check

Independent learning program for GPs

Unit 495 June 2013

Imaging

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Imaging

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The five domains of general practice
Communication skills and the patient–doctor relationship
Applied professional knowledge and skills
Population health and the context of general practice
Professional and ethical role
Organisational and legal dimensions

RACGP

This unit of *check* on radiological imaging looks at the most up-to-date and current techniques used for the diagnosis of common problems in general practice. Advances in technology mean that we now have imaging modalities that are less invasive, limit patients' exposure to ionising radiation and have better visualisation. Another benefit of new imaging modalities is that some can be used for therapeutic interventions and may be less invasive than traditional methods.

We would like to thank the authors, who are all specialists at MIA Radiology, a member of I-MED Radiology Network, for providing a wealth of information on current imaging techniques for this unit of *check*.

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The learning objectives of this unit are to:

- develop increased confidence in using appropriate imaging techniques to investigate breast pathology
- assess postmenopausal bleeding as well as identify risk factors for endometrial carcinoma
- identify the red flags in patients presenting with headache
- list the imaging modalities used for different brain pathologies
- use evidence-based guidelines to determine the probability of a patient having a pulmonary embolism
- discuss the different types of non-traumatic knee pain presenting in a young adolescent
- list the causes, investigation and treatment of greater trochanteric pain sydrome
- develop improved competence in the diagnosis and management of shoulder pain.

We hope this edition of *check* will be helpful in selecting the most appropriate investigation for your patients.

Kind regards

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GUIDE TO ABBREVIATIONS AND ACRONYMS IN THIS UNIT OF *CHECK*

AC	acromioclavicular	ELISA	enzyme linked immunosorbent assay	OA	osteoarthritis
AP	anterior posterior	FNA	fine needle aspiration	PD	proton density
BIH	benign intracranial hypertension	GTPS	greater trochanteric pain syndrome	PE	pulmonary embolism
BMI	body mass index	IIH	idiopathic intracranial hypertension	PIOPED	Prospective Investigation of Pulmonary Embolism Diagnosis
BP	blood pressure	ITB	iliotibial band	SE	stress echocardiogram/ echocardiography
CAD	coronary artery disease	LV	left ventricle	SS	supraspinatus
CC	craniocaudal	MBS	Medicare Benefits Schedule	UOQ	upper outer quadrant
CSF	cerebrospinal fluid	MLO	mediolateral oblique	V/Q	ventilation/perfusion
CT	computed tomography	MPI	myocardial perfusion imaging		
CTPA	CT pulmonary angiography	MRI	magnetic resonance imaging		
CVST	cerebral venous sinus thrombosis	MRV	magnetic resonance venogram		
ECG	electrocardiogram	NSAID	non-steroidal anti-inflammatory drug		

CASE 1

JANE IS WORRIED ABOUT BREAST CANCER

Jane, aged 36 years, presents to you having discovered a thickening with tenderness in her right breast on self-examination. She has not had any previous breast imaging. Jane's mother died of breast cancer aged 52 years, and Jane's older sister had treatment for breast cancer aged 40 years and is currently alive and well. Jane has a son and a daughter, and has no current health issues. She is not on any medication.

When you examine Jane you find symmetrical areas of thickening in the upper outer quadrant (UOQ) of both breasts, with no discrete lumps. There is no lymphadenopathy in the axillae. General examination reveals no abnormalities.

QUESTION 1

What is the most appropriate initial set of investigations?

QUESTION 2

Jane asks about going to BreastScreen. What do you recommend?

QUESTION 3

Jane is concerned about the radiation dose from a mammogram. What do you tell her?

FURTHER INFORMATION

Jane agrees to have a mammogram and ultrasound. The mammogram shows breast density of >75% with an area of subtle distortion in the left UOQ, but no other abnormality (see *Figure 1*). On ultrasound, the area of concern within both lateral breasts corresponds with normal dense breast tissue, and there are scattered simple cysts within both breasts (category 2). There are two further small, irregular hypoechoic masses more laterally within the left axillary tail, each measuring about 5–6 mm in diameter and 2 cm apart (category 4). There is also a left axillary lymph node with prominent cortical thickness of 3.1 mm (see *Figure 2*). Core biopsy of the two irregular breast masses and fine needle aspiration (FNA) of the axillary node are recommended.

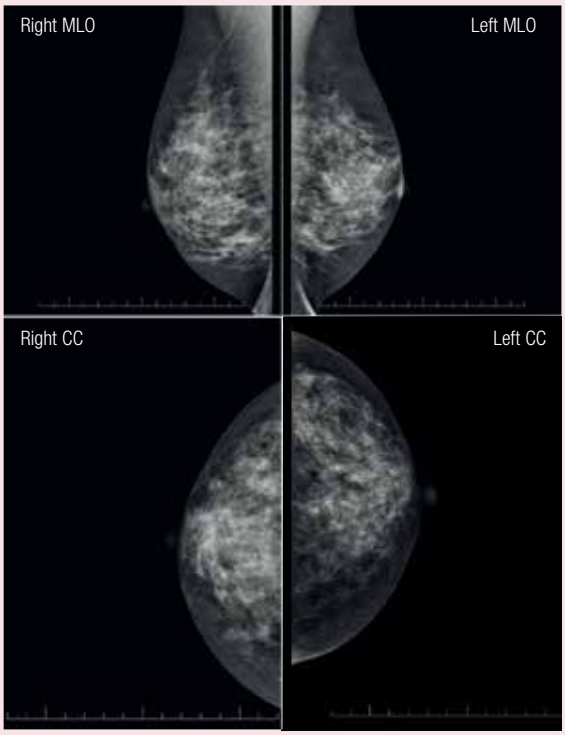


Figure 1. Bilateral mammogram showing dense fibroglandular tissue and subtle architectural distortion in the axillary tail on the left mediolateral oblique (MLO) projection. The craniocaudal (CC) view is also shown.

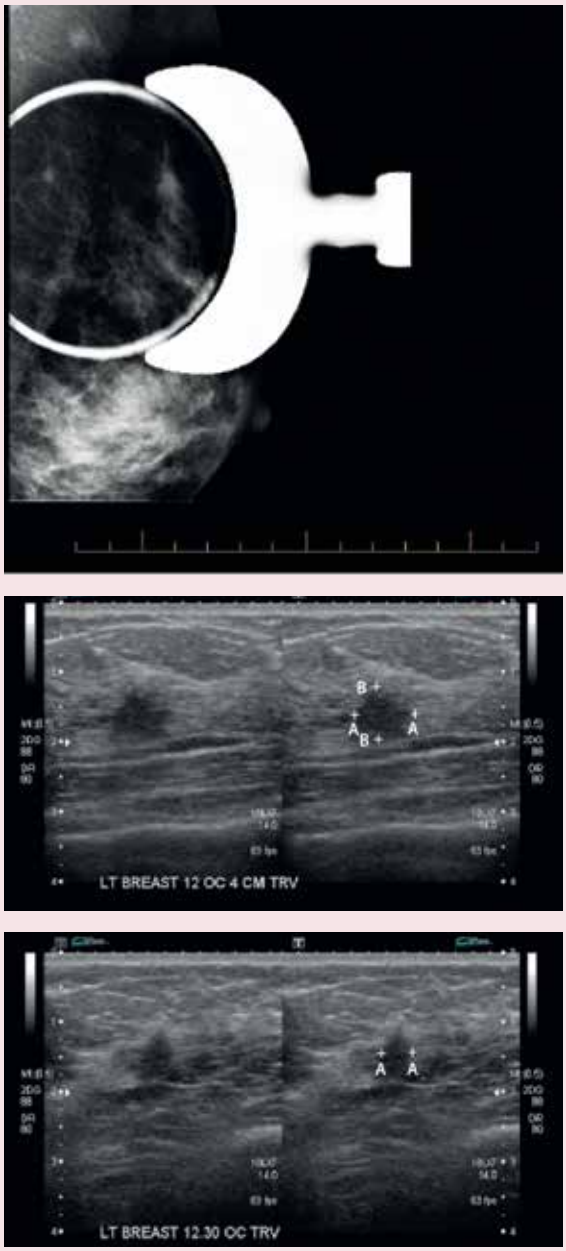


Figure 2. Work-up mammographic view showing two small spiculate masses. In the bottom and middle images, ultrasound also demonstrates two small, ill-defined hypoechoic masses with suspicious features.

QUESTION 4

What is the relevance of the categories 1–5 in breast imaging synoptic reports?

QUESTION 5

What is the relevance of breast density as stated in the mammogram report?

FURTHER INFORMATION

You refer Jane to a breast surgeon and she subsequently undergoes a breast core biopsy and axillary node FNA, both under ultrasound guidance, at a local radiology practice.

Both breast masses are invasive lobular cancers, but the axillary node is normal.

Further imaging is performed and CT and bone scans are clear of metastasis.

Jane elects to undergo bilateral mastectomy and reconstruction because of her strong family history of breast cancer and the imaging findings. Following the surgery she will take anti-oestrogen therapy but will not require any other adjuvant treatment.

QUESTION 6

Jane has a younger sister, Lily, aged 30 years. What action would you advise her to take regarding her breast health?

CASE 1 ANSWERS

ANSWER 1

Jane should have a diagnostic bilateral mammogram and ultrasound. If Jane had been younger than 35 years, an ultrasound would have been the first investigation, and a mammogram would have been performed when the suspicious masses were seen in the left breast. Women younger than 35 years are more likely to have dense breasts, therefore it is less likely that abnormalities will be detected on mammogram. When there is a breast symptom, a targeted breast ultrasound should always be performed.[1]

ANSWER 2

BreastScreen is a screening program and is not an appropriate place to investigate a breast symptom. A screening program assumes no symptoms. There is increased wait time for results and biopsy, and an ultrasound is only performed if the woman is recalled for assessment of a mammographic abnormality.

You tell Jane that she needs both mammogram and ultrasound, and that this is better undertaken at a diagnostic imaging facility.

ANSWER 3

Digital mammography requires an extremely low dose of radiation to acquire good quality images. The average radiation dose for two images of each breast (four images in total) is around 0.7 mSv, or the equivalent of approximately 4 months of environmental background radiation. For a woman younger than 40 years, this suggests a lifetime additional risk of cancer of between 1:10 000 and 1:100 000 per examination. This compares with the general risk of one in three of developing any cancer in a woman's lifetime.[2]

ANSWER 4

A standardised, management-based classification system for breast imaging findings will help to prevent interpretation errors and should improve the appropriate management of women with breast symptoms.[3]

- Category 1: no significant imaging abnormality (does not preclude biopsy of any clinically suspicious area)
- Category 2: benign findings (does not preclude biopsy of any clinically suspicious area)
- Category 3: indeterminate/equivocal findings. Requires further investigation (e.g. FNA/core biopsy). Management should be based on the outcomes of a triple test (clinical examination, imaging and biopsy or FNA). There may be a limited role for early follow-up
- Category 4: findings suspicious of malignancy. Requires further investigation. May require excisional biopsy
- Category 5: findings are consistent with malignancy. Requires further investigation (e.g. biopsy).

ANSWER 5

Breast density relates to the proportion of fibroglandular tissue within the breast, which varies considerably among women. In young women, breast tissue is highly glandular, dense and looks white on a normal mammogram. As women age, the glandular tissue generally becomes involuted and is replaced by fatty tissue. As cancers and other breast masses are white on a mammogram, the greater the background density, the lower the sensitivity for cancer detection on mammogram alone. Ultrasound is therefore of increasing importance to aid in the detection of cancer in denser breast tissue.[1]

ANSWER 6

Lily is considered at high risk of developing breast cancer because she has more than two primary relatives (mother and two sisters) who have developed the disease. Her risk of developing breast cancer is 3–6 times the normal female population, with a 25–50% lifetime risk of developing breast cancer. Lily should be referred to a cancer specialist or family cancer clinic for risk assessment, possible genetic testing and management plan. Ongoing surveillance strategies may include regular clinical breast examination, breast imaging with mammography, MRI or ultrasound and consideration of ovarian cancer risk. Individualised surveillance program may include regular clinical breast examination, and annual breast imaging with mammography, MRI or ultrasound.[4]

CASE 2

BARBARA HAS INTERMITTENT VAGINAL BLEEDING

Barbara, aged 70 years, presents with intermittent vaginal bleeding over the past 3 months. She has a history of breast cancer, which was treated with tamoxifen for 5 years. Barbara also has a history of obesity, hypertension and diabetes. She lives with her husband, John; they have no children.

QUESTION 1

What is the differential diagnosis for postmenopausal uterine bleeding?

QUESTION 2

What are the risk factors for endometrial carcinoma?

QUESTION 3

What imaging tests should you order for Barbara?

QUESTION 4

What is the risk of endometrial carcinoma for a patient on tamoxifen therapy?

CASE 2 ANSWERS

ANSWER 1

Causes of postmenopausal uterine bleeding include endometrial atrophy, benign endometrial hyperplasia, benign endometrial polyps, submucous uterine fibroids, oestrogen withdrawal and endometrial carcinoma. Of women presenting with postmenopausal bleeding, 10% will have an underlying malignancy such as endometrial carcinoma, atypical endometrial hyperplasia and cervical carcinoma.[5] Endometrial atrophy is the commonest cause of postmenopausal uterine bleeding.[6]

ANSWER 2

Risk factors for endometrial carcinoma include prolonged administration of unopposed oestrogen hormone replacement therapy (more than 5 years), tamoxifen use, hereditary non-polyposis colorectal carcinoma, obesity combined with diabetes, hypertension and exogenous or endogenous increased oestrogen.[7] Nulliparity, late menopause, early menarche and polycystic ovaries have also been implicated as risk factors. Pregnancy and the use of oral contraceptives reduce risk.

It is important to remember that 12% of endometrial cancer occurs in pre-menopausal women.[8]

In addition to being a risk factor for endometrial carcinoma, tamoxifen treatment is also associated with increased incidence of endometrial polyps and hyperplasia.[9]

ANSWER 3

Transvaginal ultrasound is the investigation of choice for women with postmenopausal uterine bleeding. An endometrial thickness of greater than 4 mm requires further investigation with an endometrial biopsy.[10] There is no benefit for ultrasound screening of asymptomatic women, even in high-risk groups such as women taking tamoxifen.[11]

Saline infusion sonohysterography is a transvaginal ultrasound performed after distension of the endometrial cavity with fluid via a fine cervical catheter. The hypoechoic fluid in the endometrial cavity provides contrast with the echogenic endometrial lining, which allows differentiation between diffuse endometrial pathology such as endometrial hyperplasia and focal pathology such as endometrial polyps or carcinoma (see *Figure 3*).[12]

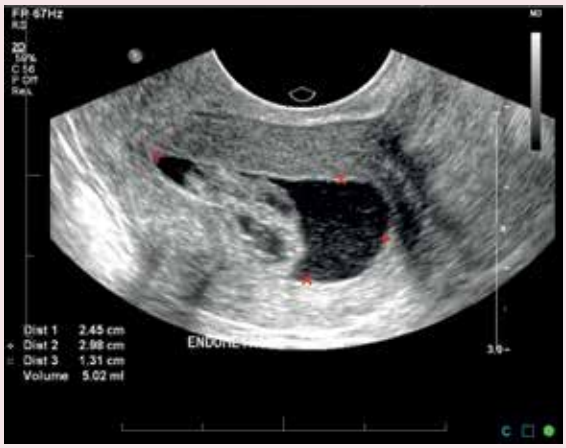


Figure 3. Sagittal ultrasound image of the uterus showing a polypoid endometrial carcinoma outlined by hypoechoic fluid (bright focus surrounded by black).

Endometrial hyperplasia affects the whole endometrium, causing diffuse thickening of greater than 5 mm. Endometrial polyps appear as focal endometrial thickening, occasionally with a vascular stalk being identified. Endometrial carcinoma may appear as endometrial thickening, a polypoid lesion or an irregular mass (see *Figures 4* and *5*).[13]

Ultrasound cannot reliably distinguish between benign endometrial hyperplasia and malignancy.[14]

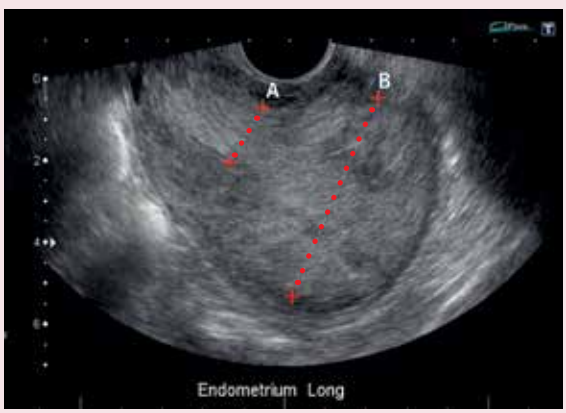


Figure 4. Sagittal ultrasound of the uterus showing irregular endometrial thickening caused by carcinoma. Thinning of the myometrium suggests myometrial invasion by carcinoma (A = lower uterine segment; B = uterine fundus).

Figure 5. Sagittal ultrasound image of the uterus showing a polypoid uterine carcinoma (outlined by callipers on the image).

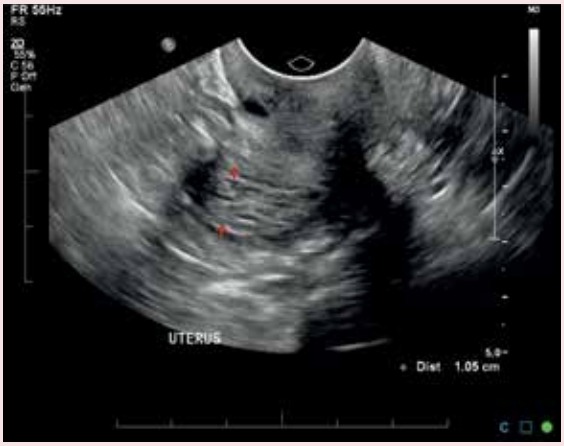


Figure 6. Sagittal ultrasound image of the uterus showing a thickened endometrium with multiple cystic spaces (between the callipers).

Endometrial biopsy is performed by a gynaecologist when there is diffuse endometrial thickening, focal endometrial thickening greater than 5 mm or an irregular endometrial mass. Definitive diagnosis of carcinoma can only be obtained by endometrial sampling, either with endometrial biopsy or dilatation and curettage.

Endometrial carcinoma spreads first by local invasion, followed by lymph node spread. Haematogenous spread is a late occurrence. CT scanning is of limited use in assessment of local invasion into the myometrium; it is used for assessment of distant metastases.[15]

MRI can be used to detect myometrial invasion, extra-uterine extension or pelvic lymphadenopathy.[16]

ANSWER 4

Tamoxifen is a selective oestrogen receptor modulator that has been used in the treatment of hormone receptor–positive breast cancer for over 30 years. It binds to oestrogen receptors, acting both as an oestrogen antagonist (in breast tissue) and an oestrogen agonist (in uterus and bone).[17] Tamoxifen increases the risk of developing endometrial carcinoma, with an increase in relative risk of 6.9 over 5 years of treatment.[18] Tamoxifen-associated carcinoma has an unfavourable prognosis compared with spontaneously developing endometrial carcinoma. Even taking this into account, the benefits of treatment with tamoxifen outweigh the risks, with the life expectancy of women with hormone receptor–positive breast cancer improved in those taking tamoxifen.[18]

Tamoxifen is also associated with an increased incidence of endometrial hyperplasia.[19] The endometrial hyperplasia associated with tamoxifen treatment typically has a cystic appearance on ultrasound (see *Figure 6*).

CASE 3

EMMA HAS WORSENING HEADACHES

Emma, aged 28 years, is an obese woman who presents to your clinic with worsening headaches over the past few months. Over the past week she has developed some mild blurred vision, but has not experienced any loss of vision. Emma has been married to Mark for 6 years. They have a daughter, Julie, aged 3 years.

QUESTION 1

In regard to headache presentations generally, what features on history and examination would raise a red flag and prompt early imaging investigation?

FURTHER INFORMATION

When you examine Emma, you find little of clinical significance except when you look at her fundus. Fundoscopy reveals papilloedema. You send Emma for an MRI, the results of which are shown in *Figures 7–9*.

Figure 7. Axial T2 fat suppressed orbits showing tortuous dilated optic nerve sheaths bulging into the back of the orbital globes – the imaging equivalent of papilloedema.

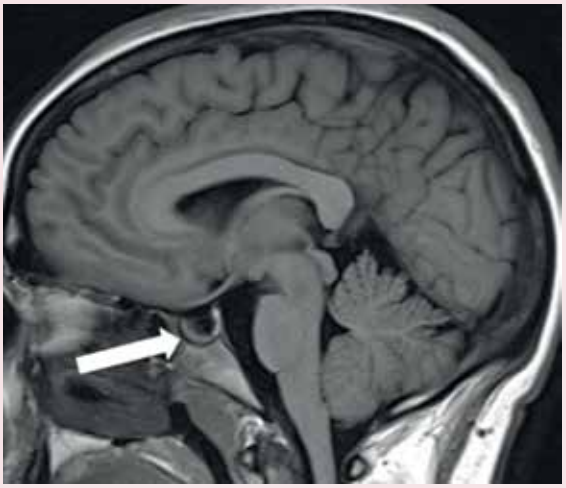


Figure 8. Sagittal T1 showing the pituitary gland flattened down, a feature known as 'partially empty sella'; this is a less specific sign of raised intracranial pressure.

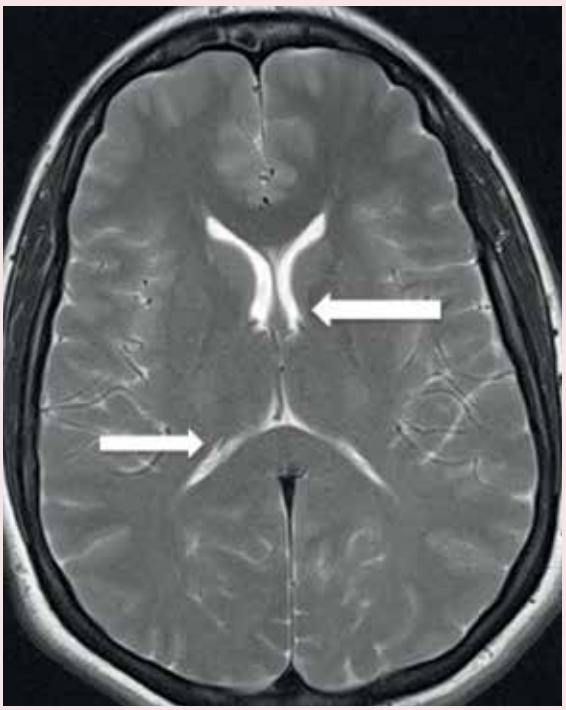


Figure 9. Axial T2 brain demonstrating normal or possibly reduced ventricle size, which excludes hydrocephalus as a cause of Emma's headaches.

QUESTION 2

What are the main differential diagnoses given these features?

QUESTION 3

How should you treat Emma?

QUESTION 4

What is the imaging investigation of choice for excluding cerebral venous sinus thrombosis (CVST)?

QUESTION 5

What imaging should be done in cases of suspected meningitis?

QUESTION 6

What initial imaging investigation is recommended for suspected giant cell arteritis?

CASE 3 ANSWERS

ANSWER 1

The features on history and examination that would raise red flags are outlined below.

- Headache type: thunderclap, different from usual migraine, positional or orthostatic headache or headache that is worsened by Valsalva, headache causing wakening, or headache that is progressively worsening
- Patient demographic: elderly, pregnant or postpartum, history of malignancy, trauma or immunocompromise
- Other signs: fever, neck stiffness, mental state alteration (personality or conscious state), neurological deficit, first episode of seizure, papilloedema
- Drug ingestion: anticoagulants, illicit drugs, immunosuppressants.[20,21]

ANSWER 2

There are two differential diagnoses for Emma's clinical and MRI findings.

1. Venous obstruction or CVST.
2. Idiopathic intracranial hypertension (IIH), which is also known as pseudotumour cerebri, and benign intracranial hypertension (BIH).

Emma's MRI shows dilated optic nerve sheaths with tortuosity bulging into the back of the orbital globes consistent with fundoscopic findings of papilloedema/raised intracranial pressure. There is an absence of venous obstruction/thrombosis or increased mass effect such as from brain oedema, hydrocephalus or tumour.

Thus, Emma's diagnosis is consistent with IIH, which is a diagnosis of exclusion.

Features of IIH include the following.

- Dural venous stenosis is a frequent finding and may be part of the underlying cause.
- It is most commonly seen in obese women of childbearing age. The other group that is affected is older men (usually non-obese), who are at twice the risk of vision loss.
- The incidence is approximately 1 per 100 000 per year; however, in women aged between 20 and 44 years who are 10% above ideal body weight, the incidence is 13 per 100 000 and in those 20% above ideal body weight, it is 19 per 100 000. Prevalence rates are higher, reflecting the chronic nature of the condition in many cases.[22]

ANSWER 3

Emma will need urgent initial assessment by an ophthalmologist to accurately determine whether she has significant alteration in her vision. As Emma has had only minimal blurred vision, her management is likely to consist of weight loss and medication such as acetazolamide to reduce cerebrospinal fluid (CSF) production.

Emma will need ongoing regular ophthalmology reviews. If her vision deteriorates, more aggressive measures such as shunt placement, venous sinus stenting or fenestration of the optic nerve sheaths may be indicated.

ANSWER 4

A combination of MRI and MR venogram (MRV) is the test of choice to exclude CVST for the following reasons. (See *Figures 10–12* for images of a patient with CVST with cerebral oedema secondary to venous hypertension.)

- MRI, in general, is more sensitive for detection of CVST at each stage after thrombosis; however, without the venographic component the diagnosis might not be excluded.
- While a CT scan is often the initial imaging choice by a GP or in an emergency department, non-contrast CT is only abnormal in approximately 30% of CVST cases.[23]
- Cerebral oedema as a complication of venous hypertension is much better appreciated on MRI than CT, and is often evident.
- MRV has the added benefits of not requiring contrast and of having no ionising radiation.
- If MRI is not available, venographic study using CT would be recommended.
- CVST accounts for 0.5–1% of strokes.[24]

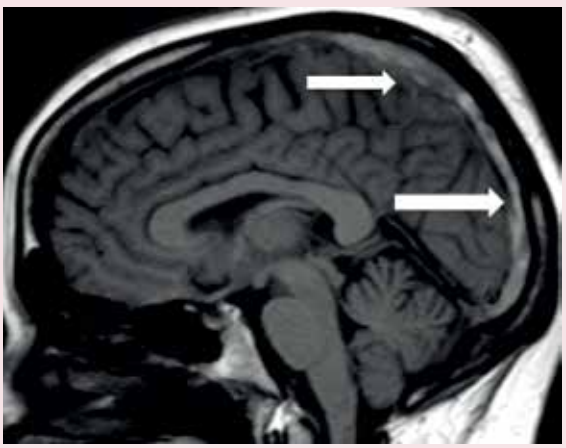


Figure 10. Sagittal T1 showing high signal within the superior sagittal sinus, which is confirmed on MRV in *Figure 15*.

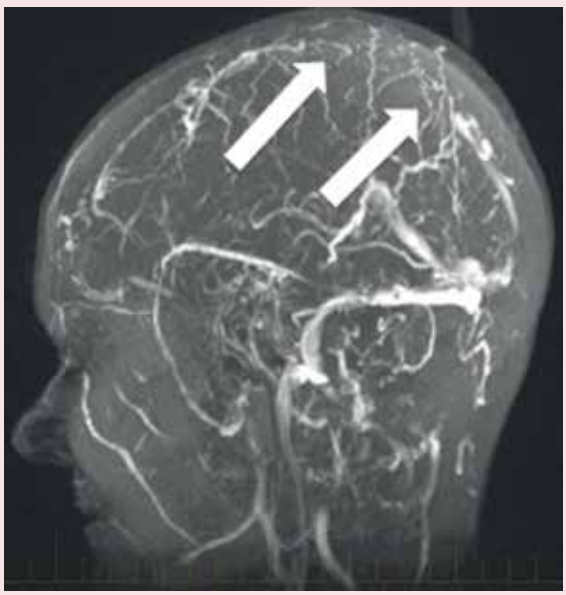


Figure 11. Sagittal MRV time of flight of venous sinus thrombosis with the thrombosed superior sagittal sinus seen as a defect/loss of continuity in the sinus (arrows).

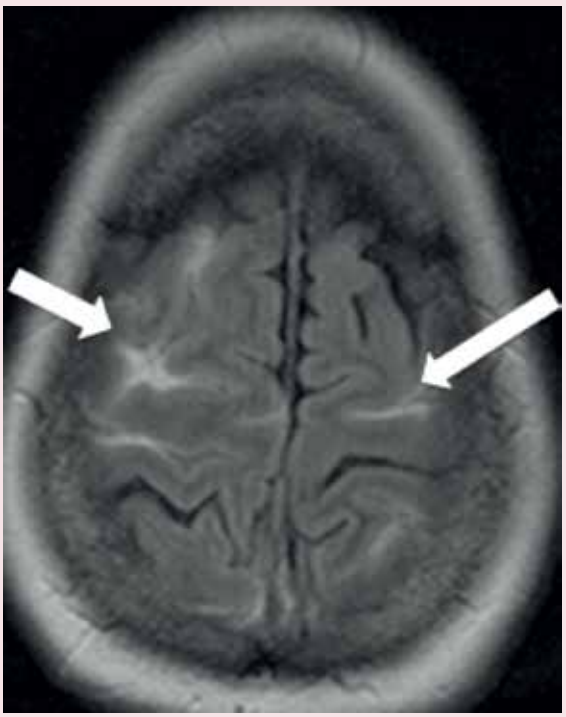


Figure 12. Axial flair – the bright areas represent brain oedema secondary to venous hypertension.

ANSWER 5

If meningitis is suspected, the taking of blood cultures and commencement of antibiotics should not be delayed by the use of imaging.[25]

Lumbar puncture should be deferred for 1 to 2 days in patients who are clinically very ill or likely to have raised intracranial pressure (e.g. altered conscious state, focal neurological deficit, papilloedema). Brain herniation from lumbar puncture may occur in the presence of a normal CT scan of the brain.[26]

MRI brain may be indicated (without delaying initial treatment) at some point if complications are suspected (e.g. CVST, intracranial abscess, immunocompromised patient).

ANSWER 6

Imaging is generally not indicated for patients with suspected giant cell arteritis. Diagnosis is based on a combination of clinical suspicion, inflammatory markers and temporal artery biopsy. Both MRI and ultrasound can be useful in equivocal or challenging cases, and can be used to help guide biopsy.

CASE 4

MATTHEW HAS A SORE KNEE

Matthew, aged 14 years, presents to your surgery with a 4-month history of pain in his left knee. He is very active and does some form of exercise on most days. He is a keen swimmer and plays water polo and cricket at school. He is otherwise well. He occasionally gets hay fever and had eczema as a child. He can't remember any injury to his knee. He has no history of knee pain prior to this episode. Matthew describes the pain as being made worse by activity. Occasionally the pain wakes him from sleep. He describes the pain as being 'all over' his knee and he is unable to pinpoint one discrete area of tenderness.

On examination, Matthew is afebrile. His BMI is 21. The skin over his left knee is normal and there is no discolouration. There is possibly some mild swelling anterior to the left tibial tuberosity, which is a little painful to palpation. Matthew tells you that the pain is not localised to just that spot and seems to be all over his knee. There is no mass palpable and his knee movements are slightly restricted.

QUESTION 1

What are the possible causes of Matthew's knee pain?

QUESTION 2

What investigations, if any, would you suggest? What are the advantages and disadvantages of the different imaging modalities?

QUESTION 3

What does the plain X-ray film in *Figure 13* show?

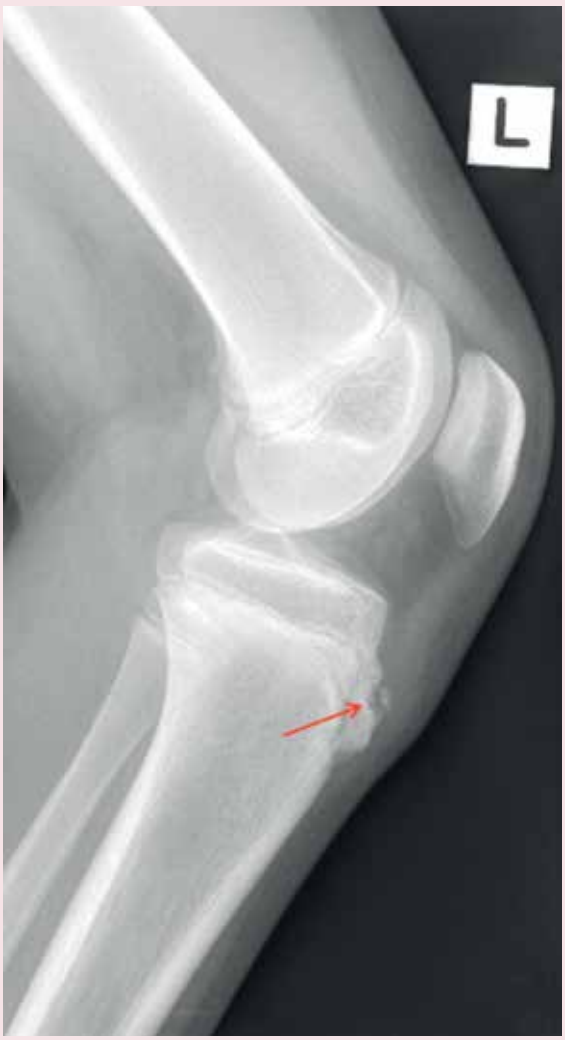


Figure 13. X-ray showing a lateral view of Matthew's left knee.

FURTHER INFORMATION

Matthew returns 6 months later with persistent, hard-to-localise knee pain.

QUESTION 4

What imaging, if any, would you recommend?

FURTHER INFORMATION

Matthew decreased his activity level as well as having an arthroscopic drilling procedure to stimulate healing. One year later his pain had resolved and a follow-up MRI (see *Figure 14*) showed almost complete healing.

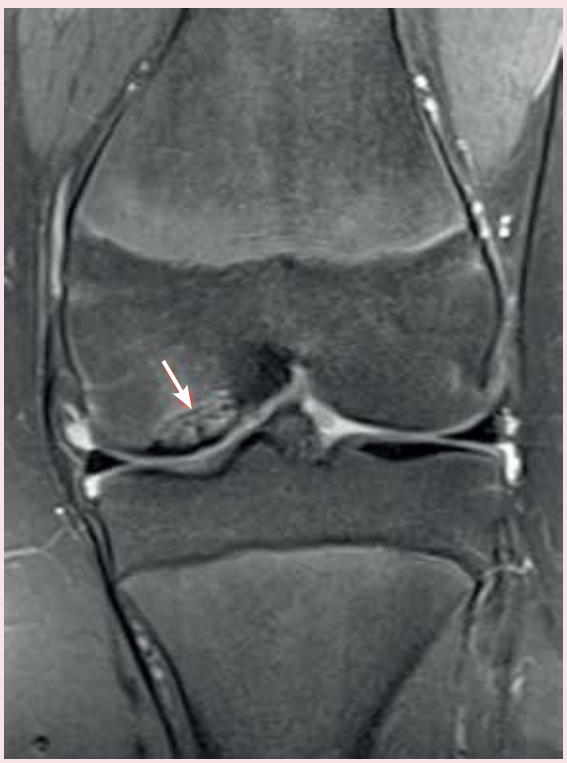


Figure 14. Resolution of osteochondral defect on MRI imaging.

CASE 4 ANSWERS

ANSWER 1

The causes of non-traumatic knee pain in a young adolescent are:[27]

- Osgood–Schlatter disease/Sinding–Larsen–Johansson disease
- osteochondral defect (osteochondritis dissecans)
- meniscal tear
- patellofemoral instability or maltracking
- bipartite patella
- synovitis – mostly post-viral
- aggressive causes such as osteomyelitis and osteosarcoma (rare).

ANSWER 2

Matthew warrants further investigation because he presents with a history of night pain and slight limitation of movement, which are not usually present in Osgood–Schlatter disease. While Osgood–Schlatter disease is the most common cause of knee pain in this age group it is important to exclude other pathology.

A plain X-ray film (anterior posterior (AP) and lateral) of the knee will provide bone detail and exclude tumour and fracture, but is unable to show causes of internal derangement of the knee.

Ultrasound does not have a role as it can only demonstrate a knee effusion. It cannot show other pathology within the knee, apart from further definition of a palpable mass if one is present.

CT is inappropriate as it only gives bone detail and does not show the muscles, tendons, ligaments, cartilage or menisci. It gives no detail of bone marrow oedema and exposes the patient to unnecessary radiation.

MRI will show articular cartilage, ligaments and tendons as well as bone detail, including the presence or absence of bone marrow oedema. It can be used for growth plate assessment and demonstrating synovial pathology within the joint, such as a knee joint effusion or synovial thickening.

ANSWER 3

The lateral view of the knee on plain X-ray in *Figure 13* shows some fragmentation of the tibial tuberosity, which is characteristic of Osgood–Schlatter disease.[28] Osgood–Schlatter disease is an osteochondrosis of the patellar tendon insertion onto the tibial tubercle. It affects patients aged 10–15 years, with up to 50% of cases being bilateral. It is likely to be secondary to chronic avulsive injury of the patellar tendon on the tibial tubercle. The chondro-osseous junction is considered the weakest component in the immature skeleton and injury often occurs here, rather than disrupting the patellar tendon itself.[27,29]

Osgood–Schlatter disease is a clinical diagnosis and doesn't require imaging unless there are unusual features such as night pain or limitation of movement. If MRI is performed it often shows a thickened patellar tendon, oedema in the fat surrounding the patellar tendon and also fragmentation of the tibial tuberosity.

Osgood–Schlatter disease is treated conservatively with advice to reduce high-level activities. It is a self-limiting condition that resolves spontaneously around the time of growth plate fusion.

ANSWER 4

The initial investigation is a repeat X-ray (see *Figure 15*) to assess bone detail and check for any changes. An irregularity of the medial femoral condyle is shown, probably an osteochondral defect.

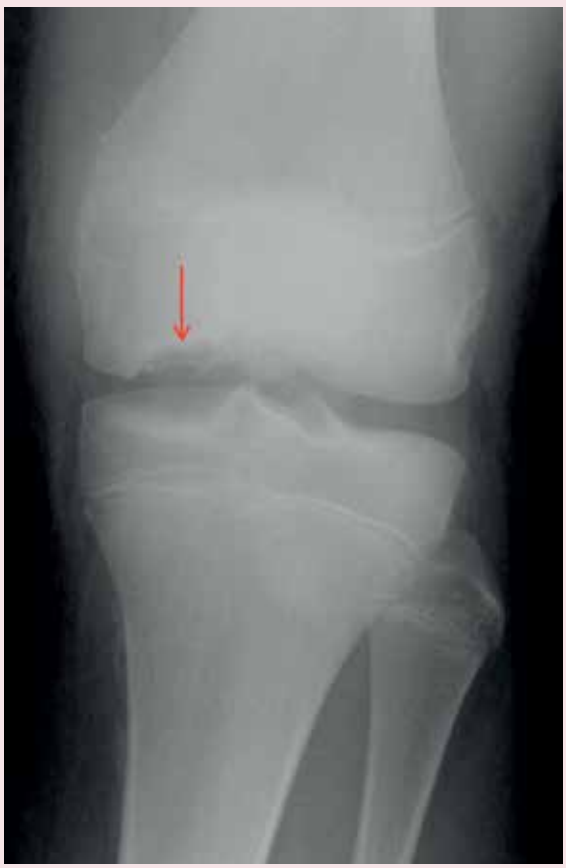


Figure 15. AP X-ray film of the knee demonstrates a lucency (arrow) in the medial femoral condyle that is consistent with an osteochondral defect.

MRI is now indicated as there are ongoing symptoms that are not consistent with Osgood–Schlatter disease.

MRI (*Figure 16*) shows a focus of abnormality of the medial femoral condyle with bone marrow oedema radiating away from it into the underlying bone. The overlying articular cartilage is intact, however the lesion has some findings of developing instability on MRI criteria.[30]

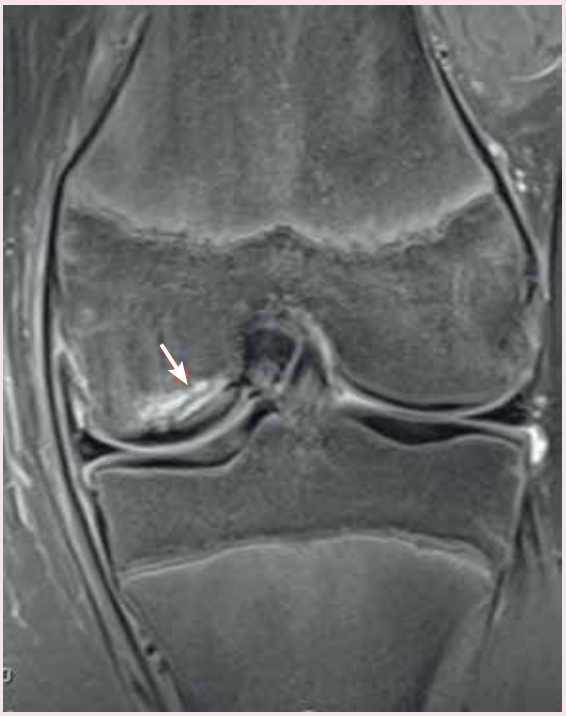


Figure 16. Coronal proton density (PD) MRI scan demonstrating that the plain X-ray film lucency in *Figure 14* corresponds to the bright focus within the medial femoral condyle in keeping with an ostechondral defect. There is a high signal cleft deep to the lesion and a few cysts (arrow), which are features of lesion instability.

Osteochondral defects occur most commonly in patients aged between 10 and 20 years and are more common in males. They often appear to be related to chronic traumatic injury, and less commonly to acute trauma.[31,32] Children with this condition often present with poorly localised knee pain that continues for more than a year. Typically, the pain increases with exercise.[33]

An osteochondral defect involves the lateral aspect of the medial femoral condyle in 75% of patients, and is bilateral in one-third of cases.[27] These subchondral bone defects or fragments may be only partially attached so they are unstable and prone to detachment, or they may be attached with fibrous tissue. MRI is useful in identifying lesion instability by showing a high signal interface between the defect and the femur.[30] A high T2 signal cleft deep to the lesion and cyst formation are used as signs of developing instability.[34] Lesion stability is important when deciding on treatment options, and this information cannot be obtained with a plain X-ray. Gadolinium enhanced studies have been used and can possibly differentiate between instability and stability,[35] but are not routinely used as usually the findings are clear on non-gadolinium studies.

Treatment of stable osteochondral lesions on MRI criteria includes an initial period of non-weight-bearing followed by a graded return to normal activity. Follow-up MRI and plain X-ray are used to monitor progress. The patient often has continued low-grade discomfort that eventually resolves. If the osteochondral defect is unstable there are a number of treatment options available. These include arthroscopic drilling to promote healing, in situ pinning of the bone fragment or chondrocyte reimplantation.[33,36]

CASE 5

SIMONE HAS CHEST PAIN

Simone, aged 21 years, is a bookkeeper who presents to your surgery with a sudden onset of chest pain when she breathes in. She tells you that she has been coughing up some blood. She also says that her right leg has been painful, swollen and mildly red for 1 week leading up to her current presentation. She lives at home with her parents and is currently not on any medication. She is allergic to penicillin. She recently travelled to the United States for a holiday.

On examination she is afebrile. Her BP is 110/70 mmHg, her heart rate is 80 beats per minute and regular, and her respiratory rate is 20 breaths per minute. Her right calf is slightly swollen with mild erythema and is tender to gentle palpation. Her chest is clear on auscultation. A pulse oximeter shows that she is mildly hypoxic with an oxygen saturation of 91% on room air.

QUESTION 1

What is the most likely diagnosis? What differential diagnoses need to be considered?

QUESTION 2

What are the risk factors for developing pulmonary embolism (PE) in a young patient? What importance should be placed on overall pre-test probability before performing imaging?

QUESTION 3

What is the most appropriate investigation for Simone?

QUESTION 4

Which tests would be the most appropriate if Simone told you that she was pregnant?

QUESTION 5

What are some newer imaging modalities in the diagnosis of PE?

CASE 5 ANSWERS

ANSWER 1

The most important diagnosis to exclude is PE. It is important to rapidly diagnose PE as it is associated with a high mortality rate. Of patients diagnosed with PE, 17.4% will die within 3 months of diagnosis[37] and delayed treatment may lead to significant morbidity as well as the development of secondary pulmonary hypertension.[38]

Other causes of pleuritic chest pain in a young adult include pneumothorax, pneumonia, pericarditis, as well as infective, inflammatory and musculoskeletal causes.

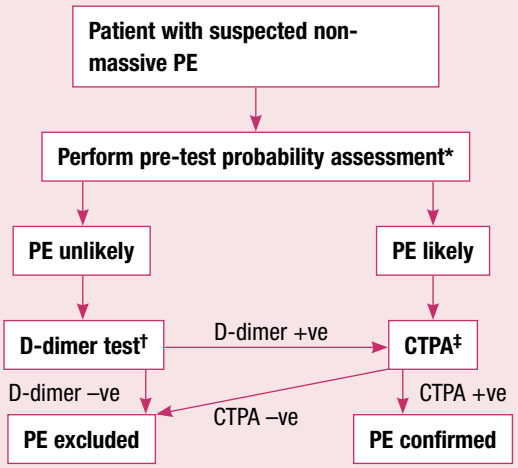
ANSWER 2

Risk factors for PE in a young patient include recent travel, pregnancy and use of the oral contraceptive pill. Other risk factors include recent surgery, malignancy and coagulation disorders.

Assessment of pre-test probability such as the Wells Pulmonary Embolism Score (see *Table 1*) in combination with biochemical testing with D-dimer (see *Figure 17*) is important in determining the most appropriate imaging pathway. The investigation of PE is based on recommendations from the Prospective Investigation of Pulmonary Embolism Diagnosis (PIOPED) II trial.[39]

Table 1. Simplifed Wells Pulmonary Embolism Score

Variable	Points
Clinical signs and symptoms of deep vein thrombosis (minimum of leg swelling and pain on palpation of the deep veins)	3.0
Alternative diagnosis less likely than pulmonary embolism	3.0
Heart rate >100 beats per minute	1.5
Immobilisation (>3 days) or surgery within the previous 4 weeks	1.5
Previous pulmonary embolism or deep vein thrombosis	1.5
Haemoptysis	1.0
Malignancy (receiving treatment, treated in last 6 months or palliative)	1.0
Clinical probability of pulmonary embolus unlikely: score ≤4 points Clinical probability of pulmonary embolus likely: score >4 points This table originally appeared in: McRae S. Pulmonary embolism. Aust Fam Physician 2010;39(7):462–6.	



* Assess pre-test probability according to simplifed Wells score (*Table 1*)

† Algorithm applies if D-dimer test used is a high sensitivity test (e.g. sensitivity >95%)

‡ Ventilation/Perfusion (V/Q) scanning can be used as an alternative imaging test in patients with renal impairment, or where there are concerns regarding radiation exposure (e.g. young women, particularly during pregnancy). If CT pulmonary angiography (CTPA) is technically inadequate, V/Q scanning can also be performed.

Figure 17. Algorithm for the diagnosis of PE. This chart orignally appeared in: McRae S. Pulmonary embolism. Aust Fam Physician 2010;39(7):462–6.

ANSWER 3

The most appropriate investigation for Simone depends on her pre-test probability, her clinical stability, imaging availability and potential risks of radiation exposure and/or iodinated contrast material.[39]

Current suggested imaging pathways are based on recommendations from the PIOPED II trial.[40] Following risk evaluation using the Wells or Geneva scoring system, a rapid enzyme linked immunosorbent assay (ELISA) D-dimer is performed. For patients with low or moderate pre-test probability with a negative D-dimer, no further investigation or treatment is necessary.[39] For patients with a positive D-dimer, further imaging investigation should be performed. In the high pre-test probability group no D-dimer testing needs to be performed – imaging investigation is suggested as first-line evaluation.[39]

The most easily assessable imaging investigations include chest X-ray, venous Doppler ultrasound, V/Q lung scan (see *Figure 18*) and CTPA (see *Figures 19*, *20* and *21*). Older tests such as catheter pulmonary angiography are invasive, associated with high radiation dose[39] and carry a risk of severe complications,[41] and are, therefore, not commonly performed.

Chest X-ray carries low sensitivity and specificity for the detection of PE, with the classic Westermark sign (oligaemia of the affected region of the lungs with decreased vessel diameter and density) seen in only 2% of cases.[42] Chest X-ray is useful in identifying other causes of pleuritic chest pain.

Venous Doppler ultrasound carries high sensitivity for the detection of symptomatic deep vein thrombosis,[43] but sensitivity is lower in the setting of PE for detection of above-knee thrombosis. The detection of a thrombus does not necessarily prove PE, and it is hence limited as a stand-alone test.[43]

The PIOPED II recommendations favour the use of CTPA over V/Q scanning for all pre-test probabilities where further investigation is required.[39] However, CTPA requires good renal function and the use of iodinated contrast material; in some situations V/Q scanning may be more appropriate.

CTPA should not be used with patients who have an allergy to iodinated contrast material and in those with impaired renal function. In these scenarios, venous Doppler ultrasound and V/Q scanning are recommended.

While the PIOPED II study recommends CTPA over V/Q scanning for women of reproductive age, the latter may be appropriate in patients suspected of having PE in the setting of a normal chest X-ray.[44] This may, therefore, be an appropriate investigation for Simone as it is associated with a lower radiation dose.[39]

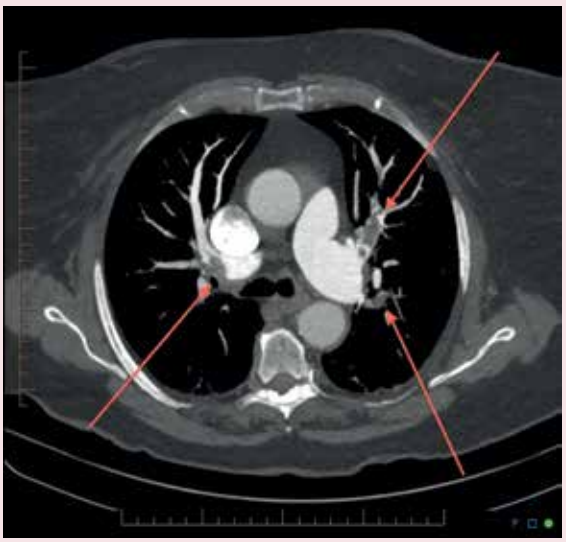


Figure 19. Axial CTPA demonstrates multiple large filling defects in major proximal pulmonary arterial branches, in keeping with acute PE.

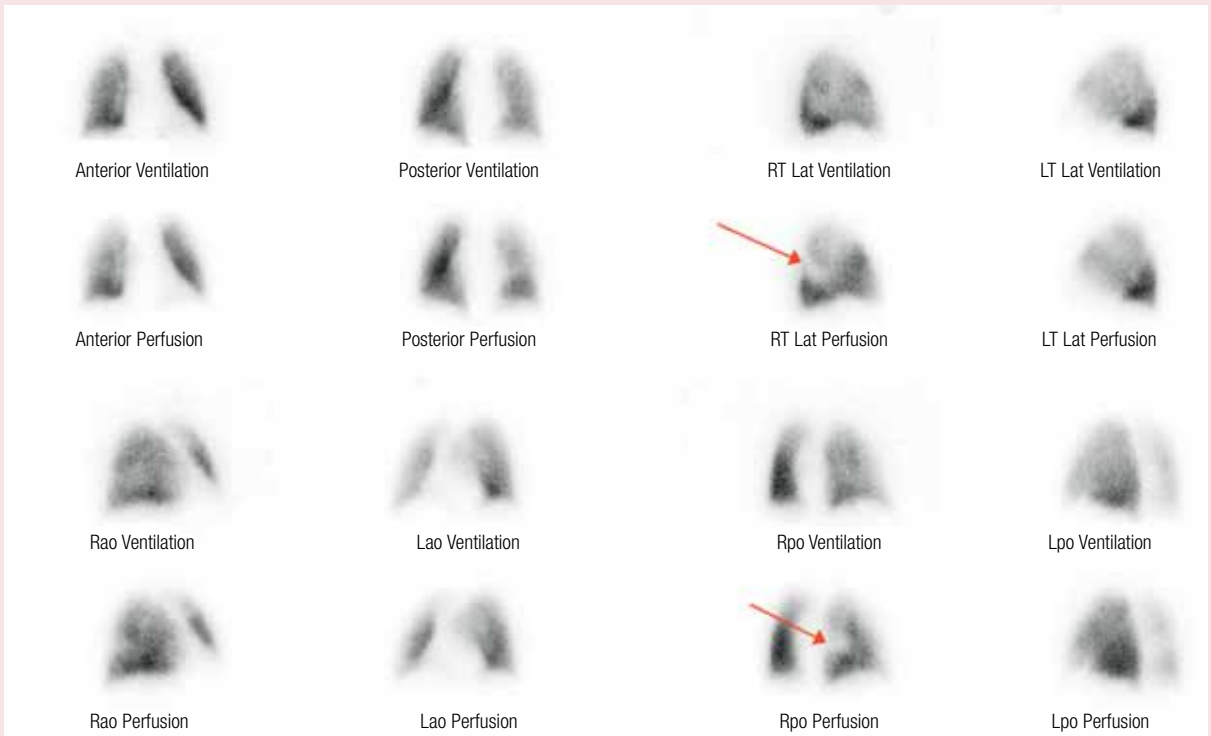


Figure 18. V/Q scanning demonstrates multiple unmatched segmental defects in keeping with PE. The largest perfusion defect is in the right mid zone.

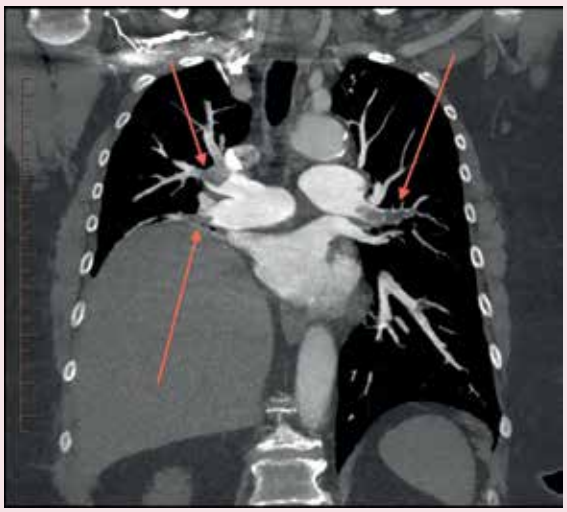


Figure 20. Coronal CTPA once again demonstrates multiple large filling defects in keeping with large PE.

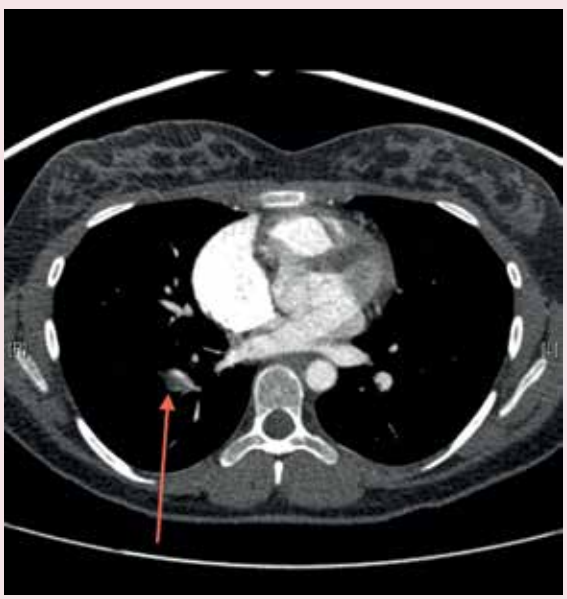


Figure 21. CTPA demonstrates a filling defect to the right lower lobe in keeping with acute PE.

ANSWER 4

The PIOPED II study recommends an initial D-dimer test for pregnant patients. If this is positive then a venous Doppler ultrasound is recommended.[39] If further imaging is required, V/Q is usually preferred over CTPA[39] because it results in a lower level of whole-body radiation. The overall foetal dose may be similar.[45] This recommendation is controversial, as a recent study showed that foetal dose may be lower with CTPA than with V/Q scanning.[46]

It is worth performing a D-dimer test during pregnancy[47] as this may avoid further investigation if it is negative. D-dimer is usually normal in the first trimester and rises in the second and third trimesters before falling to normal levels in the postpartum period.[48]

Venous Doppler ultrasound is a useful initial test due to the absence of ionising radiation. In the pregnant patient, ultrasound will detect thrombus in 13–15% of those suspected of having PE[49,50] and in 29% who are later found to have PE.

ANSWER 5

Newer imaging modalities, such as dual energy CT (*Figure 22)* and MR pulmonary angiography, are emerging and may play a greater role in the detection of PE in the future.

In addition to standard CTPA images, iodine perfusion maps can be created with dual energy CT, thereby increasing the ability to detect smaller subsegmental PE,[51] which has been difficult with standard CTPA.

Gadolinium enhanced MR angiography is an emerging technique evaluated in the recent PIOPED III trial, where it showed promising results. The advantage of using MR over CTPA or V/Q scanning is the absence of ionising radiation. MR may play a greater role in the diagnosis of PE in the future.

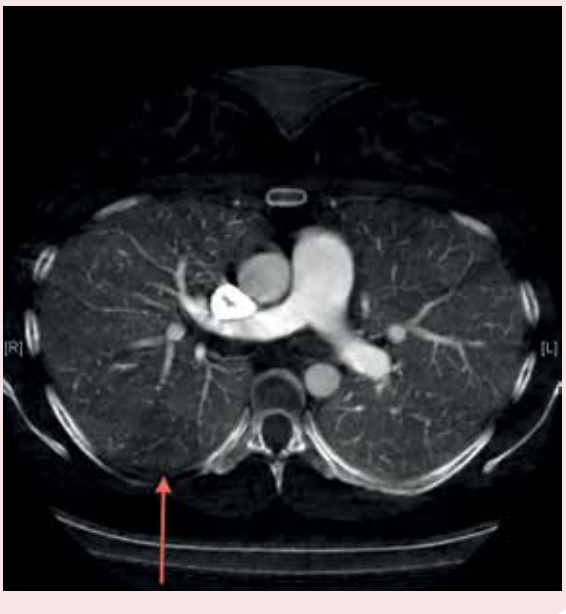


Figure 22. Approximately 2 years later, a follow-up to the CTPA shown in *Figure 21* was undertaken using dual energy CT with iodine perfusion mapping, showing a persistent wedge-shaped perfusion defect. This represents an area of hypoperfusion that may represent segmental infarction.

CASE 6

ERIN HAS PAIN IN HER HIP

Erin, aged 60 years, presents to your surgery with pain in her right hip. She has had the pain for several months now and she has noticed that it seems to be worse when she lies on her right side. She had a rotator cuff injury in her left shoulder 2 years ago that resolved with physiotherapy. She works as a receptionist at a nearby hospital and is usually fit and well. She walks on most days, is a non-smoker and only has the occasional glass of wine. On examination she has a full range of hip movement but she has focal tenderness over the greater trochanter. She has a good range of movement in her back and straight leg raising is normal on both sides. You make a clinical diagnosis of greater trochanteric pain syndrome (GTPS).

QUESTION 1

List three possible causes of GTPS.

QUESTION 2

Which patient group is most commonly affected by GTPS?

QUESTION 3

Which imaging modalities are most useful in the investigation of GTPS?

FURTHER INFORMATION

You refer Erin for an MRI to determine the cause of her chronic right hip pain. The results of her MRI are shown in *Figures 23* and *24*. You confirm the diagnosis of GTPS.

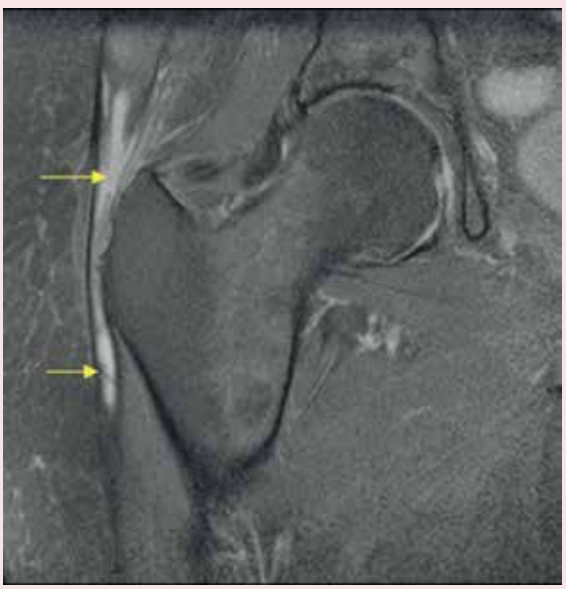


Figure 23. Coronal PD weighted MRI image with fat saturation of the right hip demonstrating bright fluid distending the greater trochanteric bursa (arrows).

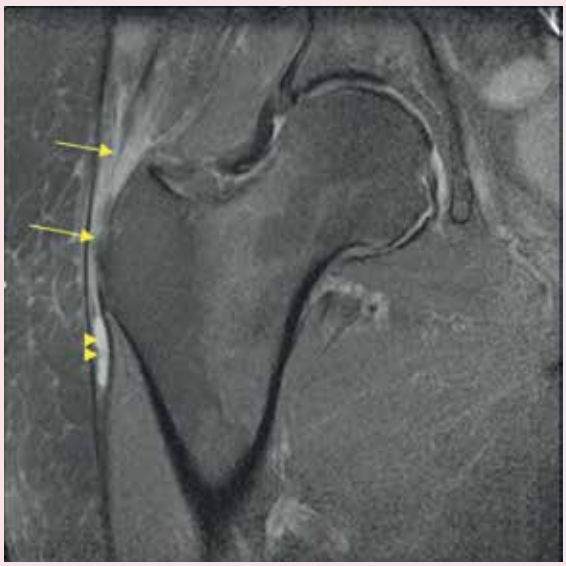


Figure 24. Coronal PD weighted MRI of the right hip with fat saturation demonstrating thickening and increased signal in the gluteus medius tendon at its insertion with focal discontinuity of the tendon fibres (arrows) indicative of tendinopathy and a partial thickness tear at the insertion. Bright fluid again demonstrated in the bursa (arrow heads).

QUESTION 4

What treatment options are available for Erin in the management of her GTPS? Does imaging have a role in the treatment of GTPS?

CASE 6 ANSWERS

ANSWER 1

GTPS has recently replaced the term 'trochanteric bursitis' to describe patients with chronic pain accompanied by reproducible tenderness in the region of the greater trochanter, buttock and lateral thigh. The reason for this is that the pain and tenderness can have many different causes including, but not confined to:

- trochanteric bursitis
- tendinopathy and tears of the gluteus medius and minimus tendons
- iliotibial band (ITB) disorders
- general or localised pathology in the surrounding tissues.[52]

With the advent of high resolution ultrasound and MRI it is becoming more apparent that tears of the gluteus medius and minimus tendons may be the leading cause of GTPS[53] and that trochanteric bursitis is commonly secondary to pathologies in the gluteus medius and minimus tendons.[54] Isolated distension of the trochanteric bursae is uncommon, and does not usually occur in the absence of gluteus medius pathology.[53]

FEEDBACK

GTPS is an important diagnosis to make, as the pain in this syndrome can refer down the lateral thigh, anterior groin and buttock, and can thus mimic the pain of nerve root compression in the lumbar spine or osteoarthritis (OA) of the hip.[52,54]

To complicate matters, patients with pre-existing low back pain and OA of the hip and knee are predisposed to developing GTPS because of altered lower limb biomechanics and abnormal force vectors across the hip joint.[55] Failure to make the diagnosis can result in costly patient referrals, inappropriate diagnostic testing and surgery that may not resolve the pain.[52]

GTPS is associated with significant impairment to the activities of daily living,[56] so an accurate diagnosis and treatment is of great benefit to the patient.

The anatomy around the greater trochanter has been likened to the 'rotator cuff of the hip'.[57] The gluteus medius and minimus tendons insert onto the greater trochanter while the adjacent trochanteric bursae, which vary in size, location and number, provide cushioning for the gluteal tendons, ITB, and the tensor fascia lata, with tension in the ITB causing repetitive friction.[54] This results in a similar spectrum of pathologies affecting the tendons and bursae as seen in the rotator cuff of the shoulder. The subgluteus maximus bursa is located lateral to the greater trochanter, between the gluteus medius tendon and the gluteus maximus muscle, and is the most frequently incriminated bursa in GTPS.[58]

Tears of the gluteal tendons and injury to the bursa can also occur with trauma.[54] In a setting of acute trauma it is important to exclude femoral fracture as a cause of pain.

Bursitis and tendon pathology can be caused by infection, especially tuberculosis, as well as crystal deposition diseases and inflammatory arthropathies such as rheumatoid arthritis.[59]

ANSWER 2

GTPS is more common in middle-aged and elderly women.[52] It has been found to be more common in patients with coexisting low back pain, knee OA, ITB tenderness and obesity. Again, an alteration in gait and altered biomechanics may be a contributing factor.[55]

GTPS is a common condition with an incidence in primary care of 1.8 patients per 1000 per year.[56]

In the younger population GTPS can also occur in athletes, particularly in runners and those performing step aerobics.[60,61]

ANSWER 3

Ultrasound and MRI are the most useful imaging modalities in the investigation of GTPS.

Ultrasound is well suited to assessing the structures around the greater trochanter due to their superficial nature. It is readily available and safe. Tendinopathy of the gluteal tendons is characterised by thickening, decreased echogenicity and sometimes increased vascularity within the tendons. Tears, which can be partial or full thickness, appear as focal discontinuities of the tendon fibres.

Trochanteric bursitis may demonstrate thickening of the bursa and/or fluid within the bursa. The ITB can be assessed for thickening and for fluid deep to it. As ultrasound is performed in real time, the exact anatomical site of the patient's focal tenderness can be determined, enabling confirmation of the clinical diagnosis of GTPS.

The hip joint can also be assessed for the presence of an effusion.

Ultrasound can be used to guide aspiration of fluid in the bursa and to inject local anaesthetic and corticosteroids into the bursa.

MRI can also be used to image the structures around the greater trochanter. A recent review determined that it had the highest correlation with surgical and clinical findings in patients with GTPS.[62]

On PD weighted images, normal tendons appear low in signal (i.e. black). Tendinopathy is characterised by thickening and increased signal (i.e. whiter and brighter) in the tendon. Tendon tears appear as focal loss of continuity of the tendon fibres with a bright fluid signal in the defect. Bursitis demonstrates thickening of the wall of the bursa and may demonstrate bright fluid distending the bursa. These pathological tissue changes and fluid collections characterised by increased signal on the PD images can be made more conspicuous by applying a fat saturation pulse, which removes the bright signal from adjacent fat.

MRI scanning will also enable an accurate assessment of the hip joint and assist in excluding other conditions such as stress fractures and avascular necrosis of the femoral neck. It is important to diagnose these conditions, especially in patients with risk factors for them, to avoid a misdiagnosis of GTPS.

ANSWER 4

Erin can be treated conservatively with non-steroidal anti-inflammatory drugs (NSAIDs), ice, weight loss and physical therapy.[58]

Erin has a 6-week course of physiotherapy and applies ice regularly. She is not keen to use NSAIDs as she developed epigastric discomfort 2 years ago when they were prescribed for her shoulder injury. She wants to know if there is any other treatment available. You suggest that she could have an ultrasound guided injection into the greater trochanteric bursa. There is evidence to support the use of a steroid injection into the bursa, with a three-fold increase in recovery at 5 years.[56] Landmark guided injections are effective in 77% of patients 1 week after injection and 61% of patients 6 months post-procedure.[63]

The technique involves placing the patient on the examination table on their side in the lateral decubitus position with the affected side uppermost. As the bursae are relatively superficial structures, they are readily accessible. Using a sterile technique the needle tip is inserted into the affected bursa under ultrasound guidance. The bursa is injected with corticosteroid and a local anaesthetic. Surgery may be considered for patients with intractable pain.[60]

FEEDBACK

Erin undergoes a trochanteric bursa injection under ultrasound guidance. An image of her trochanteric bursa before injection is shown in *Figure 25*.

Erin comes to see you 6 weeks after the procedure. She tells you she has had a significant decrease in her pain. Six months later, Erin is pain free (*Figure 26*).

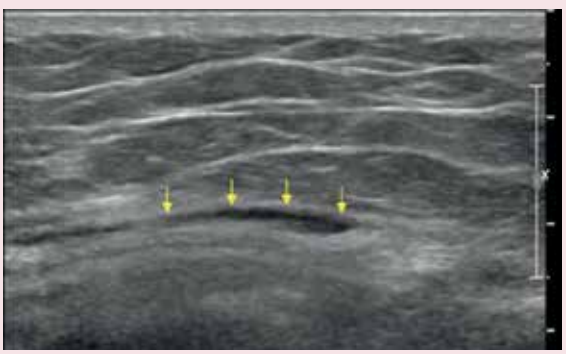


Figure 25. Ultrasound image demonstrates fluid distending the trochanteric bursa (arrows).

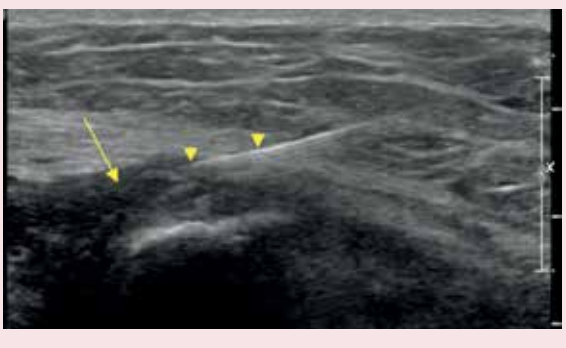


Figure 26. Ultrasound image demonstrates thickened hypoechoic gluteus medius tendon (arrow) with needle tip (arrow heads) in the trochanteric bursa.

CASE 7

MICHAEL HAS SHOULDER PAIN

Michael, aged 40 years, is an administrative assistant who presents with a sore right shoulder. He has a desk job but is a keen amateur sportsman and has recently started training for an upcoming 'pier to pub' swim. His pain is preventing him swimming freestyle and reaching out for objects on his bedside table. He is eager to have the condition diagnosed and treated because he would like to continue his training.

On examination his BP is 145/80 mmHg and his BMI is 23.5. He is afebrile. He has a painful arc of abduction between 25° and 70° but is able to 'push through' the pain and is pain free when he fully abducts his right shoulder. There is no weakness in his rotator cuff muscles and he has a good range of movement in his cervical spine.

QUESTION 1

What is your provisional diagnosis?

FURTHER INFORMATION

Michael has a painful arc of abduction and a positive Hawkins test[64] (pain on axial rotation of an already 90° abducted shoulder) as well as a positive Neer test[65] (pain with forced forward elevation against the acromion).

QUESTION 2

Do you need to do any imaging at this stage?

QUESTION 3

How does shoulder impingement occur?

FURTHER INFORMATION

Michael has completed a course of anti-inflammatory tablets and is doing physiotherapy, but when he comes to see you for a follow-up appointment, he complains of gastric symptoms and says physiotherapy is making his pain worse.

QUESTION 4

Given Michael's feedback, you decide to organise imaging. What tests would you order?

QUESTION 5

What information will they give you?

FURTHER INFORMATION

An ultrasound demonstrates a painful arc of abduction as the supraspinatus (SS) tendon passes beneath the coracoacromial ligament and mild SS tendinopathy. You decide that a bursal injection of cortisone is likely to help reduce Michael's pain and enable him to do his physiotherapy exercises properly. Ultrasound guided injection is a simple, safe and effective technique for getting the steroid into the right place (see *Figure 27*).

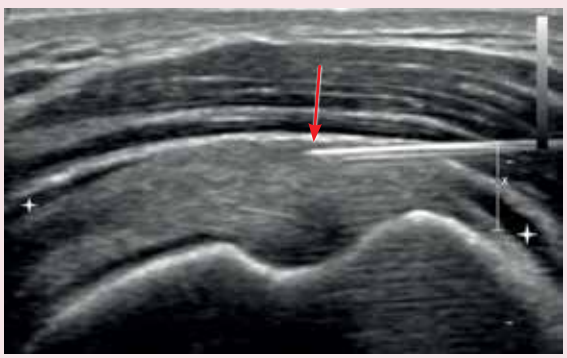


Figure 27. Needle (arrow) injecting fluid into the subacromial bursa. The injected fluid appears black (stars).

QUESTION 6

What are the potential side effects of cortisone injections that you need to warn Michael about?

FURTHER INFORMATION

At Michael's follow-up appointment, the radiology report states, and Michael confirms, that his painful arc of abduction was relieved by the anaesthetic component of the injection. This helps confirm the diagnosis of impingement.

QUESTION 7

Suppose Michael's ultrasound had shown a 15 mm focus of soft calcification within the SS tendon and he called you the afternoon following the ultrasound describing sudden onset of unrelenting pain in his shoulder when he was lifting shopping out of the car. What would you think had happened?

CASE 7 ANSWERS

ANSWER 1

The most likely diagnosis is rotator cuff tendinopathy or impingement syndrome involving his subacromial bursa and the rotator cuff tendons. If Michael has had long-standing niggling shoulder pain that has been exacerbated by the new training regimen, the possibility of calcific tendinopathy or bursitis should be considered.

In this age group a rotator cuff tear or glenohumeral arthritis are unlikely without a specific traumatic event, but Michael could have strained his acromioclavicular (AC) joint. His history is not typical of a labral tear.

ANSWER 2

If your clinical evaluation confirms the diagnosis of impingement or rotator cuff tendinopathy then Michael does not need imaging unless he fails to respond to treatment.

ANSWER 3

Shoulder impingement is caused by the rotator cuff tendons and bursa rubbing as they pass below the subacromial arch during shoulder abduction.[65] This usually occurs secondary to subtle imbalance of the glenohumeral joint with the head moving forward in the socket on abduction to trap these structures. This is why appropriate physiotherapy exercises are vital to realign the joint and make more space for the tendons to pass beneath the coracoacromial arch. Impingement is made worse if the tendons and bursa become enlarged because of this rubbing, or if a focus of calcification develops and expands the tendons. Impingement may develop if there is not enough space for the tendons to fit through the arch due to an acromial bone spur or due to expansion of the AC joint secondary to arthritis.

ANSWER 4

You would order a shoulder X-ray and ultrasound. MRI is not the first-line modality for investigation of impingement. Note that benefits for shoulder ultrasound are only payable when referral is based on the clinical indicators outlined in the Medicare Benefits Schedule item descriptions. No benefits are payable if the referrable is for non-specific shoulder pain.

ANSWER 5

While an X-ray is usually normal in this age group, it is able to show the shape of the acromion, subacromial bone spurs (see *Figure 28*), soft tissue calcification, AC joint OA and misalignment. It may also reveal the unexpected, such as glenohumeral arthritis or a bone tumour.

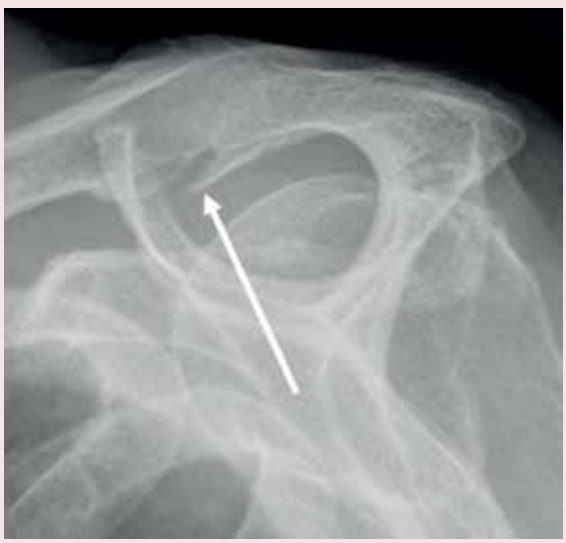


Figure 28. Small subacromial bone spur (arrow) that may rub the SS tendon. Consider surgical referral if symptoms are non-responsive to conservative therapy.

Dynamic ultrasound evaluation is the mainstay of the diagnosis of shoulder impingement. The principle criterion is observation of mechanical compression of the SS tendon and/or bursa beneath the coracoacromial arch on abduction combined with the clinical observation that this elicits patient pain.[66] Other signs include bunching or fluid distension of the bursa[67] and bunching or crowding of the SS tendon as it tries to pass beneath the coracoacromial arch.[68] Dynamic ultrasound can confirm, but not exclude, the diagnosis of impingement as false negative results of up to 18% are reported.[69] This may occur because the impingement is inactive at the time of examination or masked by anti-inflammatory or pain relief medication.

Ultrasound will demonstrate the cause of impingement such as tendinopathy, calcification in the tendon, tears of the tendon and bursitis (see *Figure 29*). A thick bursa or painless bursal bunching should never be interpreted as a sign of impingement as they can be present in up to 30% of asymptomatic shoulders (see *Figure 30*).

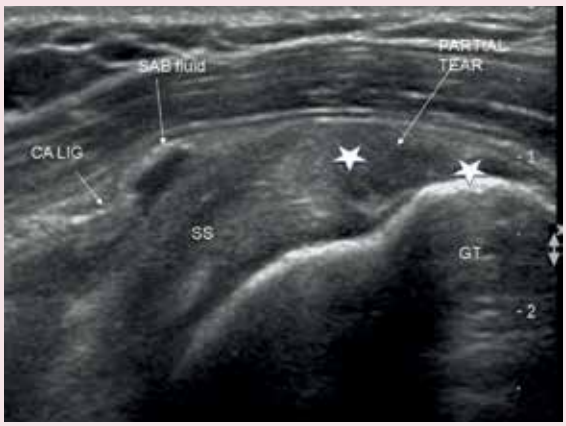


Figure 29. Fluid distending the bursa and bulging above the coracoacromial ligament as Michael starts to abduct. Note thickened, mildly hypoechoic SS tendon and the partial tear.

Figure 30. A thick but well-defined bursa in an asymptomatic man. This should not be mistaken for impingement.

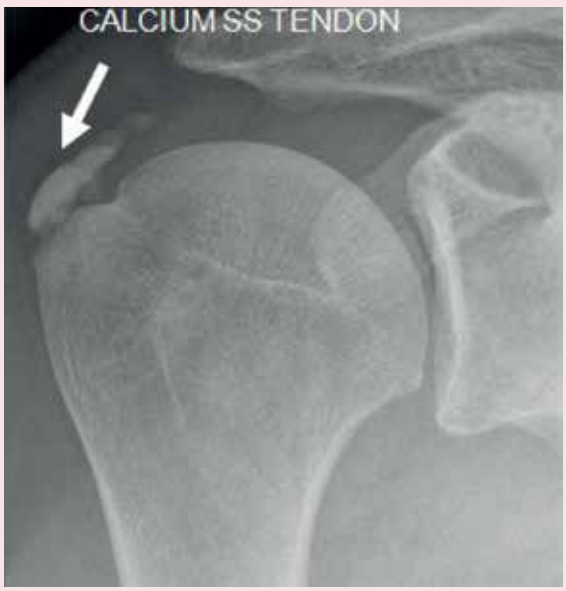


Figure 31. Calcium in the SS tendon.

ANSWER 6

The only serious but rare side effect of a cortisone injection is infection (usually staphylococcus). Other side effects reported by patients are a red skin flush usually around the head and neck, which is sometimes associated with feeling a bit under the weather with flu-like symptoms. Patients may get a flare of pain in the injected area for 1–3 days after the anaesthetic has worn off.[70] If Michael is diabetic he is likely to see a rise in his blood glucose levels for about a week, so he may need to check his blood sugars more frequently and adjust his medication.

ANSWER 7

In this scenario, some of the calcium from the tendon has extruded into the bursa setting up a severe bursitis, which is an extremely painful condition (see *Figures 31*, *32* and *33*). It is usually a self-limiting condition within a few weeks, but most patients will elect to have ultrasound guided aspiration of the calcium (see *Figure 34*) followed by steroid injection, which is an extremely effective method of pain control, usually working within days.[71] This technique is also effective for pain control of calcific tendonitis and will reduce the bulk of the tendon, making it easier for the tendon to fit through the coracoacromial arch on abduction.

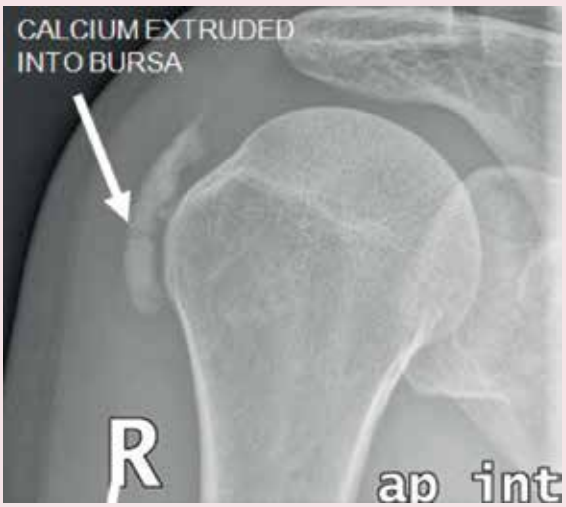


Figure 32. The calcium has extruded into the subacromial bursa, resulting in a painful bursitis.

Figure 33. Ultrasound image showing the extruded calcium in the bursa.

Figure 34. Calcium that has been aspirated from the bursa.

1. National Breast Cancer Centre. Clinical practice guidelines for the management and support of younger women with breast cancer. 2004 Camperdown: National Breast Cancer Centre. Available from http://nbcc.org.au
2. National Radiological Protection Board. X-rays: how safe are they? Chilton: National Radiological Protection Board, 2001. Available at www.hpa.org.uk/web/HPAweb&HPAwebStandard/HPAweb_C/1259151968131 [accessed 22 April 2013].
3. National Breast Cancer Centre. Breast imaging: a guide for practice. Camperdown, NSW: National Breast Cancer Centre, 2002.
4. Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice, 8th edn. East Melbourne: Royal Australian College of General Practitioners, 2012, p.65–6.
5. Davidson KG, Dubinsky TJ. Ultrasonographic evaluation of the endometrium in postmenopausal vaginal bleeding. Radiol Clin North Am 2003;41(4):769–80.
6. Lidor A, Ismajovich B, Confino E, David, MP. Histopathological findings in 226 women with post-menopausal uterine bleeding. Acta Obstet Gynecol Scand 1986;65(1):41–3.
7. Sahdev A. Imaging the endometrium in postmenopausal bleeding. BMJ 2007;334(7594):635–6.
8. Soliman PT, Oh JC, Schmeler KM, Sun CC, Slomowitz, BM, Gershenson, DM, et al. Risk factors for young premenopausal women with endometrial cancer. Obstet Gynecol 2005;105(3):575–80.
9. Nayfield SG, Karp JE, Ford LG, Dorr FA, Kramar BS. Potential role of tamoxifen in prevention of breast cancer. J Natl Cancer Inst 1991;83(20):1450–9.
10. Cancer Council Australia. Clinical practice guidelines for the treatment and management of endometrial cancer. Available at www.canceraustralia.gov.au/sites/default/files/publications/ncgc-vaginal-bleeding-flowcharts-march-20111_504af02038614.pdf
11. Gerber B, Krause A, Muller H, Reimer T, Kulz T, Makovitsky A, et al. Effects of adjuvant tamoxifen on the endometrium in postmenopausal women with breast cancer: a prospective long-term study using transvaginal ultrasound. J Clin Oncol 2000;18(20):3464–70.
12. Erdem M, Bilgin U, Bozkurt N, Erdem Al. Comparison of transvaginal ultrasonography and saline infusion sonohysterography in evaluating the endometrial cavity in pre- and postmenopausal women with abnormal uterine bleeding. Menopause 2007;14(5):846–52.
13. Bradley LD, Falcone T, Magen AB. Radiographic imaging techniques for the diagnosis of abnormal uterine bleeding. Obstet Gynecol Clin North Am 2000;27(2):245–76.
14. Nalaboff KM, Pellerito JS, Ben-Levi E. Imaging the endometrium: disease and normal variants. Radiographics 2001;21(6):1409–24.
15. Hardesty LA, Sumkin JH, Hakim C, Johns C, Nath M The ability of helical CT to preoperatively stage endometrial carcinoma. Am J Roentgenol 2001;176(3):603–6.
16. Manfredi R, Mirk P, Maresca G, et al. Local-regional staging of endometrial carcinoma: role of MR imaging in surgical planning. Radiology 2004;231(2):372–8.
17. Fisher B, Costantino JP, Wickerham DL, et al. Tamoxifen for prevention of breast cancer: report of the National Surgical Adjuvant Breast and Bowel Project P-1 Study. J Natl Cancer Inst 1998;90(18):1371–88.
18. Bergman L, Beelen ML, Gallee MP, Hollema H, Benraadt J, van Leeuwen FE. Risk and prognosis of endometrial cancer after tamoxifen for breast cancer. Lancet 2000;356(9233):881–7.
19. Kedar RP, Bourne TH, Powles TJ, et al. Effects of tamoxifen on uterus and ovaries of postmenopausal women in a randomised breast cancer prevention trial. Lancet 1994;343(8909):1318–21.
20. Neff MJ. Practice guidelines. Evidence-based guidelines for neuroimaging in patients with nonacute headache. Am Fam Physician 2005;71(6):1219–22.
21. Government of Western Australia Department of Health. A clinical decision support tool and educational resource for diagnostic imaging. Available at www.imagingpathways.health.wa.gov.au/includes/index.html [accessed 22 April 2013].
22. Acheson J. Idiopathic intracranial hypertension and visual function. Br Med Bull 2006;79–80(1):233–44.
23. Saposnik G, Barinagarrementeria F, Brown RD, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011;42(4):1158–92.
24. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. Lancet Neurol 2007;6(2):162–70.
25. Hasbun R, Abrahams J, Jekel J, Quagliarello VJ. Computed tomography of the head before lumbar puncture in adults with suspected meningitis. N Engl J Med 2001;345(24):1727–33.
26. The Royal Children's Hospital. Lumbar puncture guidelines. Available at www.rch.org.au/clinicalguide/guideline_index/lumbar_puncture_guideline [accessed 22 April 2013].
27. Sanchez R, Strouse PJ. The knee: MR imaging of uniquely pediatric disorders. Magn Reson Imaging Clin N Am 2009;17(3):521–37.
28. Stevens MA, El-Khoury GY, Kathol MH, Brandser EA, Chow S. Imaging features of avulsion injuries. Radiographics 1999;19(3):655–72.
29. Harcke HT, Mandell GA, Maxfield BA. Trauma to the growing skeleton. In: Kuhn JP, Slovis TL, Haller JO (eds). Caffey's pediatric diagnostic imaging. 10th edn. Philadelphia: Mosby, 2003, 2269–303.
30. De Smet AA, Fisher DR, Graf BK, Lange RH. Osteochondritis dissecans of the knee: value of MR imaging in determining lesion stability and the presence of articular cartilage defects. Am J Roentgenol 1990;155(3):549–53.
31. Wall E, Von Stein D. Juvenile osteochondritis dissecans. Orthop Clin North Am 2003;34(3):341–53.
32. Polousky JD. Juvenile osteochondritis dissecans. Sports Med Arthrosc 2011;19(1):56–63.
33. Kocher MS, Tucker R, Ganley TJ, Flynn JM. Management of osteochondritis dissecans of the knee: current concepts review. Am J Sports Med 2006;34(7):1181–91.
34. Bohndorf K. Osteochondritis (osteochondrosis) dissecans: a review and new MRI classification. Eur Radiol 1998;8(1):103–12.
35. Kramer J, Stiglbauer R, Engel A, Prayer L, Imhof H. MR contrast arthrography (MRA) in osteochondrosis dissecans. J Comput Assist Tomogr 1992;16(2):254–60.
36. Herring JA. Disorders of the knee. In: Herring JA ed. Tachdjian's pediatric orthopedics. 3rd edn. Philadelphia: WB Saunders Co, 2001, 789–838.
37. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999;353(9162):1386–9.
38. Pengo V, Lensing AW, Prins MH, et al. Incidence of chronic thromboembolic pulmonary hypertension after pulmonary embolism. N Engl J Med 2004;350(22):2257–64.
39. Stein PD, Woodard PK, Weg JG, et al. Diagnostic pathways in acute pulmonary embolism: recommendations of the PIOPED II investigators. Radiology 2007;242(1):15–21.
40. Stein PD, Fowler SE, Goodman LR, et al. Multidetector computed tomography for acute pulmonary embolism. N Engl J Med 2006;354(22):2317–27.
41. Mills SR, Jackson DC, Older RA, Heaston DK, Moore AV. The incidence, etiologies, and avoidance of complications of pulmonary angiography in a large series. Radiology 1980;136(2):295–9.
42. Worsley DF, Alavi A, Aronchick JM, Chen JT, Greenspan RH, Ravin CE. Chest radiographic findings in patients with acute pulmonary embolism: observations from the PIOPED study. Radiology 1993;189(1):133–6.

43. Zierler BK. Ultrasonography and diagnosis of venous thromboembolism. Circulation 2004;109:9–14.
44. Forbes KP, Reid JH, Murchison JT. Do preliminary chest X-ray findings define the optimum role of pulmonary scintigraphy in suspected pulmonary embolism? Clin Radiol 2001;56(5):397–400.
45. Hurwitz LM, Yoshizumi T, Reiman RE, et al. Radiation dose to the fetus from body MDCT during early gestation. Am J Roentgenol 2006;186(3):871–6.
46. Cook JV, Kyriou J. Radiation from CT and perfusion scanning in pregnancy. BMJ 2005;331(7512):350.
47. Eichinger S. D-dimer testing in pregnancy. Pathophysiol Haemost Thromb 2003;33(5–6):327–9.
48. Francalanci I, Comeglio P, Alessandrello Liotta A, et al. D-dimer plasma levels during normal pregnancy measured by specific ELISA. Int J Clin Lab Res 1997;27(1):65–7.
49. Perrier A, Roy PM, Aujesky D, et al. Diagnosing pulmonary embolism in outpatients with clinical assessment, D-dimer measurement, venous ultrasound, and helical computed tomography: a multicenter management study. Am J Med 2004;116(5):291–9.
50. Turkstra F, Kuijer PM, van Beek EJ, Brandjes DP, ten Cate JW, Buller HR Diagnostic utility of ultrasonography of leg veins in patients suspected of having pulmonary embolism. Ann Intern Med 1997;126(10):775–81.
51. Kang MJ, Park CM, Lee CH, Goo JM, Lee HJ. Dual-energy CT: clinical applications in various pulmonary diseases. Radiographics 2010;30(3):685–98.
52. Tortolani PJ, Carbone JJ, Quartararo LG. Greater trochanteric pain syndrome in patients referred to orthopedic spine specialists. Spine J 2002:2(4):251–4.
53. Bird PA, Oakley SP, Schnier R, Kirkham BW. Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pain syndrome. Arthritis Rheum 2001;44(9):2138–45.
54. Kingzett-Taylor A, Tirman PF, Feller J, et al. Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. Am J Roentgenol 1999:173(4):1123–6.
55. Segal NA, Felson DT, Torner JC, et al. Greater trochanteric pain syndrome: epidemiology and associated factors. Arch Phys Med Rehabil 2007;88(8):988–92.
56. Lievense A, Bierma-Zeinstra S, Schouten B, Bohnen A, Verhaar J, Koes B. Prognosis of trochanteric pain in primary care. BRJ Gen Pract 2005;55(512):199–204.
57. Bunker TD, Esler CN, Leach WJ. Rotator-cuff tear of the hip. J Bone Joint Surg Br 1997;79(4):618–620.
58. Williams BS, Cohen SP. Greater trochanteric pain syndrome: a review of anatomy, diagnosis and treatment. Anesth Analg 2009;108(5):1662–70.
59. Tanaka H, Kido K, Wakisaka A, Mine T Kawai S. Trochanteric bursitis in rheumatoid arthritis. J Rheumatol 2002;29(6):1340–1.
60. Slawski DP, Howard RF. Surgical management of refractory trochanteric bursitis. Am J Sports Med 1997;25(1):86–9.
61. Clancy WG. Runners' injuries. Part two. Evaluation and treatment of specific injuries. Am J Sports Med 1980;8(4):287–9.
62. McMahon SE, Smith TO, Hing CB. A systematic review of imaging modalities in the diagnosis of greater trochanteric pain syndrome. Musculoskeletal Care 2012;10(4):232–9.
63. Shbeeb MI, O'Duffy JD, Michet CJ Jr, O'Fallon WM, Matteson ELI. Evaluation of glucocorticosteroid injection for the treatment of trochanteric bursitis. J Rheumatol 1996;23(12):2104–6.
64. Hegedus EJ, Goode A, Campbell S, et al. Physical examination tests of the shoulder: a systematic review with meta-analysis of individual tests. Br J Sports Med 2008;42(2):80–92.
65. Neer CS 2nd. Impingement lesions. Clin Orthop Relat Res 1983;173: 70–7.
66. Read JW, Perko M. Ultrasound diagnosis of subacromial impingement for lesions of the rotator cuff. Australasian J Ultrasound Med 2010;13(2): 11–15.
67. Farin PU, Jaroma H, Harju A, Soimakillio SI. Shoulder impingement syndrome: sonographic evaluation. Radiology 1990;176(3):845–9.
68. Bureau NJ, Beauchamp M, Cardinal E, et al. Dynamic sonography evaluation of shoulder impingement syndrome. Am J Roentgenol 2006;187(1):216–20.
69. Read JW, Perko M. Shoulder ultrasound: diagnostic accuracy for impingement syndrome, rotator cuff tear, and biceps tendon pathology. J Shoulder Elbow Surg 1998;7(3):264–71.
70. Brinks A, Koes BW, Volkers AC, Verhaar JA, Bierma-Zeinstra SM. Adverse effects of extra-articular corticosteroid injections: a systematic review. BMC Musculoskelet Disord 2010;11:206.
71. Farin PU, Rasanen H, Jaroma H, Harju A. Rotator cuff calcifications: treatment with ultrasound-guided percutaneous needle aspiration and lavage. Skeletal Radiol 1996;25(6):551–4.

RESOURCES FOR DOCTORS

- The National Breast Cancer Centre's publication *Breast imaging: a guide for practice* is available for free download from Cancer Australia's website, http://canceraustralia.gov.au/publications-resources/cancer-australia-publications/breast-imaging-guide-practice [accessed 22 April 2013].
- The UK National Radiological Protection Board, with the College of Radiographers, the Royal College of Radiologists and the Royal College of General Practitioners, has produced a helpful, plain-language pamphlet called 'X-rays: how safe are they?' It is available for free download at www.hpa.org.uk/web/HPAweb&HPAwebStandard/HPAweb_C/1259151968131 [accessed 22 April 2013].
- Debra Ikeda's *Breast imaging: the requisites* provides a wealth of knowledge about diagnostic imaging of the breast.
- The American College of Radiology has developed ACR Appropriateness Criteria®, which can be found at www.acr.org/Quality-Safety/Appropriateness-Criteria [accessed 22 April 2013]. These criteria help referring physicians to make the most appropriate imaging decision for their patients.
- The Scottish Intercollegiate Guidelines Network guideline for investigation of post-menopausal bleeding can be found at www.sign.ac.uk/guidelines/fulltext/61/index.html [accessed 22 April 2013].
- Royal Australian College of General Practitioners. Guidelines for preventive activities in general practice, 8th edn. East Melbourne: Royal Australian College of General Practitioners, 2012.
- National Breast Cancer Centre. Clinical practice guidelines for the management and support of younger women with breast cancer. 2004 Camperdown: National Breast Cancer Centre. Available from http://nbcc.org.au
- Cancer Council Australia. Clinical practice guidelines for the treatment and management of endometrial cancer. Available at www.canceraustralia.gov.au/sites/default/files/publications/ncgc-vaginal-bleeding-flowcharts-march-20111_504af02038614.pdf
- The Royal Australian and New Zealand College of Radiologists has developed an excellent website for consumers and referring doctors about a range of imaging procedures. It is available at www.insideradiology.com.au [accessed 22 April 2013].

RESOURCES FOR PATIENTS

- The UK National Radiological Protection Board, with the College of Radiographers, the Royal College of Radiologists and the Royal College of General Practitioners, has produced a helpful, plain-language pamphlet called 'X-rays: how safe are they?' It is available for free download at www.hpa.org.uk/web/HPAweb&HPAwebStandard/HPAweb_C/1259151968131 [accessed 22 April 2013].
- The Department of Health and Ageing has developed a helpful website (www.cancerscreening.gov.au) that provides information about a range of cancer screening programs.
- Cancer Council Australia (www.cancer.org.au) provides information about different types of cancer, including breast cancer.
- The Royal Australian and New Zealand College of Radiologists has developed an excellent website for consumers and referring doctors about a range of imaging procedures. It is available at www.insideradiology.com.au [accessed 22 April 2013].
- An information sheet for patients about cortisone injections is available at www.vhmi.com.au/upload/Cortisone%20Injections1.pdf [accessed 22 April 2013].

Imaging

In order to qualify for 6 Category 2 points for the QI&CPD activity associated with this unit:

- read and complete the unit of *check* in hard copy or online at the *gplearning* website at www.gplearning.com.au, and
- log onto the *gplearning* website at www.gplearning.com.au and answer the following 10 multiple choice questions (MCQs) online, and
- complete the online evaluation.

If you are not an RACGP member, please contact the *gplearning* helpdesk on 1800 284 789 to register in the first instance. You will be provided with a username and password that will enable you access to the test.

The expected time to complete this activity is 3 hours.

Do not send answers to the MCQs into the *check* office. This activity can only be completed online at www.gplearning.com.au.

If you have any queries or technical issues accessing the test online, please contact the *gplearning* helpdesk on 1800 284 789.

FOR A FULL LIST OF ABBREVIATIONS AND ACRONYMS USED IN THESE QUESTIONS PLEASE GO TO PAGE 3.
FOR EACH QUESTION BELOW SELECT ONE OPTION ONLY.

QUESTION 1

Adam is a 19-year-old university student who presents with persistent knee pain. He jogs and cycles and plays competitive basketball two nights per week. He has noticed that his pain is worse after exercise. Imaging reveals that he has a left osteochondral defect in the medial femoral condyle.

When is treatment such as arthroscopic drilling or chondrocyte reimplantation recommended?

A. If pain persists after 6 weeks of non-weight-bearing
B. If the lesion is unstable
C. If the lesion is stable
D. If he has persistent pain for more than 3 months
E. If he has irregularity of the medial condyle on X-ray.

QUESTION 2

Which of the following is true of tamoxifen?

A. Tamoxifen can act as both an oestrogen agonist and an oestrogen antagonist.
B. Tamoxifen increases the risk of cervical cancer.
C. Tamoxifen is used for the treatment of receptor-negative breast cancer.
D. Tamoxifen is usually prescribed to be taken for 4 years following a diagnosis of breast cancer.
E. Tamoxifen decreases the risk of endometrial hyperplasia.

QUESTION 3

Dot, aged 65 years, is brought to your clinic by her daughter June. Dot has had intermittent vaginal bleeding for 2 months. The bleeding is not heavy, but has occurred on most days. Dot is married to Bill; June is their only child. You order a transvaginal ultrasound, which shows a smooth endometrium of approximately 3 mm. Which of the following is the most likely diagnosis?

A. Endometrial atrophy
B. Benign endometrial hyperplasia
C. Endometrial carcinoma
D. Endometrial polyp
E. Uterine fibroid.

QUESTION 4

Mariko presents to your clinic with a severe headache of 3 hours duration. The headache started suddenly and is severe and unremitting. It is generalised. Mariko also complains of photophobia. On examination, she has a temperature of 37.5°C and some slight neck stiffness. You make a provisional diagnosis of meningitis. What imaging would you have performed immediately?

A. Skull X-ray
B. CT scan
C. MRI
D. MRV
E. None of the above.

QUESTION 5

Faraza, aged 30 years, comes to see you because she has felt a lump in her right breast. On examination you can feel a swelling in the UOQ of the breast. What is the most appropriate initial investigation?

A. Mammogram
B. Ultrasound
C. Mammogram and ultrasound
D. Referral to BreastScreen
E. Biopsy.

QUESTION 6

Janet, aged 53 years, presents with a 2-month history of pain in her right hip. She walks and swims regularly and has recently started attending a gym. She noticed the pain in her right hip shortly after starting a new exercise regimen that includes lunges and squats. These exercises seem to aggravate the pain. She has tenderness to palpation over the right greater trochanter and right buttock. What is the most likely cause of her pain?

A. OA of the hip
B. Gluteus medius tear
C. Torn acetabular labrum
D. Polymyalgia rheumatica
E. OA of the spine.

QUESTION 7

Alison, aged 37 years, presents with shortness of breath and some chest discomfort. She recently returned from a trip to Sweden where she was on holiday with friends. She is afebrile and her pulse rate is 100 beats per minute. Her chest is clear and she has no calf tenderness. You suspect she could have a PE. Along with the Wells Pulmonary Embolism Score, which test can be used to determine the most appropriate imaging pathway in Alison's case?

A. D-dimer
B. ECG
C. FBE
D. Coagulation profile
E. Spirometry.

QUESTION 8

Alison (see *Question 7*) is admitted to hospital with suspected PE. While she is having blood tests and an ECG, she tells staff that her period is late and there is a possibility she could be pregnant. What is a useful first test to perform if Alison is pregnant?

A. Pulmonary angiography
B. Venous Doppler ultrasound
C. MRI
D. CTPA
E. V/Q scan.

QUESTION 9

Kyle, aged 14 years, presents to your surgery with a 6-week history of pain in his left knee. He is a keen athlete and plays sport on most days. He is otherwise well. On examination he is afebrile. He has a full range of movement in his knee but has mild tenderness on palpation of his left tibial tuberosity. What is the most likely diagnosis?

A. Viral synovitis
B. Patellofemoral instability
C. Osteochondritis dissecans
D. Osgood–Schlatter disease
E. Left meniscal tear.

QUESTION 10

Walid, aged 57 years, presents with pain in his right shoulder. He has had the pain for 2 months. He is a keen swimmer, but the pain makes it difficult for him to swim more than 20 laps. On examination he has a painful arc and internal rotation on the right causes pain. Which imaging technique will give the most information to help you make a diagnosis of impingement syndrome?

A. X-ray
B. Ultrasound
C. Dynamic ultrasound
D. MRI
E. CT scan.

check

Independent learning program for GPs