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RATIONAL IMAGING

Investigating pleural thickening

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This series provides an update on the best use of different imaging methods for common or important clinical presentations. The series advisers are Fergus Gleeson, consultant radiologist, Churchill Hospital, Oxford, and Kamini Patel, consultant radiologist, Homerton University Hospital, London. To suggest a topic for this series, please email us at practice@bmj.com.

As pleural thickening can have a benign or malignant cause, use of the appropriate imaging techniques is crucial to a correct diagnosis. The authors explore the options

A 77 year old man presented with left sided chest and back pain that did not respond to simple analgesics. He had a history of atrial fibrillation and was taking warfarin. A retired joiner, he had been exposed to asbestos in the 1960s and '70s. He was a non-smoker. Examination showed reduced air entry on the left and tenderness at the inferior aspect of the scapula. A chest radiograph showed bilateral calcified pleural plaques and pleural thickening on the left hand side (fig 1).

Background and differential diagnosis

Pleural thickening can be focal or diffuse and has various causes (table). Imaging is used for confirming the presence, nature, and extent of disease and for distinguishing benign from malignant causes. The appearances of some of these benign and malignant diseases are similar, and only the presence of invasion and/or metastatic disease are definite indicators of malignancy. However, some features seen on imaging can help differentiate benign and malignant causes.

Chest radiography

Pleural disease is often first detected or suspected on a chest radiograph. The pleural surfaces are generally seen on a chest radiograph only when they are abnormal, although the fissures may be visualised. Pleural thickening is easiest to detect on a chest radiograph at the edges of the lung, where the x ray beam passes through it tangentially and it is seen as soft tissue density inside and parallel to the chest wall (fig 1). When viewed en face (that is, when the beam passes straight through, not tangentially), the thickening is more difficult to detect, appearing ill defined and sometimes veil-like, with poorly defined margins.[1] Deposition of extrapleural fat can cause diagnostic confusion and simulate pleural disease. A chest radiograph is poor at localising and characterising pleural disease, and when malignancy is suspected other imaging modalities are usually needed for localisation (to confirm pleural involvement and to determine the sites of involvement) and to help make a diagnosis.

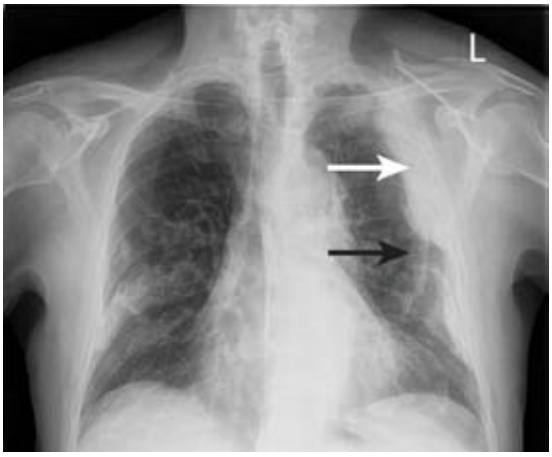

Fig 1 | Frontal chest radiograph demonstrates thickening of the pleura in the left upper zone (white arrow) and bilateral calcified pleural plaques (black arrow). The pleural thickening is best seen at the lung edges—where the x ray beam passes through it tangentially (white arrow)—as an area of soft tissue density whose medial edge runs parallel with the chest wall

Pleural plaques

Pleural plaques are benign fibrotic lesions that often calcify,[2] and they are most commonly found on the inferior parietal pleura adjacent to ribs and on the diaphragm. They are usually a sign of previous exposure to asbestos, though they can be associated with previous chest trauma (haemothorax), pleural infection, or artificial pneumothoraces used for the treatment of tuberculosis. Pleural plaques are a benign condition and not premalignant; therefore, in the absence of pleural fluid or thickening they do not themselves require regular follow-up. As pleural plaques indicate previous exposure to asbestos, patients are at risk of developing other conditions caused by exposure to asbestos such as mesothelioma and asbestosis. Patients should therefore be advised that if they develop persistent chest pain or breathlessness they should seek medical help. In England and Wales, patients do not receive compensation if pleural plaques alone are detected (although this was the case before 2007); rarely, pleural plaques may be so extensive that they cause restrictive

LEARNING POINTS

- Pleural thickening has benign and malignant causes. Malignant causes include mesothelioma and metastatic disease
- Computed tomography (CT) with intravenous contrast material is the main imaging modality for diagnosis
- Pleural plaques alone need no further follow-up
- The CT features that make malignancy more likely are pleural thickening >1 cm, nodularity, and extension on to the mediastinal surfaces
- Tissue is generally needed to confirm the diagnosis; several biopsy techniques are available

Causes of pleural thickening

	Focal	Diffuse
Benign	Pleural plaque Pleural fibroma Localised changes after radiotherapy Apical pleural thickening	Diffuse pleural thickening associated with asbestos exposure Infection usually secondary to empyema or tuberculosis Inflammation secondary to connective tissue diseases, drugs, etc
Malignant	Pleural metastasis Mesothelioma Lymphoma	Mesothelioma Pleural metastatic disease (usually adenocarcinoma—primaries include lung, breast, and ovarian cancer) Lymphoma

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Previous articles in this series
- Investigation of acute knee injury (*BMJ* 2012;344:e3167)
- Investigating the solitary pulmonary nodule (*BMJ* 2012;344:e2759)
- Investigating focal liver lesions (*BMJ* 2012;344:e657)
- Suspected early dementia (*BMJ* 2011; 343:d5568)
- Investigating suspected subarachnoid haemorrhage in adults (*BMJ* 2011;342:d2644)

lung function, and in such cases the person may receive compensation.[3] In Scotland, however, patients receive compensation if pleural plaques alone are detected.

What should be the next investigation?

Computed tomography

Intravenous contrast enhanced computed tomography (CT) is the primary imaging modality for assessing pleural disease and thickening. Its main advantage is the ability to show the entire pleura in excellent detail. Newer multidetector CT scanners enable reformatting of high resolution images in several planes, which provides additional information about anatomical relations—for example, with the diaphragm—which can aid surgical planning. Disadvantages include a radiation dose to the patient and the need for administration of iodinated contrast material, which is contraindicated in patients with poor renal function and allergy to contrast material. Overt signs of malignancy on CT scans include chest wall and bony invasion as well as metastatic disease. CT features suggestive of malignancy include circumferential pleural thickening (sensitivity 41%, specificity 100%), parietal pleural thickening >1 cm (36%, 94%), nodularity (51%, 94%), and mediastinal pleural involvement (56%, 88%).[4 5] The British Thoracic Society's guidelines for the management of mesothelioma suggest consideration of a 60 second delay to scan time after the administration of intravenous contrast material as this may improve the enhancement and demonstration of pleural disease.[6] Computed tomography (in combination with the pathological findings) is also helpful for the staging of malignant pleural mesothelioma; this is described in detail elsewhere.[7]

Ultrasonography

Ultrasonography is an excellent modality for detecting fluid collections in the pleural space.[8] However, it is less sensitive for the detection and characterisation of pleural thickening,[9] for which computed tomography is the best imaging modality. Ultrasonography may fail to detect pleural thickening that is <1 cm in thickness and is best used for detecting soft tissue when accompanied by fluid.[9] A small study has shown ultrasonography to have 73% specificity and 100% sensitivity for pleural malignancy when pleural nodules or thickening are seen with pleural fluid.[10]

Magnetic resonance imaging

Pleural malignancy enhances avidly with gadolinium based contrast, and the features used for distinguishing benign and malignant disease are similar to those used in computed tomography. Studies have suggested that magnetic resonance imaging has a sensitivity of 98-100% and a specificity of 92-93% for detecting pleural malignancy when compared with computed tomography.[2 5] However, now that recent CT technology can produce excellent multiplanar reconstructions, the role of magnetic resonance imaging is currently limited[6] as it is less available and more expensive compared with computed tomography. It can be used in evaluating local tumour extension where computed tomography is inconclusive and in those patients who cannot have iodinated contrast material. It also has the advantage of not carrying a radiation dose.

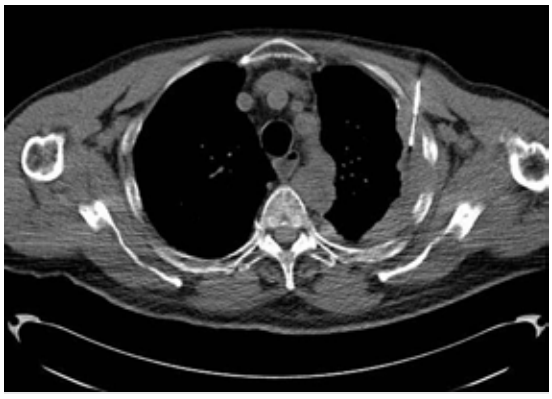

Fig 2| CT guided biopsy of left sided pleural thickening

Positron emission tomography computed tomography

Positron emission tomography computed tomography (PET-CT) combines the administration of radiolabelled biological molecules, usually glucose, with computed tomography. Metabolically active tissue is demonstrated, and as tumours are generally metabolic, PET-CT has a high sensitivity for detecting pleural malignancy. The technique is not used routinely for investigating patients with pleural thickening, but there is increasing evidence of its efficacy in differentiating benign from malignant disease. A study of 64 patients found a sensitivity of 96.8% and specificity of 88.5% for detection of malignant disease.[11] It may also have a role in determining prognosis and monitoring response to treatment.[12]

How should pleural thickening be biopsied?

A blind pleural biopsy, such as the Abrams needle technique, is an outdated procedure with low sensitivity and a high complication rate.[13] It has been superseded by image guided biopsy or thoracoscopy.

Biopsy under ultrasound or CT guidance

Guided biopsy is done under local anaesthesia by radiologists and involves obtaining core biopsies using computed tomography or ultrasonography for guidance. It has 87% sensitivity for malignancy compared with 47% for non-image guided biopsy[13] and a <1% mortality rate.[14] The commonest complication of lung biopsy is pneumothorax, which occurs in 20% of cases, of which a chest drain needs to be inserted in only 3%.[13] However, rates are much lower than this for pleural thickening as no aerated lung has to be crossed (fig 2). Other complications such as pulmonary haemorrhage and haemoptysis occur in around 5%. If pleural thickening measures greater than 0.5 cm and is in a suitable position then CT or US guided biopsy would be the first line investigation.

Medical thoracoscopy

Medical thoracoscopy, done under conscious sedation by physicians, allows the pleural surfaces to be visualised and guided biopsies to be taken. It has 90% sensitivity and 96% specificity for malignant disease[15 16] and low morbidity and mortality rates.[17] In the absence of pleural fluid thoracoscopy can be done but requires an experienced operator to induce a pneumothorax.[18] A thoracoscopy without pleural fluid is usually considered only if the pleural thickening is not suitable for biopsy done under ultrasound or CT guidance.

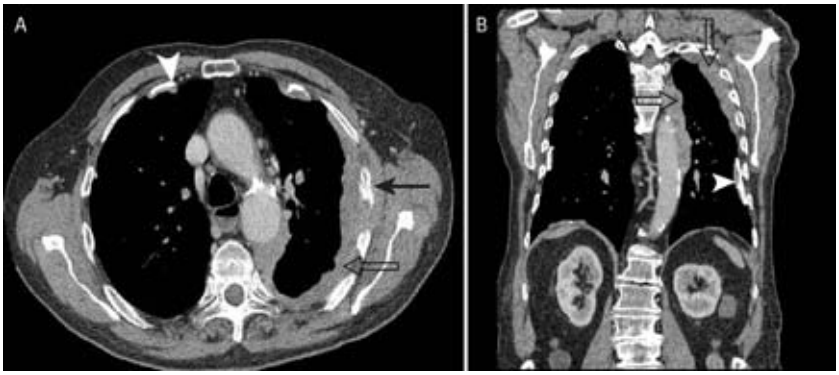

Fig 3 | Axial (left) and coronal (right) computed tomograms of the thorax after administration of intravenous contrast material. These demonstrate pleural thickening (open black arrows), which extends on to the mediastinal surface, and rib destruction (black arrow). Note the presence of incidental pleural plaques (white arrowhead)

Surgery

Biopsy under general anaesthesia is usually considered a last resort and carries the highest risk, including a significant risk of a tumour seeding, but may be required if other methods of obtaining tissue are not suitable.[14] Tissue may also be obtained if the patient has surgical resection; however, the MARS study (a randomised controlled trial examining the outcomes in patients having extrapleural pneumonectomy and trimodality treatment) did not show a survival benefit in these patients.[19]

Outcome

The patient had computed tomography, which confirmed the presence of bilateral pleural plaques and pleural thickening on the left hand side, which was thicker than 1 cm, showed evidence of nodularity, and extended on to the mediastinal surface (fig 3). A CT guided pleural biopsy was done (fig 2), and histology testing showed malignant mesothelioma of sarcomatoid type. This was staged as T4N0M0. He subsequently had palliative radiotherapy and chemotherapy.

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A PATIENT'S JOURNEY

How no one acted when they should have

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This is one of a series of occasional articles by patients about their experiences that offer lessons to doctors. The *BMJ* welcomes contributions to the series. Please contact Peter Lapsley (plapsley@bmj.com) for guidance.

A retired maxillofacial surgeon had a revision of a 14 year old hip replacement, after which he had an unpleasant series of side effects, due largely to poor clinical care

In June 2011, aged 79, I was admitted to the orthopaedic ward of my local NHS teaching hospital for a revision of a loose 14 year old hip replacement. I was clerked in by an orthopaedic associate specialist but no anaesthetist arrived.

Next morning I met the consultant anaesthetist and the surgeon in the anaesthetic room. I recognised the anaesthetist as having given me a spinal block for a previous knee replacement. He proposed a combined spinal and epidural block with heavy sedation, to which I agreed.

I was returned to the ward about 4 pm. The orthopaedic associate came to tell me that all had gone as planned, and he confirmed my ability to raise and extend my legs. When the nurse started my routine checks I told her that I was completely numb in and around my groin. Later my wife was present when I again told nurses of my numbness. We were reassured by their explanation that the area was the last to recover and this might be delayed by the epidural pain relief. Even then I remembered I had not experienced this when I had my knee done.

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- Klinefelter's syndrome—a diagnosis mislaid for 46 years (*BMJ* 2012;345:e6938)
- Kallmann syndrome (*BMJ* 2012;345:e6971)
- Restless legs syndrome (*BMJ* 2012;345:e7592)
- Thoracic outlet syndrome (*BMJ* 2012;345:e7373)
- Visual agnosia (*BMJ* 2012;345:e7342)

A DOCTOR'S PERSPECTIVE

This clearly has been a very distressing case for Mr Hopkins and it raises some interesting points.

A central neuraxial block such as a spinal and/or an epidural can be used as a sole anaesthetic technique or as an adjunct to general anaesthesia to facilitate postoperative pain management. In this case, a combined spinal and epidural technique was used to facilitate intraoperative anaesthesia and postoperative analgesia. In long operations such as revision of hip replacement, heavy sedation or a general anaesthetic is usually also administered.

The dense numbness that Mr Hopkins experienced in the perineal area is indeed normal after a spinal block, and it is the last area to return to normal, but sensation should return within six to 10 hours maximum. The concurrent or independent use of a lumbar epidural does complicate matters. Although it is routine practice in most hospitals to assess leg weakness hourly after an epidural is sited (to try to diagnose as early as possible any cases of nerve or cord damage associated with, for example, a haematoma or abscess), sensation is less commonly checked.

Clearly on catheterisation for a painless full bladder, it would not have been unreasonable to have questioned whether this was to be expected from an epidural. As the frequency of epidural use has dropped over the years, nurses' and juniors doctors' exposure to these advanced techniques has also diminished.

It seems that about 24 hours after returning to the ward, the epidural catheter had come out. It is fair to say that after this point, any residual weakness in the legs or numbness in the legs or perineum should have been attributable to nerve damage, until proved otherwise. Unfortunately, it was over 48 hours after this point at which the possibility of a cauda equina syndrome was considered.

If neuraxial haematoma is suspected, then magnetic resonance imaging must be done as soon as possible, with an aim for surgical decompression ideally within eight hours to achieve the best neurological outcome. Thus, appropriate procedures must be in place for monitoring patients' post-spinal anaesthetics or those with epidurals in situ, with associated teaching packages for the ward staff.

Serious complications after both spinals and epidurals remain statistically very low, with the overall incidence of permanent (lasting more than six months) nerve damage reported as between 1 in 25 000 and 1 in 50 000.[2]

This is little comfort to Mr Hopkins.

Gavin Werrett, consultant anaesthetist

When it was time to sleep I advised the nurse that I had not passed urine but had no desire to do so. As I was on intravenous fluids, I was surprised when I woke the next morning at about 6 am that I didn't need a bottle. However, I could palpate a suprapubic swelling, which I assumed was my bladder, even though pressure on it was not uncomfortable. I asked for a doctor as I believed I needed catheterisation. About 8 am a junior doctor arrived. After palpating my abdomen he called for a catheter tray and relieved me of about 1.5 litres of urine, although I was unaware of any manipulation. Leaving me with an indwelling catheter attached to a bed bag, he departed without, it turned out, making any record in the notes or reporting to a senior doctor.

As it was a Friday, I expected a visit from the orthopaedic team, but only a specialist nurse in pain relief came to see me. I had minimal discomfort. In the afternoon I found I was lying on a wet sheet, with copious mucus covering my buttocks. As the nurses changed the sheet they discovered that the epidural tube had come out of my back which must have contributed to the wetness. That evening in my wife's presence I told the nurses my numbness was unchanged despite the loss of the epidural at an unknown time. One nurse suggested I would soon recover if she got into bed with me. I was amused, my wife was not.

On Friday night I could not sleep and requested night sedation, for which the duty doctor was called. When he arrived, after considerable delay, he was the same doctor who had passed my catheter. I learnt that he was the general surgical foundation year 2 doctor covering orthopaedics out of hours.

On Saturday morning, my only visitors were a specialist nurse from orthopaedics, who said he would ring the anaesthetist, and the pain relief nurse. Subsequently I learned the anaesthetist was on annual leave and unavailable. By now I was aware of the uncomfortable numbness involving my buttocks and posterior thighs. Gas bubbles escaped in the mucus between my legs.

Sunday continued in a similar vein; I was confined to bed by foot pumps and without any medical visit. When my wife arrived in the late afternoon I told her of my increasing concern at being unable to raise medical interest in my condition, the aetiology of which had to be more complicated than delayed epidural recovery. My wife spoke to a houseman, who told her my problems would be dealt with on Monday.

When my wife returned home, she rang our daughter, a consultant rhinologist. She rang the ward and was told that the registrar could not be called because I was not an emergency. My daughter fortunately was able to ring the orthopaedic consultant at home to ask him, "What is wrong with my father?" About 9 pm, three junior doctors came in turn, armed with pins to contemplate the possibility of a cauda equina syndrome. On Monday morning I had a visit from my orthopaedic consultant, who ordered that I be starved in case of the need for further surgery. I then had an emergency magnetic resonance scan, and in the early evening the report was reviewed by a spinal surgeon, who told me of a haematoma lying posteriorly at L1-L2 and that he considered surgical decompression was not justified. The window of opportunity to bring a possible early recovery thus remained closed.

Space limitation does not allow the description of the unpleasantness of the two and a half weeks I spent in hospital. Care of incontinence was not on the same level as the kindness of the nurses. The urinary bed bag was changed to a flip-flow valve and, after discharge, to intermittent catheterisation. Both were easy to forget in the absence of bladder sensation, which led to several "accidents" when abdominal pressure was raised—for example, when standing or coughing.

After discharge, four episodes of haematuria, one of which caused an emergency admission, resulted in prolonged antibiotics and an attempt by my excellent urologist to get me off clopidogrel. Eight months after the operation I have stopped using catheters. My bladder started to function weakly at

about seven weeks after surgery and gradually improved. It remains weak and lacks sensation. When I stand, a pain-like sensation tells me I must urinate. Fifteen minutes later I can pass almost the same amount again.

The back end remains a problem. I am not incontinent but I have some problems differentiating between gas and solids. The district incontinence service introduced me to the self administered Peristeen rectal washout system, paid for, as were the urinary catheters, by my general practice's budget. Now that the rectal catheter has been redesigned and the balloon does not burst, this is an excellent system, which prevents "accidents." However, the process is time consuming and there is a learning curve. I cannot empty normally but require the combination of abdominal muscle contraction, manual compression, and agitation of the abdomen. I use the Peristeen system almost every day.

The profound saddle anaesthesia has made a partial recovery. The perineal and buttock areas are dulled and paraesthesic. The perianal tissues have sensation but this is abnormal and the area feels isolated from the surrounding tissue. There is no sensation produced by the passage of solids.

Initially I seemed to be sitting on a log that was very uncomfortable. Now the sensation is of sitting on a leather strap with my tuberosities unprotected. The literature states the elderly male has a poorer prognosis for a full recovery with this presentation. Time will tell. Happily my hip functions reasonably well and I am due to have a knee replacement in about a month's time.

NHS healthcare delivery is rarely out of the media and there are frequent reports of misadventure.[1] Medical students are apparently trained in teamwork and communication but not in the care of the ill. The presidents of the Royal College of Physicians and the Royal College of Surgeons have warned of the effect on junior doctors of the European Working Time Directive, shift work, days off, reduced hours, and clinical experience. Doctors' numbers may have increased but not their expertise. Similar problems exist in the nursing profession as nurses train to do the work that was previously the remit of doctors while leaving nursing to healthcare assistants.

The secretary of state for health has recognised there are problems in the NHS at weekends owing to the absence of senior doctors. My recent difficulties result from all of the above. Additionally there is a failure to train doctors and nurses adequately. If the discipline of anaesthesia recognises that central nerve blocks can cause complications with serious implications for patients, all of the staff who provide postoperative care must be trained to recognise them. Reluctance to contact senior staff for advice must be eliminated. Note taking and communication between nurses, between nurses and junior doctors, and between junior and senior doctors require rethinking. If consultants stop doing ward rounds and supervising junior doctors, it is necessary to redefine who is ultimately responsible for patient care. Instead of spending vast sums settling negligence claims (£1bn in 2010), would this money not be better used to provide increased staffing and training, particularly for out of hours care? As a non-medical friend said to me, "If they can't even look after you, who will they look after?"

Competing interests: None declared

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ANSWERS TO ENDGAMES, p 40

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ANATOMY QUIZ

Ultrasound scan of the right upper quadrant of the abdomen, transverse plane

A: Right lobe of the liver
B: Right kidney
C: Fat in the right renal sinus
D: Gallbladder
E: Hepatorenal recess
F: Inferior vena cava

STATISTICAL QUESTION

Analysing case-control studies: adjusting for confounding

Statements *a*, *c*, and *d* are true, whereas *b* is false.

PICTURE QUIZ

A sinister cause of shoulder pain, with numbness and weakness in the ipsilateral hand

1 The radiograph shows an area of opacification in the right lung apex but is otherwise normal.

2 A superior sulcus tumour (Pancoast tumour) is the most likely diagnosis. The differential diagnoses include another primary thoracic tumour and a metastatic deposit of cancer from a different origin.

3 A percutaneous needle biopsy (our patient's mass consisted of squamous cells; combined with the clinical features and imaging we diagnosed a Pancoast tumour). Further investigations (for staging) include positron emission tomography-computed tomography and magnetic resonance imaging of the thoracic inlet.

4 The most common symptom is pain in the shoulder, which often radiates to the arm, scapula, and the ulnar surface of the hand. Other symptoms include weakness and atrophy of the intrinsic muscles of the hand, paraesthesia of the ulnar aspect of the ipsilateral arm, and Horner's syndrome.

5 Treatment depends on tumour stage, the patient's fitness, and the patient's wishes. Refer all cases to a lung cancer multidisciplinary team, which will make recommendations about management. Offer patients with localised cancers chemoradiotherapy (as neoadjuvant treatment) and surgical resection, or continuous hyperfractionated accelerated radiotherapy. Treatment of advanced non-small cell lung cancer is palliative, with the aim of improving symptoms and quality of life.