases. When dense fibrosis prevails within the tumor, the mass can be quite echogenic. Hypoechoic regions may develop as a result of degeneration or atrophy\[10\].

On CT, fibroids may be hypo-, iso- or hyperdense relative to normal uterus but they often exhibit coarse peripheral or central calcification. The enhancement pattern (Fig. 4) is variable. On MR most fibroids have relatively homogeneous low T2 signal intensity because of collagenous material. Cellular fibroids contain less collagen and have intermediate T2 signal intensity. Degenerating fibroids often have a bright T2 signal intensity and often contain thick septations or mural nodules. After the administration of gadolinium, these lesions may robustly but inhomogeneously enhance\[48\].

Endometrial incidentalomas

The endometrium demonstrates a wide spectrum of normal and pathologic appearances on cross-sectional imaging in patients with cancer. Asymptomatic patients may develop polyps, submucosal fibroids, endometrial hyperplasia, or endometrial adenocarcinoma, tamoxifen-associated changes, intrauterine fluid collections, and endometrial adhesions. The appearance of the endometrium is related to multiple factors, including the patient’s age, stage in the menstrual cycle, and whether there is ongoing hormonal replacement or tamoxifen therapy\[60–63\].

In women of menstrual age, the endometrium typically reaches a maximum thickness of up to 15–18 mm during the midsecretory phase. The appearances of normal and abnormal endometrium, such as seen in endometrial hyperplasia, may overlap. Cyclic ovarian changes parallel the endometrial changes in the follicular and luteal phases\[60–63\].

When evaluating the postmenopausal appearance of the endometrium (Fig. 5), the presence of vaginal bleeding, the presence of hormonal replacement therapy, a history of breast cancer, ovarian cancer, the BRCA gene, polycystic ovarian disease, and other risk factors should also be incorporated into the degree of clinical suspicion. The normal postmenopausal endometrium should appear thin, homogeneous, and echogenic. A double-layer thickness of less than 5 mm without focal thickening excludes significant disease and is consistent with atrophy. Homogeneous, smooth endometria measuring 5 mm or less are considered within the normal range with or without hormonal replacement therapy. The endometrium in a patient undergoing hormonal replacement therapy may vary up to 3 mm if cyclic estrogen and progestin therapy is being used. The endometrium appears thickest before progestin exposure and thinnest after the progestin phase. A patient undergoing unopposed estrogen therapy with endometrial thickening exceeding 8 mm should be considered for biopsy, whereas patients receiving progesterone in addition to estrogen can be rescanned at the beginning or end of the following cycle to determine if there has been a change in endometrial thickness\[60–63\].

Endometrial polyps (Fig. 6) are a common cause of postmenopausal bleeding and are most frequently seen in patients receiving tamoxifen. Although endometrial polyps may be visualized at transvaginal ultrasound as non-specific endometrial thickening, they are frequently identified as focal masses within the endometrial canal\[60–63\].

Endometrial hyperplasia

Endometrial hyperplasia (see Fig. 5) is an abnormal proliferation of endometrial stroma and glands and represents a spectrum of endometrial changes ranging from glandular atypia to frank neoplasia. A definitive diagnosis can be made only with biopsy, and imaging cannot reliably allow differentiation between hyperplasia and carcinoma. Nearly one-third of endometrial carcinoma is believed to be preceded by hyperplasia\[60–63\].

Intrauterine fluid collections

Although a trace amount of fluid within the postmenopausal endometrial canal may be normal, any significant fluid collection requires careful evaluation of the uterus and adnexal structures for masses. Intrauterine fluid collections are associated with both endometrial and
cervical cancers and an obstructing tumor must be excluded even when cervical stenosis has been identified clinically.

Endometrial fluid collections are often seen in premenopausal patients and are most commonly associated with menstruation, early uterine pregnancy, or the pseudogestational sac in an ectopic pregnancy. Other benign causes of obstruction leading to intrauterine fluid production include polyps, infection, and submucosal fibroids.

**Effects of tamoxifen**

Tamoxifen is commonly administered to breast cancer patients and has a proestrogenic effect on the endometrium that causes it to appear thickened, irregular, and cystic on ultrasound. The degree of endometrial thickening corresponds to the duration of tamoxifen therapy. This medication is associated with an increased prevalence of endometrial hyperplasia, polyps, and carcinoma and nearly 50% of patients receiving this medication may develop an endometrial lesion within 6–36 months. Accordingly, any patient who develops bleeding while taking tamoxifen requires evaluation.

**Incidental vascular abnormalities**

Diffuse or focal dilation of pelvic vessels is occasionally observed in patients with abdominal and pelvic neoplasms on MDCT, MR and ultrasound. There are two major mechanisms that may account for this vascular dilation. First there may be increased blood flow through collateral vessels associated with a neoplasm such as uterine fibroids, gestational trophoblastic disease, ovarian solid tumors, and mesenteric tumors, all of which may be associated with an increased number of draining vessels. The assessment of these draining vessels can assist in identification of tumor origin. Second, dilated collateral channels may result from benign or malignant venous obstruction or stenosis, portal hypertension, and left renal venous compression between the aorta and superior mesenteric artery leading to dilation of the left gonadal vein.
Pelvic congestion syndrome occurs in 10% of women and results from dilated, tortuous, and congested veins produced by retrograde flow through incompetent valves in the ovarian veins (Fig. 7). Within this group of patients, up to 60% may develop pelvic congestion syndrome. In general, pelvic congestion syndrome is considered an under diagnosed cause of chronic pelvic pain because of the non-specificity of the observations made with conventional imaging[65].

Incidentally found prominent lymph nodes

Lymph node evaluation is an essential part of the staging and surveillance of the oncology patient[66]. With the advent of CT in the mid 1970s came the ability to non-invasively image the pelvic lymph nodes and multiple studies have described normal size criteria for these nodes. These size measurements were established with older scanners, with non-helical technology. With refinements in MDCT it has become possible not only to image enlarged lymph nodes but also normal-sized lymph nodes particularly in the sigmoid mesocolon and small bowel mesentery. However, little has been published describing the size criteria of normal mesenteric and mesocolic lymph nodes as these nodes were not reliably detected before the advent of MDCT.

Figure 6 Incidental robustly enhancing endometrial polyp (arrows) is depicted on these axial (A) and coronal (B) discovered during a staging CT in this 58-year-old menopausal woman with gastric cancer. She had no pelvic symptoms or bleeding.

Figure 7 Engorged parametrial vessels (white arrows) are incidentally depicted on these axial (A) and coronal (B) images obtained during a staging CT in this 38-year-old woman with breast cancer without pelvic symptoms. Black arrows, bilateral ovarian cysts.
The incidental finding of prominent pelvic lymph nodes, particularly on coronal reformatted images, has become more common as a result of several factors: faster scanning times and bolus tracking allow for much easier detection of lymph nodes; thin collimation improves spatial resolution and the depiction of these lymph nodes; there is less volume averaging of these nodes with adjacent bowel and blood vessels; and scrolling on picture archiving and communication systems permits better differentiation between lymph nodes and blood vessels\[67\].

In a study of 120 trauma patients, Lucey et al.\[68\] found >5 mesenteric lymph nodes with the mean size of the largest lymph node measuring 5 mm in 47% of patients. They concluded that mesenteric and mesocolic lymph nodes <5 mm in an otherwise healthy population require no further evaluation. Borderline sized pelvic lymph nodes in the oncologic patient, however, are less easily dismissed. Knowledge of the regional nodal spread of each tumor is essential in determining their significance.

Once a pelvic lymph node is visualized, there are a number of imaging features that are modestly helpful in determining whether a lymph node is involved by tumor. The shape of a pelvic lymph node can also be helpful diagnostically. A normal lymph node has an oblong kidney bean-shaped morphology with a fatty hilum. It has a smooth contour with the exception of the hilum, which is perforated by small lymphatic channels and blood vessels. Tumor-filled nodes are more likely to have an irregular border and tend to be more round than oblong with a short to long axis of 0.81 compared with 0.57 for benign nodes\[69,70\].

The internal architecture of a lymph node is also a useful diagnostic feature. Preservation of a normal fatty hilum indicates a more benign node, whereas central necrosis can be seen with metastatic involvement. On MR, heterogeneous signal intensity on T2-weighted images is more likely to be found in malignant lymph nodes\[70\].

There is a lack of consensus regarding the normal size limit in the diagnosis of pelvic tumor nodal metastases. There is a need for specific size criteria for different types of cancers. Although in general there is a threshold of 8 mm short-axis diameter for pelvic lymph nodes and 10 mm for abdominal retroperitoneal lymph nodes, it has been found that 8 mm is suspicious for metastases in testicular cancer and that nearly 60% of nodes involved by rectal cancer are <5 mm in diameter\[69\].

The size of benign inguinal lymph nodes (Fig. 8) is quite variable, with 15 mm short-axis diameter considered the upper limit of normal. This figure is useful in patients with tumors that do not usually drain to inguinal nodes, but it is insensitive in tumors that do. In one series of vulvar cancers, 8 mm short-axis diameter had a sensitivity of only 52% for metastatic node involvement\[66\]. This emphasizes the importance of knowledge of the regional nodal drainage pathways of individual tumors and the limitations of using size criteria.

**Conclusions**

Incidental ovarian and uterine masses, dilated pelvic vessels, and prominent lymph nodes will inevitably be uncovered with increasing frequency as technical improvements in cross-sectional imaging develop.
The pretest probability of clinically relevant disease is low but of uncertain magnitude in patients with malignancy. It may become necessary to slide down the receiver operating curve somewhat to decrease the false-positive fraction and avoid overcalling incidental pelvic lesions that could have a negative effect on overall patient care.

References

[1] Pickhardt PJ, Kim DH, Meiners RJ, et al. Colorectal and extracolonic cancers detected at screening CT colonography in 10286 asymptomatic adults. Radiology 2010; 255: 83–8. doi:10.1148/ radiol.09090939. PMID:20308446.

[2] Lumberaras B, Donat L, Hernandez-Aguado I. Incidental findings in imaging diagnostic tests: a systematic review. Br J Radiol 2010; 83: 276–89. doi:10.1259/bjr/98067945. PMID:20335439.

[3] Flicker MS, Tsoukas AT, Hazra A, Dachman AH. Economic impact of extracolonic findings at computed tomographic colonography. J Comput Assist Tomogr 2008; 32: 497–503. doi:10.1097/RCT.0b013e1816290291. PMID:18664832.

[4] Solomon R, Whiteley WN, Warlow C. Screening using whole-body magnetic resonance imaging scanning: who wants an MRI? Br J Radiol 2007; 80: 284–92. doi:10.1259/bjr/50066770. PMID:17038411.

[5] Rosen PP, Groshen S, Kinne DW, Hellman S. Nonmammary malignant neoplasms in patients with stage I and stage II breast carcinoma. Am J Clin Oncol 1989; 12: 169–74.

[6] Moore RG, Chung M, Granai CO, et al. Incidence of metastasis to the ovaries from nongynecologic primary tumors. Gynecol Oncol 2004; 93: 87–91. doi:10.1016/j.ygyno.2003.12.039. PMID:15047218.

[7] Chang WC, Meux MD, Yeh BM, et al. CT and MRI of adnexal masses in patients with primary nonovarian malignancy. AJR 2006; 186: 1039–45. doi:10.2214/AJR.04.0997. PMID:16554576.

[8] Kim SH, Kim WH, Park KJ, et al. CT and MR findings of Krukenberg tumors: comparison with primary ovarian tumors. J Comput Assist Tomogr 1998; 20: 393–8. doi:10.1097/00004728-199605000-00011. PMID:8626898.

[9] Ha HK, Back SY, Kim SH, et al. Krukenberg’s tumor of the ovary: MR imaging features. AJR 1994; 164: 1435–9.

[10] Choi HJ, Lee JH, Kang S, et al. Contrast-enhanced CT for differentiation of ovarian metastasis from gastrointestinal tract cancer: stomach cancer versus colon cancer. AJR 2006; 187: 741–5. doi:10.2214/AJR.05.0944. PMID:16928939.

[11] Choi HJ, Lee JH, Kim YH, et al. Computed tomography findings of ovarian metastases from colon cancer: comparison with primary malignant ovarian tumors. J Comput Assist Tomogr 2005; 29: 69–73.

[12] Brown DL, Doublet PM, Miller FH, et al. Benign and malignant ovarian masses: selection of the most discriminating gray-scale and Doppler sonographic features. Radiology 1998; 208: 103–10.

[13] Curtin JP, Barakat RR, Hoskins WJ. Ovarian disease in women with breast cancer. Obstet Gynecol 1994; 84: 449–52.

[14] Struwing JP, Hartge P, Wacholder S, et al. The risk of cancer associated with specific mutations of BRCA1 and BRCA2 among Ashkenazi Jews. N Engl J Med 1997; 336: 1401–8. doi:10.1056/NEJM199705153362001. PMID:9145676.

[15] Lamovec J, Bracko M. Metastatic pattern of infiltrating lobular carcinoma of the breast: an autopsy study. J Surg Oncol 1994; 48: 28–33. doi:10.1002/jso.2930480106. PMID:1653879.

[16] Hann LE, Lui DM, Shi W, et al. Adnexal masses in women with breast cancer: US findings with clinical and histopathologic correlation. Radiology 2000; 216: 242–7.

[17] Gore RM, Meyers MA. Pathways of abdominal and pelvic disease spread. In: Gore RM, Levine MS, editors. Textbook of gastrointestinal radiology. 3rd ed. Philadelphia, PA: Saunders; 2008, p. 2099–118.

[18] Weiner Z, Beck D, Shiteiner M, et al. Screening for ovarian cancer in women with breast cancer with transvaginal sonography and color flow imaging. J Ultrasound Med 1993; 12: 387–93.

[19] Hale RW. Krukenberg tumor of the ovary: a review of 81 records. Obstet Gynecol 1968; 32: 221–5.

[20] Webb MJ, Decker DG, Hussey E. Cancer metastatic to the ovary: factors influencing survival. Obstet Gynecol 1975; 45: 391–6.

[21] Holtz F, Hart WR. Krukenberg tumors of the ovary. Cancer 1982; 50: 2438–47. doi:10.1002/1097-0142(19821201)50:11<2438::AID-CNCR2820501132>3.0.CO;2-X.

[22] Kim SH, Kim WH, Park KJ, et al. CT and MR findings of Krukenberg tumors: comparison with primary ovarian tumors. J Comput Assist Tomogr 1996; 20: 393–8. doi:10.1097/00004728-199605000-00013. PMID:8626898.
Kalish GM, Patel MD, Gunn MLD, Dubinsky TJ. Computed tomography and magnetic resonance features of gynecologic abnormalities in women presenting with acute or chronic abdominal pain. Ultrasound Q 2007; 23: 167–75. doi:10.1097/RUQ.0b013e31815202df. PMid:17805165.

Osborn BM, Robboy SJ. Lymphoma or leukemia as ovarian tumors: an analysis of 42 cases. Cancer 1983; 52: 1933–43. doi:10.1002/1097-0142(19831115)52:10<933::AID-CNCR2820521026>3.0.CO;2-8.

Koyama T, Mikami Y, Saga T, et al. Secondary ovarian tumors: spectrum of CT and MR features with pathologic correlation. Abdom Imaging 2007; 32: 784–95. doi:10.1007/s00261-007-9186-4. PMid:17318680.

Young RH, Hart WR. Metastases from carcinomas of the pancreas simulating primary mucinous tumors of the ovary: report of seven cases. Am J Surg Pathol 1989; 13: 748–56. doi:10.1097/00000478-198909000-00004. PMid:2764222.

Tsili AC, Dalkalitis N, Paraskevaidis E, Tsampoulas K. Multi-detector CT features of benign adnexal lesions. Acad Radiol 2010; 17: 31–8. doi:10.1016/j.acra.2009.06.014. PMid:19734064.

Tsili AC, Tsampoulas C, Charisiadi A, et al. Adnexal masses: accuracy of detection and differentiation with multidetector computed tomography. Gynecol Oncol 2008; 110: 22–31. doi:10.1016/j.ygyno.2008.03.022. PMid:18486202.

Tsili AC, Tsampoulas C, Argyropoulou M. Comparative evaluation of multidetector CT and MR imaging in the differentiation of adnexal masses. Eur Radiol 2008; 1049–57.

Hricak H, Chen M, Coakley FV, et al. Complex adnexal masses: detection and characterization with MR-imaging multivariate analysis. Radiology 2000; 214: 39–46.

Hamm B, Huch-Kubic RA, Fleige B. MR imaging and CT of the female pelvis: radiologic-pathologic correlation. Eur Radiol 1999; 9: 3–15. doi:10.1007/s003300050620. PMid:9933373.

Zhang J, Mironov S, Hricak H, et al. Characterization of adnexal masses using feature analysis at contrast-enhanced helical computed tomography. J Comput Assist Tomogr 2008; 32: 533–40. doi:10.1097/RCT.0b013e3181568890. PMid:18664838.

Potter AW, Chandrasekhar CA. US and CT evaluation of acute pelvic pain of gynecologic origin in nonpregnant premenopausal patients. Radiographics 2008; 28: 1645–59. doi:10.1148/rg.286085504. PMid:18936027.

Ewa Kuligowska E, Deeds L, Lu K. Pelvic pain: overlooked and underdiagnosed gynecologic conditions. Radiographics 2005; 25: 3–20. doi:10.1148/rg.251045411. PMid:15653583.

Szklaruk J, Tam J, Apaydin V, et al. Imaging of pelvic congestion syndrome using transabdominal and transvaginal sonography. AJR 2004; 182: 683–8.

Morgan FR, Szklaruk J. Learning the nodal stations in the abdomen. Br J Radiol 2007; 80: 841–8. doi:10.1259/bjr/64292252. PMid:17959923.

Moore RG, DePasquale SE, Steinhoff MM, et al. Sentinel node identification and the ability to detect metastatic tumor to inguinal lymph nodes in squamous cell cancer of the vulva. Gynecol Oncol 2003; 89: 475–9. doi:10.1016/S0090-8258(03)00130-6.

Lucey BC, Stuhlfaut J, Soto JA. Measuring lymph nodes seen at imaging: causes and consequences. Radiographics 2005; 25: 351–65. doi:10.1148/rg.252045108. PMid:15798054.

Lucey BC, Stuhlfaut JW, Soto JA. Measuring lymph nodes: detection and significance on MDCT. AJR 2005; 184: 41–4.

McMahon CJ, Rofsky, Pedrosa I. Lymphatic metastases from pelvic tumors: anatomic classification, characterization, and staging. Radiology 2010; 254: 31–47. doi:10.1148/0.15148.2541090361. PMid:20032141.