A middle-aged man with postoperative fever

Your patient, a 58-year-old man, is about to undergo surgery for a carcinoma of the rectum. He does not have any significant past medical history. He is not on any medications and has never smoked cigarettes.

Operations are classified according to the potential for bacterial contamination of the wound and hence their risk of infection.

Q1 What are the categories and how would this operation be classified?

This patient is in a high-risk category for the development of septic complications after surgery.

Q2 What steps should be taken preoperatively to reduce the risk of wound infection?

Appropriate measures are taken to reduce the chance of wound infection. The surgeon resects the rectal tumour with difficulty and performs a stapled end-to-end anastomosis 8 cm above the dentate line.

Five days post procedure, the patient begins to complain of pain in the wound. He has a low-grade fever, but his pulse and pressure are normal. He is mobile and is tolerating a light diet. On physical examination his chest is clear, and the abdominal wound is tense, fluctuant, erythematous and tender at the superior aspect. The remainder of his examination is normal.

Q3 What is your diagnosis and what should you do?

The wound is incised and a small amount of pus drained. The patient improves, but on the morning of discharge, the patient reports ‘just not feeling well’. The nursing observation chart for the last few days is shown (Figure 2.1).
From the history and casenotes it is ascertained that the patient underwent a large bowel resection 10 days earlier and the operation notes record that the procedure was ‘difficult’. Apart from the changes noted on the nursing observation chart, nothing else abnormal is found on examination. In particular, his abdomen is soft and the wound infection has resolved.

A rectal examination is performed. You note some fullness posteriorly and the anastomosis cannot be felt. You arrange a number of investigations, including a CT scan (Figure 2.2).
Your other investigations are unremarkable apart from an elevated white cell count of $18 \times 10^9/l$.

Q5 What does the scan show and what is your plan of action?

The patient is returned to the operating room and the abscess drained per rectum. The antibiotics are continued until his temperature settles and the patient is discharged home 3 weeks after his operation.

An anastomotic leak was diagnosed in this patient as the source of intra-abdominal sepsis. Anastomotic leaks may declare themselves by discharge of faecal material through a drain or by systemic signs of sepsis. A contrast study can confirm the site and extent of the leak. Compromise of the blood supply to the anastomosis is considered to be the cause of most anastomotic leaks. Not all anastomotic leaks will lead to abscess formation, and many are only found on routine contrast studies. When a pelvic abscess develops it will often discharge spontaneously into the rectum and no further intervention is necessary.

A1 As part of your preoperative work-up of any patient about to undergo surgery, you want to identify any risk factors that may have an untoward influence on postoperative recovery. One important aspect is the assessment of the risk of infection (particularly chest, wound and operative site). The type of operation has a considerable bearing on the risk of infection. There are four categories into which operations are classified according to their risk for the development of wound infection. These are:

- clean
- clean-contaminated
- contaminated
- dirty.

Clean cases (e.g. hernia repair) where the gastrointestinal, respiratory or gynaecologic tract are not entered have low infection rates (<0.5%) with any contaminant likely being of skin origin. Clean-contaminated cases such as cholecystectomy for cholelithiasis have mild infection rates of 1–5% and are associated with entry into the foregut or biliary tree, areas considered to be sterile. Contaminated cases involve procedures on the colon (including the appendix), where the high bacterial content produces a 10–30% infection rate. Dirty cases are any cases where purulence is drained. Your patient is about to undergo an operation on a part of his gut with a normally high bacterial flora content – that is, it will be a ‘contaminated’ procedure.

A2 The important and most likely sources of infecting organisms are the colon and the skin. The bowel is prepared by emptying it of its faecal content, most commonly by using an isotonic, electrolyte-rich lavage. The patient drinks 3–4 l of the solution the day before surgery, producing a large catharsis. Hypovolaemia may be induced by this procedure and patients are instructed to also consume absorbable electrolyte solutions. Alternatively, if the patient is not particularly fit the preparation is done as an inpatient with simultaneous intravenous fluid replacement.

On the morning of surgery the patient will be given a shower using a bactericidal soap and the surgical site clipped immediately prior to operation.

Approximately 1 hour to surgical incision, the patient will be given a dose of intravenous broad-spectrum antibiotics (e.g. a second-generation cephalosporin), the goal being adequate tissue penetration of antibiotic at the time of the incision.

For clean and clean-contaminated cases, the role of prophylactic antibiotics is not so clear cut. Several other factors enter into the overall risk of postoperative infection and influence decisions.
on the usage of prophylactic antibiotics. For example, an inguinal hernia repair would not usually require antibiotics, but if mesh was incorporated into the procedure, the patient would probably be given a single dose of an antibiotic as a prophylactic measure. Emergency procedures have a higher risk than those performed electively. Patients in a poor state of health, with malignancy, immunosuppression (diabetes) or malnutrition are at increased risk. Antibiotics are more likely to be used in these groups of patients.

A3 The patient has a surgical wound infection. Incision and drainage of the wound are necessary. Incision should be extensive enough to allow easy drainage of the wound and easy wound care. If small and localized, such an infection may be treated at the bedside. Saline-soaked gauze can be used to dress the wound. Debridement may be necessary in more severe cases. If the patient has a cellulitic component to the wound infection or has a systemic response to the infection, antibiotics will be necessary. Patients who have prosthetic devices (e.g. joint replacements), are at risk of prosthesis infection (a catastrophic complication) and will need antibiotics.

A4 Over the last 72 hours the patient has had multiple fever spikes with associated tachycardia. The patient most likely has an underlying infection and a complete work-up is necessary. This should include:
- a full history and physical examination
- complete blood picture
- blood cultures taken when febrile
- urine culture
- chest radiograph.

As the patient has undergone a low anterior resection of the rectum, an intra-abdominal fluid collection may have accumulated in the pelvis or subdiaphragmatic spaces with leakage from the anastomosis being the most likely source. Therefore, CT scan of the abdomen and pelvis is also needed.

A5 The CT view of the pelvis in this patient shows an abscess behind the rectum. There are pockets of gas within the abscess cavity. This suggests that an anastomotic leak has occurred and the patient has had a septic response to this leak. The patient should be started on a broad-spectrum antibiotic and the abscess drained.

**Postoperative fever, focusing on infection**

**Aetiology**

Six different sites are considered:
- The pulmonary tract. Generally seen in the immediate postoperative period secondary to atelectasis or subsequently pneumonia.
- Urinary tract (common in patients with indwelling catheters).
- Cellulitis in venous access sites.
- Surgical wound infections generally present 3–7 days postoperatively.
- Intra-abdominal sepsis from abscess is considered after the 5th–7th postoperative day.
- Occasionally the blood may become seeded with bacteria (bacteraemia or septicaemia depending on the clinical presentation). This is usually from contamination of central catheters particularly in critically ill patients in intensive care units.

**Prevention**

The most important aspect of therapy.

Preoperative identification of risk factors:
- category of surgical procedure
- co-morbidities (e.g. diabetes, prosthetic heart valve).

Measures taken to reduce risk of infection:
- prophylactic use of antibiotics
- bowel preparation
- improvement in general state of patient:
  - chest physiotherapy and use of bronchodilators in patients with chronic obstructive pulmonary disease
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**REVISED POINTS** — cont’d

- correction of any anaemia or malnutrition
- use of topical antiseptic measures at site of operation
- good surgical technique and use of prophylactic perioperative antibiotics
- early ambulation and aggressive pulmonary toilet
- appropriate analgesia to aid good respiratory effort and mobilization
- early removal of indwelling catheters as soon as the patient can void.

**Diagnosis**

The work-up of a postoperative fever is directed at the five most common sources. After a complete physical examination with careful inspection of the surgical wound, blood test and cultures may be added. Chest X-ray and urinalysis are generally considered and if intra-abdominal abscess is suspected, CT scan or ultrasound can be diagnostic and can be used in treatment.

**Management**

When a focus of infection is determined, antibiotic usage should be tailored to likely organism(s) and risk of systemic sepsis.

- a localized collection is best drained
- antibiotics should be given when:
  - there is evidence of spreading infection
  - the patient is septic
  - host defences are reduced (e.g. immunosuppression)
  - any infection would have grave consequences (e.g. presence of a prosthetic heart valve or joint replacement).

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**ISSUES TO CONSIDER**

- What are the important differences between the terms 'bacteraemia', 'septicaemia' and 'septic shock'?

- How would you counsel this patient before his operation? What else does he need to know about, apart from the risk of wound infection?

- There are other potential risks and complications, related specifically to this operation and to major surgery in general. How would you go about reducing the risk of some of these other potential problems?

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**FURTHER INFORMATION**

www.emedicine.com/MED/topic2702.htm

A textbook description of the diagnosis and management of intra-abdominal abscesses.
You are asked to assess a 77-year-old man in the emergency department. He has presented with vomiting for several weeks, which has worsened in the last few days.

**Q1** What history will help determine the cause of the vomiting?

The patient tells you that he vomits once or twice a day and it is getting worse. It tends to occur in the evenings but is not always related to his evening meal. The vomitus contains clear fluid and partially digested food, sometimes containing food he ate several meals ago. There is no bile and the smell is not particularly offensive. Sometimes prior to vomiting he has mild upper abdominal discomfort but there is usually no warning. There has not been much pain but he has experienced bloating recently. He thinks he might have lost weight in the last few weeks and feels tired and lethargic. His bowel function is normal but less frequent, especially in the last week. He has not had fevers or jaundice. He is otherwise well and your review of systems is negative. His past history includes a recent chest infection and a myocardial infarction 3 years ago. He underwent a colonic resection for diverticular disease 5 years earlier. His only medication is aspirin and he does not drink or smoke.

**Q2** What is the most likely cause of the vomiting and what could be the significance of the recent chest infection?

On examination he looks unwell. His peripheries are warm and well perfused, but there is loss of the normal skin turgor. The pulse rate is 100 bpm and his temperature is 37.4°C. His blood pressure on lying is 140/90 mg Hg and 125/85 mm Hg on sitting. Examination of his cardiorespiratory systems is unremarkable. The upper abdomen looks distended and on palpation it is soft with mild epigastric tenderness. There is nothing else abnormal to find in the abdomen. The bowel sounds are normal and rectal examination is unremarkable.

**Q3** What other physical sign might you expect in this patient?

The patient has a marked succussion splash.

**Q4** What are you going to do next?
Vomiting

Chest and abdominal radiographs are performed (Figures 15.1 and 15.2).

An intravenous cannula is inserted and 1 litre isotonic saline is run in over 2 hours. Blood is collected for a complete blood picture and electrolytes.

<table>
<thead>
<tr>
<th>Investigation 15.1 Summary of results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin 142 g/l</td>
</tr>
<tr>
<td>MCV 76 fl</td>
</tr>
<tr>
<td>MCH 29.5 pg</td>
</tr>
<tr>
<td>MCHC 280 g/l</td>
</tr>
<tr>
<td>Sodium 133 mmol/l</td>
</tr>
<tr>
<td>Potassium 3.2 mmol/l</td>
</tr>
<tr>
<td>Chloride 85 mmol/l</td>
</tr>
<tr>
<td>Bicarbonate 27 mmol/l</td>
</tr>
<tr>
<td>Urea 16 mmol/l</td>
</tr>
<tr>
<td>Creatinine 0.13 mmol/l</td>
</tr>
<tr>
<td>Uric acid 0.31 mmol/l</td>
</tr>
<tr>
<td>Glucose 4.4 mmol/l</td>
</tr>
<tr>
<td>Cholesterol 3.5 mmol/l</td>
</tr>
<tr>
<td>LDH 212 U/l</td>
</tr>
</tbody>
</table>

Q5 What are the abnormalities and how would you interpret these findings?
Clinical problems

To aid with fluid management, a urinary catheter is inserted and intravenous fluid titrated to achieve an output of at least 30 ml/h. In the first hour after admission the patient is given 500 ml fluid and another 2 l isotonic saline over the following 4 hours.

During the third litre of fluid replacement, the patient’s serum electrolytes are repeated (Investigation 15.2).

How would you interpret these radiographs?

Your insertion of a wide-bore nasogastric tube is promptly followed by drainage of 2 litres of turbid fluid. Particles of recognizable food are seen in the drainage bag, but no bile.

How would you manage fluid replacement?

<table>
<thead>
<tr>
<th>Investigation 15.2 Serum electrolytes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium</td>
</tr>
<tr>
<td>Potassium</td>
</tr>
<tr>
<td>Chloride</td>
</tr>
<tr>
<td>Urea</td>
</tr>
<tr>
<td>Creatinine</td>
</tr>
</tbody>
</table>
This is a view of the antrum, with an arrow pointing to the pylorus.

Q8 What fluids would you prescribe over the next 4 hours?

The patient is monitored in a high-dependency unit and once he is stable an endoscopy is performed (Figure 15.3).

This patient had one of the more unusual causes of gastric outlet obstruction. He underwent a laparoscopic gastroenterostomy to bypass the obstructed duodenum.

Q9 What does Figure 15.3 show?

The endoscopist is unable to get her instrument through the pylorus, which appears rigid. She dilates the pylorus with a balloon dilator and takes some blind biopsies through the pylorus. These confirm the diagnosis of adenocarcinoma and a CT scan is performed (Figure 15.4).

Q10 What does the CT scan show?
A1 To establish the likely cause of his vomiting you will need the following information.

- Details of the vomiting:
  - when it started, how many times a day it occurs and whether the frequency has changed over this period
  - if there is any associated anorexia or nausea
  - if it is projectile
  - what the contents of the vomitus are (e.g. recognizable food, blood, bile. Remember old blood is described as ‘coffee-grounds’ in appearance)
  - any relationship to food (triggered by food? How long after a meal does it occur?).

- Any associated symptoms, particularly gastrointestinal (dysphagia, pain, distension, bowel change, jaundice, loss of weight).

- Drug history, including alcohol consumption.

- General state of health and past history (e.g. diabetes).

Apart from gastrointestinal causes, you need to consider:

- Central nervous system disorders: raised intracranial pressure deprives the vomiting centre of oxygen and triggers vomiting. Nausea may be absent and the vomiting projectile.

- Medications: these may cause vomiting by irritation the gastrointestinal tract or by acting centrally.

- Infection: especially urinary tract in the elderly.

- Severe pain: vomiting is common in renal colic and myocardial infarction.

- Metabolic disorders including diabetic acidosis, uraemia, Addisonian crisis and thyrotoxicosis. Acidosis and hypokalaemia directly affect the vomiting centre.

- Shock: this causes decreased central oxygenation.

- Vertigo.

- Migraine: this mildly raises intracranial pressure.

- Psychiatric: bulimia, psychosomatic.

A2 From history alone, this patient almost certainly has gastric outlet obstruction. In this setting the stomach will fill to capacity and then empty itself by vomiting, often without warning. As a result there may be no preceding nausea and the vomiting may be projectile. As the stomach is expansile there may not be an obvious relation to food, with vomiting only occurring after several meals. Typically there will be partially digested food but no bile. As the vomiting associated with gastric outlet obstruction can be profuse and abrupt there is always the risk of pulmonary aspiration. The patient’s recent chest infection may be a reflection of this complication.

Other gastrointestinal causes should be considered. Oesophageal obstruction usually leads to rapid regurgitation of undigested food and dysphagia is a prominent symptom. Obstruction distal to the stomach will produce a vomitus that contains bile or is faeculent, so described because of its offensive smell and taste, consistency and colour. These patients will usually have other gastrointestinal symptoms (e.g. colicky abdominal pain and distension in small bowel obstruction).

The two most common causes of gastric outlet obstruction in the adult are:

- Malignancy:
  - carcinoma of the antrum
  - extrinsic duodenal compression by pancreatic cancer.

- Chronic duodenal ulceration.

All other causes are rare and include:

- Hypertrophic (adult) pyloric stenosis.

- Pancreatic abnormalities:
  - congenital (annular, heterotopic)
  - pancreatitis +/− pseudocyst.

- Other neoplastic processes:
  - carcinoma of the ampulla of Vater
  - carcinoma of the biliary system
  - duodenal or gastric polyps
  - enlarged lymph nodes
  - functional (adynamic stomach).

This patient’s history could be consistent with benign disease or an underlying malignancy.

A3 The classic sign of gastric fullness is the succussion splash, which is elicited by listening over the abdomen while rocking the patient.
Fluid and food is heard ‘splashing’ in the unemptied stomach. It is always important to explain to the patient what you are about to do beforehand, as this manoeuvre can be disconcerting.

A4 This patient needs resuscitation. He is dehydrated and relatively hypovolaemic, as judged by loss of normal skin turgor and his postural drop in blood pressure. He will require:
- Insertion of an intravenous cannula.
- Rapid infusion of isotonic saline.
- Insertion of a nasogastric tube and gastric aspiration.
- Oxygen by face mask.
- Collection of blood samples for a complete blood picture and biochemistry.
- Radiography of the chest and abdomen.

A5 The red cell indices show microcytosis and hypochromia. Although his haemoglobin is normal, he is dehydrated and haemoconcentrated. Thus, it is likely he has an underlying iron deficiency anaemia.

The urea is disproportionately elevated relative to creatinine and this prerenal impairment is a reflection of dehydration.

The biochemical changes that are sometimes seen in total gastric outlet obstruction and profuse vomiting can be complex. Patients with prolonged gastric outlet obstruction can present with a metabolic alkalosis and a paradoxical aciduria, but such extreme biochemical changes are rare.

In this case, the patient has a mild hypokalaemia accompanied by a hypokalaemia and hypochloraemia. This is a reflection on the profuse and prolonged vomiting directly causing electrolyte loss. In such circumstances, the patient may lose large quantities of hydrogen and chloride ions, with subsequent renal retention of bicarbonate to compensate for the loss of chloride in the vomitus.

A6 The chest radiograph shows a large gastric air bubble under the left diaphragm, with a fluid level. The lung fields are clear and there is no evidence of any pneumonic process. The abdominal radiograph shows a massively dilated stomach, full of fluid. The greater curve of the stomach can be seen at the level of the iliac crests, and the transverse colon is compressed into the pelvis. The ring of metal staples from his previous surgery can be seen low in the pelvis.

A7 As the patient’s primary problem is dehydration, crystalloid fluid is appropriate and isotonic saline should be used rather than dextrose. The patient is dehydrated and will need rapid fluid replacement to maintain renal function and prevent acute tubular necrosis. In the older patient a careful assessment of cardiac function is important to determine the risk of developing cardiac failure with rapid fluid replacement. As this patient has a history of myocardial ischaemia, careful monitoring is necessary. Central venous pressure monitoring may be appropriate.

Commence intravenous fluids at 1 litre over 1–2 hours while monitoring urine output and respiratory function, including saturation. Hourly urine measures are imperative and fluid regimen should be adjusted accordingly. If initial output is low, fluid boluses should be given. Potassium should be replaced but cautiously until urine output is established.

Once fluid deficit has been corrected, fluid rate should be determined by maintenance requirements plus losses. In this setting nasogastric losses may be large and it is preferable to replace the amount separately with 0.45% saline with 1 g (13.9 mmol) potassium per litre.
maintains a urine output of at least 30 ml/h and recheck cardiopulmonary status and electrolyte measurements frequently.

A9 There are food particles scattered throughout the stomach. As endoscopy is normally performed in the fasted state with the stomach empty of food, this is abnormal. The pylorus is narrowed and there appears to be a mass in this area pushing back into the antrum.

A10 This is a section through the upper part of the abdominal cavity and shows contrast in the stomach. The right lobe of the liver is to the left of Figure 15.4. There is a mass arising from the gallbladder fossa which is compressing the duodenum. There is another mass (of lymph nodes) pressing the duodenum from behind. This is likely to be a carcinoma of the gallbladder, with direct extension into adjacent structures (including the duodenum) and local lymph node spread.

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**Gastric outlet obstruction**

**Definition**
Obstruction to gastric outflow due to some disease process at the level of the antrum, pylorus or duodenum.

**Aetiology**
The two important causes are:
- Distal gastric malignancy.
- Duodenal ulcer disease (6–8% of all cases will develop obstruction).

Gastric outlet is seen less frequently because:
- The incidence of distal gastric cancer is declining.
- Duodenal ulcer disease tends to be treated earlier and more effectively (PPIs and *Helicobacter pylori* eradication).

**Clinical features**
- Profuse vomiting of bile-free vomitus, containing recognizable food, often from meals eaten days earlier.
- May present with severe dehydration, drowsiness and confusion.
- Characteristic finding of a succussion splash.
- Complex biochemical changes which can make resuscitation difficult.

**Management**
- Resuscitation.
- Rehydration
  - correction of electrolyte imbalances
  - monitoring of urine output
  - large-bore nasogastric tube and nasogastric fluid replacement.
- Identify the underlying cause.

**Investigations**
- Serum biochemistry.
- Arterial blood gas analysis (metabolic alkalosis is common).
- Radiology
  - plain films (prominent gastric bubble, gastric distension)
  - contrast study (endoscopy preferred, but may not be feasible if the stomach remains full of food)
  - CT to identify structures outside the digestive tract that may be causing compression.
- Endoscopy for
  - precise identification of cause of obstruction
  - biopsy
  - possible dilatation of the narrowed segment.

**Treatment**
**Peptic ulcer disease**
- Aggressive medical therapy
  - *H. pylori* eradication
  - acid suppression.
  - If still obstructed (after 2 weeks’ therapy)
    - endoscopic dilatation (50% success rate) or
    - surgery (pyloroplasty or gastroenterostomy).

**Gastric malignancy**
- Resection.
- Bypass.
Other treatments depending on the cause.
ISSUES TO CONSIDER

- What electrolyte disturbances would you expect after prolonged vomiting, such as in hyperemesis gravidarum? Remind yourself of acid–base balance and how it is regulated.
- What are the normal daily requirements of potassium and sodium?
- How would a person with small bowel obstruction present, and what are the common causes?

FURTHER INFORMATION

www.emedicine.com/med/topic2713.htm
A textbook approach to the topic of gastric outlet obstruction.
A 35-year-old woman presents to the emergency department complaining of 5 days of progressive generalized headache and nausea. Her blood pressure is taken and is recorded at 250/140 mm Hg. The patient states that she has a family history of hypertension and was noted to be hypertensive for the first time 2 years ago. She had no investigations, but was treated with verapamil 80 mg tds. The patient stopped taking this after a few months as it made her constipated.

Q1 From what might this patient be suffering? What further history is required from her?

Q2 What would you look for on examination?

The patient does not have any relevant previous illnesses and had a tubal ligation 6 years ago. She complains of sweating, palpitations and anxiety. She takes no drugs, but smokes 10 cigarettes a day and consumes 100 gm alcohol a week.

On examination the patient is anxious and sweaty, but otherwise appears fit. Her pulse rate is 120 bpm and regular and her blood pressure 250/140 mm Hg. Significant abnormalities include a forceful but undisplaced apical cardiac impulse without evidence of left ventricular failure, and a bruit in the left side of her abdomen. Urinalysis reveals 1+ (0.3 g/l) albumin only. Her retina is shown in Figure 49.1.

Q3 What does this retinal photograph show?

The changes seen on fundoscopy are consistent with the clinical picture of severe hypertension.
Q4 What type of treatment should immediately be instituted?

You insert a radial arterial line for accurate monitoring of blood pressure and admit her to the high dependency ward. Her blood pressure improves with your chosen therapy, and you now have time to think about the possible cause of her hypertensive crisis.

Q5 What are the possible causes of this woman’s hypertension and what investigations would you like to organize?

The patient’s complete blood picture, ESR, biochemistry, urine microscopy and culture and chest X-ray were normal. The ECG showed a sinus tachycardia and left axis deviation, but was otherwise normal. Urinary catecholamines were within normal limits. Renal ultrasound is normal. The patient went on to have the investigation shown in Figure 49.2.

Q6 What is this investigation? What does it show?

The patient then had another investigation (Figure 49.3).
Q7 What is the investigation and what does it show? How can this condition be treated?

The patient had the most marked stenotic segment dilated via percutaneous transluminal balloon angioplasty, with a dramatic improvement in her blood pressure.

An important lesson in this case is that the patient was inadequately assessed on her first presentation with hypertension 2 years earlier. At that time the minimum investigation should have included urinalysis, serum biochemical analysis, ECG and possibly echocardiography (to assess for left ventricular hypertrophy), chest X-ray and lipid profile. In addition, there should have been careful follow-up and counselling to stop smoking.

In view of her young age, a specific underlying cause of her hypertension should have been considered. Further, if the abdominal bruit had been listened for (and been present and found) at her initial presentation, her fibromuscular dysplasia may have been diagnosed and the subsequent emergency avoided.

Patient education is vital to prevent loss to follow-up. If unacceptable side effects occur, a switch to an alternative medication will promote compliance. Otherwise, their hypertension may go untreated for long periods.

A N S W E R S

A1 She may have poorly controlled essential hypertension or hypertension secondary to underlying kidney disease, and an intercurrent problem such as a viral illness which has produced the headache and nausea. However, she has marked hypertension for a young person and may be in a hypertensive crisis. In this situation encephalopathy can occur which would be suggested by the headache and nausea. Her past medical history will be important as she may give a history of known poorly controlled hypertension or renal disease. You must inquire as to whether she may be pregnant (pre-eclampsia).

You should ask her about the severity of her previously diagnosed hypertension, and whether she knows of her recent blood pressure readings – if it has been progressively increasing over several months the immediate risk is lower than if this has been a sudden rise over a few days or weeks.

Ask her about symptoms associated with hypertensive encephalopathy such as irritability, visual disturbances, confusion, altered consciousness and seizures.

Ask about symptoms that might suggest an underlying disorder to account for her hypertension, such as:
A N S W E R S — cont’d

- Renal disease: thirst, polyuria, nocturia, dysuria, haematuria, colic, lethargy and general malaise of uraemia.
- Phaeochromocytoma: sweating, palpitations, anxiety and tremor, particularly occurring in paroxysms.
- Cushing’s syndrome: truncal weight gain, thinning of skin, easy bruising, weakness of proximal limb muscles, striae, hirsutism.

You will also need to ask about symptoms suggestive of hypertensive damage to the retina (visual deterioration) or the cardiovascular system, including acute myocardial ischaemia or failure (angina, dyspnoea, orthopnoea, ankle swelling), and aortic dissection (back pain).

A drug history is vital, including past use of analgesics (particularly NSAIDs: analgesic nephropathy), current use of drugs associated with hypertension, e.g. oral contraceptive pill, sympathomimetics (e.g. nasal decongestants), steroids, some antidepressants (including venlafaxine) and combinations of antidepressants associated with a risk of serotonergic syndrome (e.g. SSRIs and monoamine oxidase inhibitors). The patient’s use of tobacco, alcohol and other illicit drugs, particularly cocaine and amphetamine derivatives, should also be explored.

A2 On examination you will need to look for evidence of hypertensive damage to:

- The CNS (level of consciousness, visual fields, focal neurological deficits).
- The retina (hypertensive retinopathy, especially haemorrhages, new exudates or optic disc swelling).
- The heart (left ventricular hypertrophy, left ventricular failure).
- The aorta (unequal pulses in aortic dissection).
- The kidney (haematuria or proteinuria on urinalysis).

Note: The presence of any acute features, especially neurological symptoms or signs, retinal haemorrhages, exudates or optic disc swelling, and abnormalities on urinalysis, suggest the presence of accelerated/malignant hypertension requiring emergency treatment.

Features to suggest underlying aetiology:

- Cushingoid habitus.
- Delayed femoral pulses in coarctation of the aorta.
- Palpable hydronephrotic or polycystic kidneys.
- Abdominal bruits.
- Appearance of uraemia.
- Sweating, tremor and tachycardia, and rarely, abdominal masses in phaeochromocytoma.
- Generalized oedema and/or abnormal urinalysis in glomerulonephritis.
- Evidence of a connective tissue disorder such as SLE or skin manifestations of vasculitis.

A3 Figure 49.1 shows:

- Hard exudates.
- Cotton wool spots.
- Flame haemorrhages.

This is grade 3 hypertensive retinopathy. There is considerable hard exudate in the posterior pole with formation of a partial macular star.

In grade 4 retinopathy optic disc swelling would also be present, but there is no evidence that clinical outcomes differ on the basis of the fundoscopic findings and both grade 3 and grade 4 should be regarded as indicators of hypertensive emergency.

A4 This is a medical emergency.

- Her blood pressure should be lowered gradually over several hours, aiming for no more than a 20–25% reduction in mean arterial pressure, or a reduction to no lower than 160/100 mm Hg, over the first 1–2 hours. A careful balance is required between reducing the pressure rapidly enough to prevent or reverse hypertensive encephalopathy and reducing it too rapidly with the attendant risk of cerebral hypoperfusion and infarction (stroke).
- Strict bed rest and intra-arterial blood pressure monitoring are desirable if the facilities are available (e.g. on a high dependency ward).
- Blood pressure reduction using a short-acting parenteral agent such as a sodium nitroprusside or labetalol infusion, titrated according to the blood pressure response, is safer than fixed doses of intravenous, oral or sublingual antihypertensive agents.
The use of oral nifedipine capsules is no longer recommended because they have a rapid onset of action and reach very high peak plasma concentrations, resulting in the potential for sudden uncontrolled blood pressure reduction and precipitation of stroke.

Once blood pressure is lowered, a combination of antihypertensive agents will probably be needed to maintain good control, the choice depending on the characteristics of the patient. Nifedipine tablets (not the slow-release OROS or GITS formulations) can be used at this stage in an initial dose of 20 mg, particularly in combination with selective beta-blockade (e.g. atenolol 50 mg).

This patient has a renal bruit; in patients with clinical evidence of possible renal artery stenosis, nifedipine and betablockade may be safer and more effective than therapy based on an angiotensin converting enzyme (ACE) inhibitor.

Note that betablockers can cause a paradoxical and possibly dangerous rise in hypertension in the presence of phaeochromocytoma, while angiotensin converting enzyme (ACE) inhibitors (e.g. captopril) can precipitate a marked deterioration in renal function in patients with bilateral renal artery stenosis.

Pregnancy: pre-eclampsia.

Adrenal disorders: e.g. phaeochromocytoma, Cushing’s syndrome, primary aldosteronism (Conn’s syndrome).

Drug associated: e.g. oral contraceptive pill (common), corticosteroids, sympathomimetics, alcoholism.

Cardiovascular e.g. coarctation of the aorta. With her anxiety, sweating and resting tachycardia, it is prudent to exclude a phaeochromocytoma, but her abdominal bruit suggests the possibility of renal artery stenosis. Baseline investigations should include:

- Complete blood picture and ESR.
- Electrolytes, including blood glucose, urea and creatinine.
- Urine microscopy (for casts and cells).
- Chest X-ray and ECG (to detect left ventricular hypertrophy).

The next investigation of choice would be a non-invasive screening test for renal artery stenosis. Several different tests can be done, and these include duplex ultrasound scanning, spiral CT angiography and gadolinium-enhanced magnetic resonance angiography (MRA).

Currently, the most commonly available test is a radionuclide scan of the kidneys, which will give data about the blood supply and function of each kidney. The diagnosis of renal artery stenosis can be made with increased certainty by rescanning 1 hour after oral captopril (an ACE inhibitor) is given. This is because when renal perfusion is markedly reduced (as with renal artery stenosis) the glomerular perfusion and filtration is dependent on efferent arteriolar resistance (‘back-pressure’). ACE inhibitors reduce angiotensin-mediated post-glomerular vasoconstriction and can cause a dramatic deterioration in glomerular filtration and renal function. Uptake of a radiopharmaceutical ($^{99m}$Tc DTPA), which is cleared by glomerular filtration, will be reduced. This will be seen as ‘blanching’ of the affected kidney on the renal scan.

Given the clinical context, this patient should also be screened for a phaeochromocytoma by assessing production of catecholamines. This is usually done by performing a 24-hour urine collection, assayed for excreted catecholamines and their metabolites.

A5 This woman may have essential (primary) hypertension as she does have a positive family history and the hypertension may be exacerbated by smoking and alcohol. However, the severity of her hypertension at her age suggests a specific underlying cause. The common causes of secondary hypertension include:

- Renal parenchymal disease:
  - Unilateral, e.g. pyelonephritis, obstructive or reflux nephropathy, dysplasia, trauma.
  - Bilateral, e.g. any cause of chronic renal failure, obstructive or reflex nephropathy, diabetes mellitus, analgesic nephropathy, polycystic disease, pyelonephritis, glomerulonephritis, interstitial nephritis.
- Renovascular disease: renal artery stenosis secondary to atheroma, fibromuscular hyperplasia (especially in younger patients), trauma.
A6 This is a radionuclide scan of the kidneys. It shows (views taken from behind) delayed perfusion and delayed function of the left kidney and then late hyperconcentration of the isotope in the left kidney. The right kidney contributes 65% of total renal function and the left kidney contributes 35%. These results strongly suggest left renal artery stenosis, and the diagnosis should be confirmed by renal angiography, which remains the gold standard for definition of the renal arterial anatomy.

A7 This is a renal angiogram and shows contrast in the left renal artery. There are several narrowed segments in the left renal artery and the origin of the vessel looks normal. This pattern of narrowing is referred to as ‘beading’. There are no changes to suggest atheromatous disease of the arterial tree and the cause of the abnormality is fibromuscular dysplasia. This condition is usually treated by percutaneous balloon angioplasty, or by open surgery.

REVISION POINTS

Hypertensive emergencies

Incidence
Less than 1% of all cases of hypertension; secondary hypertension accounts for about 5% of all cases of hypertension but 25–50% of cases of hypertensive emergency.

Risk factors
- For hypertension: family history, obesity, alcohol abuse.
- For hypertensive crisis: secondary hypertension (esp. renal disease, renovascular disease), oral contraceptive pill, smoking.

Presentation
Headache, visual impairment, dizziness, anxiety, disorientation, tremor, seizures, nausea, vomiting, abdominal pain.

Clinical features
- Usually severe hypertension (diastolic BP > 120) but may occur at moderate BP levels if BP has risen rapidly from a low baseline.
- Grade 3 or 4 retinopathy (haemorrhages, exudates +/- optic disc swelling).
- Proteinuria is common.
- May be focal neurological findings, acute left ventricular failure.

Prognosis
Untreated prognosis is very poor, with 5-year survival about 1%; with antihypertensive treatment 5-year survival is about 75%.

Early management
- Investigations: plasma electrolytes, urea and creatinine, blood picture, ESR; ECG; chest X-ray; urinalysis and urine microscopy.
- Intra-arterial BP monitoring and intensive care, with infusion of sodium nitroprusside or labetalol titrated to response, aiming for no more than 20–25% reduction in BP over first 2 hours.

Later management
Investigations for secondary hypertension as clinically indicated and specific management if a cause is found.

Combination oral antihypertensive drug therapy according to clinical features (usually require at least two drugs for initial control). Captopril, nifedipine (slow release), atenolol, prazosin have all been used successfully. Thiazide diuretics could be used if not volume depleted.

Renovascular hypertension

Epidemiology
- Accounts for approximately 3% of all cases of hypertension and about 15% of cases of hypertensive emergencies.
- Atheromatous renovascular disease is the most common form (seen mainly in elderly men with vascular risk factors).
- Fibromuscular hyperplasia may account for up to one-third of cases and is diagnosed
most commonly in women between the ages of 30 and 50 (bilateral in about 50%).

**Diagnosis of renal artery stenosis**
- The best method for non-invasive screening is currently controversial.
- The captopril-challenged isotopic renal scan has been the gold standard.
- Newer techniques such as spiral CT angiography, duplex ultrasonography and MR angiography are being evaluated.

All four techniques have reasonable sensitivity and specificity when performed skilfully, but only renal perfusion scanning indicates functional significance of any stenosis.

If non-invasive testing is highly suggestive of renal artery stenosis, then an arterial digital subtraction angiogram, with or without venous renin measurements, should be performed.

**Treatment**

Indications for treatment are uncontrollable hypertension and deteriorating renal function.

Best treated by balloon angioplasty, or surgery if this is unsuccessful; in fibromuscular dysplasia, 60% of patients treated by percutaneous balloon angioplasty will remain cured at the end of 12 months and long-term prognosis is good.

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**ISSUES TO CONSIDER**

- What non-pharmaceutical methods can hypertensives employ to lower their blood pressure?
- How would you control hypertension secondary to a phaeochromocytoma?
- What problems are associated with continuous infusions of sodium nitroprusside?

**FURTHER INFORMATION**

- [www.bloodpressure.com](http://www.bloodpressure.com) An excellent commercial site with information and links for patients and physicians alike.
- [www.ash-us.org](http://www.ash-us.org) Website of the American Society for Hypertension with lots of links.