



# Abdominal mass/hepatosplenomegaly

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## Introduction

The identification of an abdominal mass can be a stressful time for the patient. It is imperative to carefully formulate a differential diagnosis based upon the abdominal findings and the clinical context: *patients rarely present with just an abdominal mass*. A prompt diagnosis may enable the patient to quickly be reassured of a benign cause or rapidly provided with access to appropriate specialist care and treatment.

No table can be entirely inclusive on this topic; we are confident you could recall additional causes under each aetiological heading listed in Table 1.1.

**Scenario 1.1**

*A 75-year-old woman presents on a Saturday to the emergency department; struggling to cope at home, she reports a 4-week history of progressive abdominal swelling. During this time she has not been eating well and has lost a significant amount of weight. On examination she is cachectic and jaundiced; she has ascites and an epigastric mass which is 5 cm in diameter, craggy in nature and tethered to the underlying structures.*

This patient has been deteriorating for some time and unfortunately (*but not uncommonly*) presented out of hours. She probably has advanced cancer, and this should be recognized by the admitting doctor from the outset; the differential diagnosis at this early stage is wide and so this diagnosis should not be given to the patient without further confirmation.

## History

A careful and comprehensive history is required. In particular:

*Systemic enquiry ('red flags' for malignancy)*

- Fever and night sweats
- Weight loss (unintentional  $\geq 3$  kg)
- Dysphagia
- Change in bowel habit
- Blood per rectum
- Visible haematuria
- Intermenstrual bleeding
- Anorexia

*Family history*

- Malignancy
- Polycystic kidney disease

*Drug history*

- Prescribed
- Over the counter

*Social history*

- Alcohol consumption (binges?)
- Travel history, including areas visited (rural or urban), activities engaged in (safaris, water sports, etc.); did they take malaria prophylaxis and have the recommended vaccinations?
- Sexual history (protected, high risk partners?)
- Intravenous drug use (clean needles or shared?)
- Occupation (e.g. sheep farming, associated with hydatid disease)

*Past medical history*

- Tuberculosis
- Diverticular disease
- Inflammatory bowel disease
- Solid tumours or haematological malignancy

The presence of 'red flags' requires the exclusion of malignancy as a priority when planning investigations, bearing in mind that they can clearly be compatible

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**Table 1.1** Common and important causes of an abdominal mass by aetiology

Aetiology	Diagnosis
Neoplastic	<i>Malignant</i> <i>Primary malignancy</i> <ul style="list-style-type: none"><li>• Pancreatic</li><li>• Colorectal</li><li>• Hepatocellular carcinoma</li><li>• Gastric</li><li>• Renal</li><li>• Ovarian</li><li>• Endometrial</li></ul> <i>Secondary/metastatic disease</i> <ul style="list-style-type: none"><li>• Hepatic metastasis</li><li>• Lymphadenopathy (testicular spread to para-aortic nodes)</li><li>• Splenomegaly (CML)</li><li>• Peritoneal spread</li></ul> <i>Benign</i> <ul style="list-style-type: none"><li>• Uterine fibroids</li><li>• Lipoma (anterior abdominal wall)</li></ul>
Infective	<i>Abscess</i> <ul style="list-style-type: none"><li>• Diverticular</li><li>• Appendix</li><li>• Empyema of the gallbladder (may progress from cholecystitis)</li><li>• Crohn's disease</li><li>• Liver abscess (most commonly caused by ascending pathogens from biliary tract)</li></ul> <i>Hepatomegaly</i> <ul style="list-style-type: none"><li>• Malaria</li><li>• Leptospirosis</li><li>• Viral hepatitis</li></ul> <i>Splenomegaly</i> <ul style="list-style-type: none"><li>• Immune hyperplasia (bacterial endocarditis, EBV, etc.)</li></ul>
Vascular	<i>Abdominal aortic aneurysm</i> <i>Hepatomegaly</i> <ul style="list-style-type: none"><li>• Congestion (right heart failure/tricuspid regurgitation, may be pulsatile)</li><li>• Budd–Chiari syndrome (hepatic vein occlusion, normally by thrombus)</li></ul> <i>Splenomegaly</i> <ul style="list-style-type: none"><li>• Portal hypertension (liver cirrhosis)</li></ul>
Inherited	<i>Renal</i> <ul style="list-style-type: none"><li>• Autosomal dominant polycystic kidney disease (ADPKD)</li></ul> <i>Hepatomegaly</i> <ul style="list-style-type: none"><li>• Riedel's lobe (normal variant)</li><li>• ADPKD with hepatic cysts ( &gt; )</li></ul>

Mechanical obstructive	<i>Intestinal distension</i> <ul style="list-style-type: none"><li>• Obstruction</li><li>• Constipation</li></ul> <i>Renal enlargement</i> <ul style="list-style-type: none"><li>• Hydronephrosis (severe)</li></ul> <i>Bladder</i> <ul style="list-style-type: none"><li>• Urinary retention</li></ul>
Degenerative	<i>Hernia</i> <ul style="list-style-type: none"><li>• Umbilical</li><li>• Paraumbilical</li><li>• Spigelian</li></ul>
Inflammatory	<i>Pancreatic pseudocyst</i> <i>Hepatomegaly</i> <ul style="list-style-type: none"><li>• Sarcoidosis</li><li>• Early cirrhosis with portal hypertension (with splenomegaly)</li><li>• Non-alcoholic steatohepatitis</li></ul> <i>Splenomegaly</i> <ul style="list-style-type: none"><li>• Sarcoidosis</li><li>• Systemic lupus erythematosus</li></ul>
Metabolic	<i>Hepatomegaly</i> <ul style="list-style-type: none"><li>• Amyloidosis</li></ul>
Iatrogenic	<i>Renal transplant</i> (right or left iliac fossa mass with scar overlying) <i>Implants</i> <ul style="list-style-type: none"><li>• Buscopan pump (used in multiple sclerosis)</li><li>• Gastric pacemaker</li></ul> <i>Embedded peritoneal dialysis catheter</i> (inserted ready for externalizing when dialysis is required) <i>Incisional hernia</i>
Idiopathic	<i>Splenomegaly</i> <ul style="list-style-type: none"><li>• Idiopathic thrombocytopenic purpura</li></ul>
Physiological	<i>Pregnancy</i>

with other aetiologies: a patient with a liver abscess will develop anorexia and lose weight, so initially it is important to keep an open mind and equally open differential diagnosis. Try not to increase the patient's anxieties at this early stage.

Examination

Perform a full examination of the patient, including: respiratory, cardiovascular and neurological examinations alongside the abdominal examination. Many pathologies resulting in an abdominal mass or organomegaly will have manifestations in other systems

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as well. There are often some subtle clues to the aetiology which may be present, so take care to examine for *anaemia, splinter haemorrhages and lymphadenopathy*.

Once organomegaly or a mass has been identified, examine it carefully and clearly document:

- site (consider which organ it may be, or is associated with)
- size
- shape
- nature (smooth or craggy, soft or firm)
- mobility
- reducibility (if it could represent a hernia)
- appearance of the overlying skin (scar, stretch marks).

In addition a *digital rectal examination and urinalysis are essential elements of the clinical examination which must not be forgotten*.

After completing the examination you may have findings that require you to go back and revisit the history. Patients rarely mind a few additional questions asked after or during the examination; it shows their physician is interested and considering their case carefully.

## Investigation

### Blood tests

- FBC
- U&E
- LFT
- CRP
- Calcium
- Clotting screen
- Tumour markers (use selectively, with caution)
- Blood film (if haematological malignancy is suspected)
- If infective symptoms
  - Blood cultures
  - Urine cultures
  - Stool culture (if diarrhoea)
  - According to history; malaria screening, HIV, hepatitis serology, CMV, EBV

These can be helpful in narrowing the differential diagnosis and identifying the unwell from the clinically stable patient.

- Anaemia is *never normal* and always has a cause or causes; it should prompt haematinics (B<sub>12</sub>, folate, iron studies) to be checked and acted upon.

- Raised inflammatory markers suggest infection, but are significantly raised in IBD and frequently elevated with neoplasia.
- Low albumin, raised platelet count and anaemia all suggest neoplasia, but are compatible with significant inflammation of other causes.
- Tumour markers *should be used with caution in assisting initial diagnosis* as none are specific for a given malignant disease; they are more useful for monitoring response to treatment:
  - CA19-9, pancreatic cancer
  - CA125, ovarian cancer
  - CEA, colorectal cancer.

## Radiological imaging

Always give careful consideration to deciding the most appropriate initial imaging modality (Table 1.2). The aim is to gain *accurate diagnostic information in the shortest time frame with the minimum of risk to the patient*. The modality selected will depend upon the differential diagnosis formed, which guides *the question you are asking of the imaging*:

- Has this well 45-year-old woman with nodular hepatomegaly whose grandfather died of renal failure got ADPKD with multiple liver cysts?
- *Ultrasound scanning will rapidly answer this question with virtually no risk to the patient.*
- In the 53-year-old male smoker who drinks 50 units of alcohol a week, who has lost 6 kg of weight over 2 months and examination revealed craggy hepatomegaly and anaemia, the question needing an answer is 'has he got cancer?' and if so 'what is the primary and has it metastasized?'
- *CT chest, abdomen and pelvis with contrast is the first-line imaging modality of choice, giving diagnostic and prognostic information, also clarifying potential biopsy sites for histological confirmation.*

It is not the case that USS is more sensitive for liver lesions than CT, but it does avoid exposing the patient to the risks of ionizing radiation and iodinated contrast.

## Surgical exploration

Despite modern imaging modalities, in certain circumstances surgical exploration by laparotomy or more frequently laparoscopy into the cause of an abdominal mass is still warranted. This needs careful

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**Table 1.2** Comparison of different abdominal imaging modalities

Modality	Advantages	Disadvantages
Ultrasound	Excellent in experienced hands Modality of choice for RUQ pain, acute jaundice and pelvic masses	Limited by body habitus Limited by operator experience Limited information on metastatic spread or tissue invasion Images not standardized and more difficult to interpret for someone who has not performed the scan
CT	Quick to perform Readily available Wealth of experience in interpretation Can detect metastatic spread, and tissue invasion Easily reviewed by multidisciplinary team (MDT)	High dose of ionizing radiation Limited resolution compared to MRI Iodinated contrast exposure (see Chapter 39)
Abdominal X-ray	Good acute investigation to exclude obstruction and toxic bowel dilatation	If normal may need further imaging Will often require CT imaging if abnormal
MRI	Excellent diagnostic detail	Not readily available Difficult to interpret (limited expertise) Slow image acquisition Claustrophobic patients often struggle with the examination

Adapted from the American College of Radiology, 2010 [1].

consideration and discussion between the physician, surgeon and patient.

Strengths

- Direct visualization
- Potential to biopsy abnormal tissues if seen
- Can proceed to definitive operation (if included in the consent process)

Weaknesses

- Risk of general anaesthesia and paralysis
  - Operative risk (infections, bleeding, pain, etc.)
  - Expensive
  - Emotional trauma and anxiety provoking
- Although laparoscopy can be an excellent tool in the evaluation of the abdominal mass, it is rarely required.

Endoscopy

Endoscopy (*gastroscopy and colonoscopy*) is usually performed in response to imaging showing a suspected lesion of gastric or colonic origin. It is the only investigation to provide direct visualization of the gastrointestinal mucosa. *Gastroscopy* is the first-line investigation for patients presenting with dysphagia or an epigastric mass, alongside CT imaging of the abdomen. Endoscopic ultrasound can provide excellent

definition of mucosal and deeper infiltration of cancers. It is often the modality of choice for determining operability of cancers, particularly oesophageal.

Acute management of ‘suspected cancer’

Scenario 1.1 continued

*A CT chest, abdomen and pelvis is requested. The CT imaging reveals a tumour at the head of the pancreas (Figure 1.1), which was invading local structures. Metastases are evident in the liver and a large amount of ascites is present. Following the CT scan the patient and her son were keen to know what it showed. It was explained that a pancreatic mass was seen and this might represent cancer; however, it was made clear to them that at this point the diagnosis was not confirmed, but further results would soon be available and diagnostic certainty was needed before any specific treatments could be planned. Abdominal paracentesis was performed and ascitic fluid sent for urgent cytological examination. The next day the cytology from her ascitic fluid had been processed and showed adenocarcinoma cells.*

It is clear from the previous imaging discussion that a contrast-enhanced CT of chest, abdomen and pelvis was the most appropriate first-line imaging. This was likely to reveal the nature of the primary tumour

with information about distant spread. When cancer is suspected, cytology or histology should always be obtained if possible to confirm the diagnosis and classify the type of cancer. *Beware the many cancer mimics which have caused physicians to mistakenly diagnose cancer from CT scans!*

For this patient urgent sampling of the ascitic fluid for cytology was the easiest and safest way to try to confirm the diagnosis.

Ensuring the diagnosis is followed up – the MDT approach

NICE recommends that every patient with a new diagnosis of cancer is reviewed by an MDT (multidisciplinary team) meeting. Such meetings (e.g. upper GI, lower GI, gynaecological) commonly involve the relevant physician, surgeon, radiologist, pathologist, oncologist and oncology specialist nurse. The patient’s current performance status and co-morbidities are considered alongside the CT imaging, cytology and pathology results. The tumour is staged according to the TNM classification (Box 1.1). Initial treatment is decided: surgery, chemotherapy, radiotherapy or symptom control and palliative care.

**Box 1.1 TNM staging of cancer**  
**Tumour** – T1–T4 describes different levels of local invasion specific to the type of tumour  
**Nodes** – N0–N3  
**Metastases** – M0/M1 indicates presence or absence of metastases

Within the NHS, quality targets exist for the investigation of symptoms suggestive of a cancer diagnosis and subsequent treatment [2–4].

- All patients referred with suspected cancer from a GP have a maximum wait of 2 weeks from referral to see a specialist.
- All cancer patients should wait no more than one month (31 days) from diagnosis to first definitive treatment.
- A maximum 2-month (62-day) wait from urgent GP referral for suspected cancer to first definitive treatment for all cancers.

If a patient is referred to the acute medical take with suspected cancer, the admitting team should always consider whether outpatient investigation would be

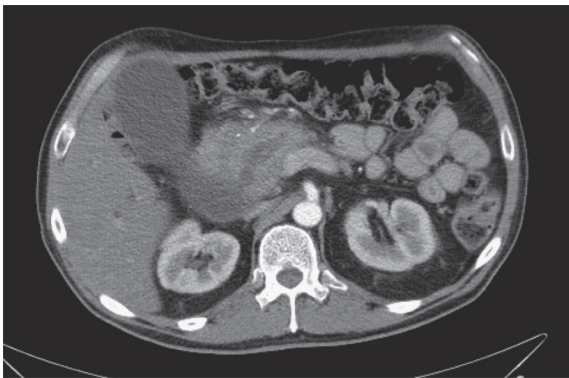


Figure 1.1

more appropriate. Systems are in place to ensure the rapid outpatient investigation of patients with suspected malignancy. Admission may be required for associated problems:

- symptom control, e.g. pain, breathlessness
- inability to cope in their current state and home circumstances
- emergency complication, e.g. acute cord compression.

If they are to be discharged, *robust arrangements for appropriate investigations and follow-up should be in place at discharge*; this should not impede early discharge.

Communicating bad news

It is frequently the responsibility of the medical team looking after the patient on the ward to communicate the prognosis and treatment plan to the patient. It is devastating for most patients and relatives when a diagnosis of cancer is given and this must be given with the utmost sensitivity. Once the diagnosis has been confirmed, it should be communicated in as timely a manner as possible. In this setting some patients are fully aware of the likely cause of their recent ill health, others are either in denial or blissfully ignorant of the true situation.

Communication of a cancer diagnosis requires a thoughtful and empathic approach. In some situations the hospital palliative care team and oncology nurse specialists can be called upon to help support these conversations. They can offer continuity, accessibility and time to patients and their families unrivalled by physicians; their role should never be underestimated and they should be involved as early as possible in such cases.

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The principles of breaking bad news

- Choose an appropriate location; quiet and unlikely to be disturbed.
- Allow time to perform this vital role properly without rushing the patient or their relatives.
- Ensure that you know who you are speaking to and have the patient's consent.
- Check prior knowledge.
- Send a 'warning shot'.
- Explain in clear and easy to understand language the diagnosis and the likely outcome.
- Do not shy away from words like 'cancer'; all too often 'tumour' or other medical terminology is used, which clouds the situation.
- Ensure that the family and patient have a follow-up plan and have a point of contact (often the specialist cancer nurse).

Communication should be honest and upfront at all times. There is often heightened emotion which can be represented as anger towards medical and nursing staff in this situation; this should be handled sensitively and not taken personally.

Imagining yourself in the patient's or the relative's shoes is a distressing and uncomfortable exercise, but it clearly helps empathy with the patient and better appreciation of their reactions. The need for simple clear unambiguous language in delivering this news can never be overstated. Repetition of the message can also be valuable as it can be 'blocked out' initially by patients or their relatives.

Enlargement of liver and/or spleen

Hepatosplenomegaly

The commonest cause of hepatosplenomegaly in the UK is liver cirrhosis with portal hypertension (Table 1.3). This can present acutely and is often seen by acute physicians and intensivists.

The acute complications seen in these patients are generally related to the portal hypertension:

- Ascites with associated risk of spontaneous bacterial peritonitis (see Chapter 3)
- Oesophageal and gastric varices, which can bleed torrentially (see Chapter 26)
- Hepatic encephalopathy, certainly under-recognized at admission (see Chapter 36)
- Hepatorenal failure, less common at presentation but also indicates a poor prognosis; bear in mind

that hypovolaemia is the commonest precipitator of acute kidney injury in this patient group.

In addition, significant hepatosplenomegaly is commonly encountered in the context of both myeloproliferative and lymphoproliferative disorders. Outside the UK, the following causes predominate:

- malaria
- tuberculosis
- schistosomiasis
- kala-azar.

Isolated hepatomegaly

Hepatomegaly commonly results from cirrhosis (usually alcoholic in origin), malignancy and congestion due to cardiac failure (Table 1.4). It can be misdiagnosed in patients with hyperinflation due to COPD, due to downward displacement of the liver by the diaphragm. It may arise acutely, when it is frequently painful and/or tender as in viral or alcoholic hepatitis, or chronically, and more commonly painless as in fatty liver or amyloidosis. The presence of hepatomegaly should be confirmed by ultrasound imagining, which will provide information regarding the liver architecture.

The size of the liver is not an indicator of the severity of disease as demonstrated in ADPKD where occasionally massive hepatomegaly occurs with minimal impairment of liver function.

Isolated splenomegaly

The spleen is the largest lymphoid organ at 7–10 cm in length, and only has efferent lymphatic vessels. It is generally only palpable when twice normal size. For this reason splenomegaly is often diagnosed on imaging rather than examination. The functions of the spleen should be recalled when managing the patient with splenomegaly.

*Splenic functions* include:

- reservoir of erythrocytes and thrombocytes, in case of significant haemorrhage
- destruction and recycling of old erythrocytes, life span ≈120 days
- immune function destroying many bacteria and viruses that enter the circulation, especially important for encapsulated bacteria: *Pneumococcus*, *Meningococcus*, *Haemophilus influenzae* type b (Hib), *Klebsiella*, salmonella and group B streptococcus
- haematopoiesis during gestation.

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**Table 1.3** Common causes of significant hepatosplenomegaly that may present on the acute medical take

Aetiology	Comments
<i>Portal hypertension</i> <ul style="list-style-type: none"><li>• Liver cirrhosis</li><li>• Budd–Chiari syndrome</li></ul>	Stigmata of chronic liver disease Rapid development of ascites with pain
<i>Myeloproliferative disorders</i> <ul style="list-style-type: none"><li>• Chronic myeloid leukaemia</li><li>• Myelofibrosis</li></ul>	
<i>Lymphoproliferative disorders</i> <ul style="list-style-type: none"><li>• Lymphoma</li><li>• Chronic lymphatic leukaemia</li></ul>	Associated lymphadenopathy
<i>Infectious</i> <ul style="list-style-type: none"><li>• Viral hepatitis</li><li>• EBV, CMV, toxoplasmosis</li><li>• Brucellosis</li><li>• Leptospirosis</li><li>• Malaria, schistosomiasis, kala-azar, tuberculosis</li></ul>	Stigmata of intravenous drug use Glandular fever like illnesses Farmers Environmental exposure to pathogen, jaundiced Patient travel history, outside of UK (although TB increasingly present in some UK communities)
<i>Metabolic</i> <ul style="list-style-type: none"><li>• Amyloidosis</li><li>• Gaucher's disease (glucocerebrosidase deficiency)</li></ul>	May have evidence of primary cause May be massive splenomegaly in patient with known diagnosis
<i>Blood dyscrasias</i> <ul style="list-style-type: none"><li>• Sickle cell disease</li><li>• Thalassaemia major</li></ul>	Characteristic features in patient with known diagnosis

*Thankfully for the asplenic patient it is not the seat of 'one's emotions or passions', as our predecessors thought!*

The cause of enlargement may be pathology requiring upregulation of these functions:

- *Increased turnover of red blood cells* as in inherited spherocytosis.
- *Increased immune activity* due to infection, most commonly in the UK due to glandular fever (Epstein–Barr virus), but the infective causes are numerous and include several tropical diseases:
  - malaria\*
  - leishmaniasis\*
  - trypanosomiasis
  - ehrlichiosis
  - brucellosis
  - typhoid fever.
- Abnormal immunoregulation in some patients with autoimmune conditions such as SLE and rheumatoid arthritis can also result in splenomegaly.
- *Haematopoiesis* and splenic enlargement can occur in adult life if the bone marrow is failing, as in myelofibrosis\*.

Other mechanisms (and examples) for development of splenomegaly include:

- *Infiltrative*: amyloid, sarcoidosis, metastases or in Gaucher's disease\*
- *Neoplastic*: lymphomas, chronic lymphocytic leukaemia (CLL) and chronic myeloid leukaemia\* (CML)
- *Congestive*: increased pressure within the venous system such as portal hypertension or more rarely splenic vein thrombosis.  
(\*May cause 'massive' splenomegaly >20 cm in length.)

**Acute complications of splenomegaly**

*Left upper quadrant pain*: this can be the presenting feature of patients with splenomegaly, most likely occurring as with most organ pain from rapid expansion or inflammation.

*Hypersplenism* is an abnormally high rate and premature destruction of circulating blood cells; this may result due to the underlying cause of the splenomegaly such as in chronic malaria or TB. The complications include:

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**Table 1.4** Common causes of significant isolated hepatomegaly that may present on the acute medical take

Aetiology	Comments
<i>Cancer</i> <ul style="list-style-type: none"><li>• Hepatocellular carcinoma</li><li>• Secondary deposits</li></ul>	Hepatoma may occur in context of cirrhosis Malignancy often causes tender nodular hepatomegaly
<i>Toxic</i> <ul style="list-style-type: none"><li>• Alcoholic hepatitis</li><li>• Alcoholic cirrhosis</li></ul>	Stigmata of alcoholic liver disease Acute alcoholic hepatitis is frequently painful
<i>Lymphoproliferative disorders</i> <ul style="list-style-type: none"><li>• Lymphoma</li><li>• CLL</li></ul>	Associated lymphadenopathy
<i>Infectious</i> <ul style="list-style-type: none"><li>• Viral infections, e.g. hepatitis, EBV</li><li>• Leptospirosis</li><li>• Hydatid disease</li><li>• Amoebic abscess</li></ul>	History of intravenous drug use or recent glandular fever like illness Environmental exposure to pathogen, jaundiced Sheep farmers, especially in Wales Tropical exposure
<i>Liver congestion</i> <ul style="list-style-type: none"><li>• Cardiac failure</li><li>• Budd–Chiari syndrome</li><li>• Tricuspid regurgitation</li></ul>	Other features of right heart failure Rapid development of ascites with pain Pulsatile liver
<i>Metabolic/granulomatous</i> <ul style="list-style-type: none"><li>• Haemochromatosis</li><li>• Amyloidosis</li><li>• Sarcoidosis</li></ul>	Slate grey skin pigmentation, hypogonadism, diabetes mellitus, arthritis, cardiomyopathy May be evidence of primary cause Lupus pernio, erythema nodosum, hilar lymphadenopathy, hypercalcaemia
<i>Miscellaneous</i> <ul style="list-style-type: none"><li>• Riedel’s lobe</li><li>• COPD</li></ul>	Tongue-like enlargement of right lobe of liver. Benign anatomical variant Spurious apparent enlargement due to downward displacement of diaphragm

- thrombocytopenia and potentially spontaneous bruising
  - anaemia
  - leucopenia and potentially serious secondary sepsis.
- Splenic rupture:* any patient with an enlarged spleen should be cautioned to avoid contact sports or heavy lifting activities which may increase the risk of splenic rupture. This will often cause a large intra-abdominal bleed and hypovolaemic shock, requiring urgent surgical assessment. This is a recognized cause of death in patients with EBV infection.

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# Abdominal pain

Stephen Haydock and Gareth Walker

## Introduction

Establishing the cause of abdominal pain can be diagnostically challenging in certain patient groups such as the elderly, women (especially if pregnant) and immunosuppressed. A successful approach should be based upon:

- understanding of the mechanisms of how pain is generated and/or perceived in relation to abdominal structures
- recognizing that patterns of presentation exist based upon the localization of the pain, its quality and radiation
- recognizing ‘red flags’ suggestive of significant and potentially life-threatening pathology
- recognizing that chronic abdominal pain is often functional in origin.

Abdominal pain is commonly described in terms of parietal, visceral or referred pain.

*Parietal pain:* afferent nerves originate in the parietal peritoneum and enter the ipsilateral dorsal root ganglion at the corresponding superficial dermatomal level. The pain receptors respond to irritation from infection, chemical or other inflammatory processes. It is usually perceived as a sharp, well-localized pain and results in localized tenderness and guarding that can progress to generalized peritonitis with abdominal rigidity and rebound tenderness.

*Visceral pain:* afferent nerves originate in the walls of hollow organs (responding to ischaemia, distension and inflammation) and capsules of solid organs (responding to capsular stretching). They enter the ipsilateral and contralateral cord at multiple levels. The pain is therefore poorly characterized (dull, cramping, aching) and poorly localized (being felt generally around the mid-line). It is interpreted at the cortical level as originating

from the approximate spinal cord level determined by the embryological origin of the organ:

- *Epigastric pain* due to organs of *foregut* origin (stomach, duodenum, biliary system)
- *Periumbilical pain* due to organs of *midgut* origin (small bowel, appendix, caecum)
- *Hypogastric/suprapubic pain* due to *hindgut* organs (colon, intraperitoneal parts of genitourinary system).

*Referred pain* is perceived as arising distant from its source and results from convergence of nerve fibres at the spinal cord. It also reflects embryological origins. Examples include:

- scapular pain due to biliary colic
- groin pain due to renal colic
- shoulder pain due to blood or infection causing diaphragmatic irritation
- ureteric obstruction causing ipsilateral testicular pain.

## Acute abdominal pain

### Scenario 2.1

You are asked to review a 75-year-old woman on the care of the elderly ward in the early hours of the morning. She has been awoken from sleep with a generalized abdominal pain. On examination she looks unwell, has a tachycardia of 130 beats per minute, irregularly irregular, with blood pressure of 100/70 having been 140/95 prior to the pain. She has mild generalized abdominal tenderness but no guarding or rebound tenderness. Her bowel sounds are increased. Her medication includes digoxin and warfarin for atrial fibrillation, prednisolone 30 mg for a recent diagnosis of polymyalgia rheumatica and regular tramadol.

## Chapter 2: Abdominal pain

This patient is unwell and requires urgent resuscitation and surgical referral. The suspicion, given the atrial fibrillation, generalized pain and increased bowel sounds, is of gut ischaemia. The absence of severe pain, tenderness, guarding and rebound is still consistent with this, especially in a patient on regular high dose steroids and analgesia. The presentation of an acute abdominal problem in the elderly is often less clear than in a younger individual and often results in significant delays in diagnosis and appropriate life-saving intervention.

### Clinical assessment of acute abdominal pain

*Some patients with acute abdominal pain may be very unwell and require urgent resuscitation; as always this is the priority.*

#### History

- When did the pain start?
- Did it come on suddenly or gradually?
  - Sudden onset suggests vascular occlusion (ischaemic gut, torsion), small tubular obstruction (biliary or ureteric colic).
  - Gradual onset suggests inflammatory disorder or bowel obstruction.
- Can you describe the quality of the pain? Dull or sharp, constant or waxing/waning, colicky?
  - Burning pain of dyspepsia
  - Tearing pain of dissection
  - Colicky pain of biliary or ureteric colic
  - Sharp localized pain of peritoneal inflammation.
- Where in abdomen do you feel it? Localized or generalized?
- Does it radiate anywhere? Shoulder, groin, back?
- Does anything make it better or make it worse?
  - Relation to meals (peptic ulcer better, biliary colic worse)
  - Relation to position and movement (agitation of renal colic, curled up, still patient with peritoneal inflammation).
- Has it changed in quality or location since onset?
  - Generalized visceral pain becomes localized as overlying peritoneum is inflamed.
  - Progression of renal stones down ureter.
  - Progression of a vascular dissection.
- Other associated symptoms:
  - cardiorespiratory (may present as upper abdominal pain)
  - vomiting
  - diarrhoea
  - rectal bleeding
  - dysuria, frequency
  - scrotal swelling, penile discharge
  - vaginal discharge
  - fever
  - weight loss
  - vaginal bleeding and discharge.
- Take a detailed medical, drug and social history. In particular note:
  - previous abdominal disease and surgery
  - recent sexual contact for females; was protection used?
  - alcohol intake
  - smoking history
  - previous surgery or recent instrumental procedures
  - immunosuppression due to HIV or drugs
  - vascular disease including hypertension, atrial fibrillation
  - other major co-morbidities that would influence surgical risk.

The location of the pain and its radiation can help narrow the differential diagnosis.

*But remember the possible pitfalls:*

- Poor localization of visceral pain
- Localization of pain in relation to embryological origins
- Anatomical variants, e.g. retrocaecal appendix
- Change in the nature of pain over time, e.g. poorly localized pain of appendicitis becoming localized with onset of peritonitis.

#### Examination

*A full examination of all major systems is required as abdominal pain may result from other systems (cardio-vascular, respiratory, musculoskeletal) but in particular:*

- Is the patient unwell, do they look pale, are they sick and in pain?
- Review the observations; are they tachycardic, tachypnoeic, hypotensive, pyrexial (elderly patients with infection may be hypothermic)?
- Remember restlessness of renal colic and the still, curled up patient with peritonitis.