Mrs Jamila Ahmed is a 25-year-old woman who is referred to the medical assessment unit by her GP. Mrs Ahmed does not speak English, but her husband acts as an interpreter. She gives a 1-month history of a cough productive of clear sputum which has now become green with streaks of blood. She also complains of fevers and drenching night sweats and has lost approximately 10 lb in weight. She has no significant past medical history, is not on any regular medication and does not smoke or drink alcohol.

What are your main differential diagnoses at this stage?

- **Pulmonary tuberculosis (TB).** Cough, haemoptysis, night sweats and weight loss are classic features of TB.
- **Other lower respiratory tract infection:**
  - *pneumonia* – this is an important cause of haemoptysis, purulent sputum and fevers. A 1-month history is probably too long for acute bacterial pneumonia. However, viral infection complicated by bacterial pneumonia could have a more prolonged course.
  - *bronchiectasis* – clinical features are consistent with bronchiectasis, although the normal past medical history and relatively short (1 month) onset of symptoms make this less likely.
- **Underlying malignancy.** Haematological malignancies, such as lymphoma and leukaemia, cause ‘B’ symptoms of weight loss and night sweats. These conditions could cause chest symptoms by suppressing normal immunity and predisposing to infection.
- **Underlying HIV infection.** This could cause systemic symptoms of weight loss and predispose to opportunistic respiratory infection.

If you suspect tuberculosis what should you do next?

Tuberculosis is caused by the bacterium *Mycobacterium tuberculosis*. This can be caught from a person with active pulmonary TB who has *M. tuberculosis* aerosolized in respiratory droplets expelled from their lungs. These droplets are released when the person coughs, speaks or spits. Tuberculosis is difficult to treat and can be fatal, therefore infection should be prevented whenever possible.

If she has TB, it is important to ensure that Mrs Ahmed does not infect other hospital patients or staff, particularly patients who are immunocompromised. UK guidelines (www.nice.org.uk/page.aspx?o=CG033) state that patients with suspected pulmonary TB admitted to hospital should be isolated in a side room to reduce the risk of spread. If they leave the room they should wear a surgical mask. People entering the room should wear a mask if there is suspicion of drug-resistant TB or if the patient is generating respiratory droplets (cough, sputum induction or bronchoscopy).

Mrs Ahmed should be isolated until the diagnosis has been proved and treatment commenced, or until active TB has been excluded with three negative sputum samples examined for *M. tuberculosis* by microscopy.

**RED FLAG**

Have a low threshold for suspecting TB in patients with pulmonary illness.

If you have any suspicion of pulmonary TB, isolate the patient first and ask questions afterwards.

**How infectious is tuberculosis?**

According to the World Health Organization (WHO), a person with active pulmonary TB can infect 10–15 people per year. The risk is greatest in family members or other people who have close and frequent contact with the infected person. Health care workers are also at risk of contracting TB as they can be exposed to the infection many times throughout their career.

For more information see the WHO TB fact sheet: www.who.int/mediacentre/factsheets/fs104/en
What happens when a patient is infected with *Mycobacterium tuberculosis*?

**Primary tuberculosis**

When *M. tuberculosis* reaches the pulmonary alveoli it:
- Invades alveolar macrophages and replicates
- Is taken up by (but does not replicate in) dendritic cells and is transported to local lymph nodes
- A few bacteria may ‘escape’ into the bloodstream

Infection by *M. tuberculosis* causes a local inflammatory reaction at infected sites, resulting in formation of granulomas (Box 106; Figs 82–84) which contain the infection. Possible outcomes at this stage include:
- Eradication of infection and healing, sometimes with calcification
- Latent infection
- Failure of the immune process, resulting in widespread dissemination of *M. tuberculosis* throughout the body, causing miliary TB

In the lung, the site of primary infection is usually the periphery of the lung (subpleural) in the mid or upper zones. The small area of granulomatous inflammation seen on chest X-ray is known as a Ghon’s focus or complex in conjunction with hilar lymphadenopathy.

A patient with primary TB is usually not unwell and may not know they have been infected until they have a chest X-ray or develop postprimary TB. They are not infectious.

**Postprimary tuberculosis**

A total of 5–10% of people infected with *M. tuberculosis* will go on to develop active disease at some point in their life, with the greatest period of risk being in the first 2 years after infection. The risk of developing active TB is
much greater in patients infected with both *M. tuberculosis* and HIV.

In postprimary TB, the immune response to *M. tuberculosis* is the main cause of tissue damage. Postprimary TB therefore only occurs once the immune response to *M. tuberculosis* has developed (2–10 weeks). It is triggered by:

- Reactivation of latent infection resulting from a reduction in host immunity (most common; Box 107)
- Re-infection with *M. tuberculosis*

In the lung, the main pathological processes are:

**Box 106 Formation of caseating granulomas**

- Invasion of alveolar macrophages by *Mycobacterium tuberculosis* stimulates an inflammatory reaction
- The infected macrophages become surrounded by T and B lymphocytes and fibroblasts (Fig. 82)
- Within the aggregate of cells (granuloma), T lymphocytes (CD4+ secrete cytokines. This activates infected macrophages to destroy their invading bacteria. CD8+ T lymphocytes also kill infected cells
- Activated macrophages may fuse together to form giant cells (Fig. 83)
- Dead macrophages and giant cells accumulate as necrosis at the centre of the granuloma
- With the naked eye the necrotic material generated by tuberculous granulomas looks like cheese (Fig. 84) and so is called ‘caseation’

**Box 107 Risk factors for tuberculosis**

Development of tuberculous disease usually requires exposure to *Mycobacterium tuberculosis* infection followed (or accompanied) by suppression of host defence mechanisms, allowing reactivation of infection.

**Risk factors for exposure to Mycobacterium tuberculosis**

- Overcrowded living conditions
- Homelessness
- Infected family member
- Residence in or immigration from place with high prevalence of TB
  - worldwide – Eastern Europe/Russia, Asia, Africa, South America
  - in UK – areas of cities, particularly those with high migrant population

**Risk factors for reactivation or potentiation of infection**

- Human immunodeficiency virus (HIV)
- Drugs (e.g. corticosteroids, chemotherapy, disease-modifying drugs for inflammatory conditions)
- Malignancy
- Diabetes mellitus
- Debilitation, especially old age
- Alcohol excess, substance abuse

NB. Neonates and young children are also susceptible to TB as a result of underdeveloped immune systems and are particularly at risk of miliary TB.
• **Consolidation or cavitiation.** This usually occurs in the lung apices or superior segments of the lower lobes as these have higher oxygen tension which supports bacterial growth. Processes include:
  - further formation of granulomas
  - necrosis and caseation, causing lung cavities
  - fibrosis
• **Pleural infection.** This may cause a pleural effusion and/or thickening
• **Bronchiectasis.** Obstruction of large airways by enlarged lymph nodes can cause pulmonary collapse and bronchiectasis
• **Miliary tuberculosis.** This affects the whole body. In the lung it may be seen on chest X-ray as multiple small nodules throughout both lung fields. This form of TB is particularly serious and needs urgent treatment as it carries 20% mortality even with urgent treatment

Patients with consolidation, cavitation or bronchiectasis may have cough or haemoptysis. Patients with extensive parenchymal disease or a large pleural effusion may be breathless. Patients may also have systemic features of disease such as lethargy, fevers, sweats and weight loss.

Patients with active pulmonary disease may shed *M. tuberculosis* into their lung secretions. These patients are infectious.

**KEY POINT**

There is a crucial difference between TB infection and tuberculous disease:
• **Tuberculosis infection.** The patient has live *M. tuberculosis* organisms somewhere in their body but their immune system is controlling the infection effectively and they are not unwell or infectious
• **Tuberculous disease.** This usually occurs when the immune system becomes weakened and latent infection is reactivated. This results in tissue destruction and the patient becomes unwell. If the lung is affected they may be infectious.

**Mrs Ahmed is originally from Somalia and came to the UK 3 years ago shortly after marrying. She now has a 2-year-old child. She is not aware of any previous exposure to TB or possible immunodeficiency.**

**What are your concerns when taking a full history from Mrs Ahmed?**

Patients have a right to confidentiality. Mrs Ahmed is unable to speak English and her husband is acting as an interpreter. There may be information relating to her condition that she does not wish to share with him. This is highlighted by the difficulty of taking a history of possible HIV exposure in these circumstances.

**How might you address these difficulties?**

One option is to speak to Mrs Ahmed at a separate time without her husband present. An independent Somali interpreter could be used who is either present in person or on a three-way phone line using a service such as Language Line.

**Mrs Ahmed agrees to be examined. Her temperature is 37.6°C. There is no evidence of lymphadenopathy. On auscultation there are crepitations over the right upper zone of her chest. No other abnormalities are identified.**

**Does her examination yield any new information?**

The pyrexia fits with a diagnosis of TB or other respiratory infection. Right upper zone crepitations would be consistent with consolidation or other pathology at this site.

**What initial investigations would you perform?**

The aim of initial investigation is to look for evidence to support your clinical diagnosis of pulmonary TB and rule out other diagnoses including pneumonia, bronchiectasis or malignancy.
• **Chest X-ray.** This would be particularly useful to look for features of TB or other lung disease
• **Full blood count and inflammatory markers.** These would be carried out to look for:
  - raised neutrophil count and C-reactive protein (CRP) consistent with bacterial infection
  - anaemia and raised inflammatory markers of chronic disease such as TB
abnormal cell counts consistent with haematological malignancy or immunosuppression

- Biochemical profile. To look for abnormalities in renal, hepatic or bone profiles – sites that might be affected by TB

Mrs Ahmed’s chest X-ray is shown in Fig. 85. Her blood test results were as follow:

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Haemoglobin</td>
<td>9.8 g/dL</td>
<td>(12–16 g/dL)</td>
</tr>
<tr>
<td>Mean cell volume (MCV)</td>
<td>84 fl</td>
<td>(80–96 fl)</td>
</tr>
<tr>
<td>White cell count</td>
<td>$10.8 \times 10^9$/L</td>
<td>(4–11 $\times 10^9$/L)</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>$8.2 \times 10^9$/L</td>
<td>(3.5–7.5 $\times 10^9$/L)</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>$0.8 \times 10^9$/L</td>
<td>(1.5–4 $\times 10^9$/L)</td>
</tr>
<tr>
<td>Platelets</td>
<td>$220 \times 10^9$/L</td>
<td>(150–400 $\times 10^9$/L)</td>
</tr>
<tr>
<td>Erythrocyte sedimentation</td>
<td>57 mm/h</td>
<td>(1–20 mm/h)</td>
</tr>
<tr>
<td>CRP</td>
<td>77 mg/L</td>
<td>(&lt;10 mg/L)</td>
</tr>
<tr>
<td>Sodium</td>
<td>131 mmol/L</td>
<td>(135–145 mmol/L)</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.1 mmol/L</td>
<td>(3.5–4.8 mmol/L)</td>
</tr>
<tr>
<td>Liver/bone/renal profiles</td>
<td>Normal</td>
<td></td>
</tr>
</tbody>
</table>

**How do you interpret these investigations?**

**Chest X-ray**

The striking abnormalities are:

- Consolidation, which is dense white shadowing containing black air bronchograms
- Cavitation of the lung within the consolidation, seen as a large black hole
- The abnormality is in the right upper lobe, defined at the bottom of the shadow by the horizontal fissure

**Blood tests**

Abnormalities identified are:

- Normocytic anaemia which can be caused by chronic disease such as TB
- Mildly elevated neutrophil count but low lymphocyte count, which could indicate suppression of cell-mediated immunity
- Mildly elevated CRP and ESR, indicating underlying inflammation
- Hyponatraemia (low sodium). Tuberculosis, pneumonia and lung abscess can cause syndrome of inappropriate antidiuretic hormone secretion (SIADH), with dilution of plasma sodium by retention of excess water

**How do these results affect your differential diagnosis?**

Cavitating upper lobe consolidation on chest X-ray is highly suspicious for TB.

Other causes of cavitlation (Box 108) are less likely as staphylococcal or klebsiella pneumonia would cause more systemic upset, lung cancer is unlikely in a 25-year-old non-smoker and purulent sputum and weight loss point away from a pulmonary embolus.

**Box 108 Causes of cavitating lung disease**

A lung cavity is an abnormal hole in the lung tissue where lung parenchyma has been destroyed. Causes include the following.

**Cancer**

- Primary lung cancer
- Lung metastases

**Infection**

- Tuberculosis
- Bacterial pneumonias (Staphylococcus spp., Klebsiella spp.)

**Infarction**

- Pulmonary embolus
Lung cavities can become infected with bacteria (abscess), or with Aspergillus, which can grow into a fungal ball called a mycetoma.
What tests could confirm the diagnosis of pulmonary tuberculosis?

**Sputum analysis**
Three morning sputum samples should be sent for:
- **Direct microscopy with specific staining.** The sample can be stained with Ziehl–Neelsen stain (red) or auramine (fluorescent). The bacteria have lipid-rich cell walls, which require phenol to make them permeable to dyes. This means that once they are stained the colour cannot be removed by acidic or alcohol washes. If these acid-fast bacilli (AFB) are seen on microscopy the sample is designated ‘smear positive’, which indicates high infectivity
- **Culture.** All samples are sent for culture:
  - smear-positive samples are cultured to determine whether the mycobacterium is tuberculosis or an atypical form and to determine antibiotic sensitivities to guide treatment
  - smear-negative samples may still contain AFB in numbers too small to be seen. The absence of *M. tuberculosis* needs to be confirmed by prolonged culture
- **Microscopy, culture and sensitivity** to detect the presence of other organisms that could cause pneumonia with cavitation (Box 108)

**Tuberculosis skin test**
This is also known as the tuberculin test and detects immunological evidence of infection with *M. tuberculosis*. The antigen used is tuberculin, a glycerine extract of the tubercle bacillus which is prepared as a purified protein derivative. The test is performed by:
- **Intradermal injection of a standard dose of tuberculin into the forearm away from visible blood vessels using one of two methods:**
  - Mantoux test (named after the French physician who developed it). A single needle is used to inject tuberculin
  - Heaf test (named after a British physician). Six tiny needles in a circle are used to inject tuberculin
- Measurement of the delayed-type hypersensitivity reaction to tuberculin:
  - Mantoux test is read 48–72h after tuberculin injection
  - if there has been previous exposure to bacterial protein, an immune response will be mounted around the injection site
  - the reaction is quantified by measuring the diameter of induration (palpable raised hardened area) across the forearm (perpendicular to the long axis) in millimeters. Absence of induration should be recorded as ‘0 mm’. Erythema (redness) should not be measured.
  - the tuberculin skin test detects an immune response to TB antigen. It is therefore positive in those with active disease, latent infection, treated (cured) TB or those who have received vaccination against TB. The results must therefore be interpreted with care (Table 10).

Mrs Ahmed gives three early morning sputum samples and they are all smear-negative for AFB on microscopy. She was not sure whether she had previously had a BCG vaccination, but on examination she did not have a scar on the upper arm or lateral thigh region. Her Mantoux test was read at 48h and measured as having produced induration of 2 mm.

**What is BCG vaccination?**
BCG stands for bacille Calmette–Guérin which is a live attenuated vaccine against TB. The vaccine was developed to confer immunity to infection with *M. tuberculosis* and prevent tuberculous disease. It is usually given into the upper arm around the insertion of the deltoid muscle. However, the vaccine is not universally effective:

<table>
<thead>
<tr>
<th>Table 10 Interpreting the tuberculin skin test (Mantoux).</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mantoux induration</strong>*</td>
</tr>
<tr>
<td>Result</td>
</tr>
<tr>
<td>Possible interpretations</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

*Using 10 tuberculin units (0.1 mL 100TU/mL [i.e. 1:1000 concentration]).
Part 2: Cases

- Not everyone who is vaccinated develops an immune response to tuberculin
- Those who do develop an immune response to tuberculin are not all protected against TB
- Even where there is an effect, the duration is not known
- There is no evidence it is effective in people >35 years

Vaccination also interferes with the use of tuberculin skin tests to look for TB infection. In view of the limitations of BCG vaccination it is no longer given to everyone in the UK, but is used selectively in:

- Tuberculin-negative new immigrants from high prevalence countries aged ≤35 years
- Tuberculin-negative household contacts of pulmonary TB
- Neonates of ethnic minority groups (e.g. Indian Subcontinent or Black African)
- Tuberculin-negative, previously unvaccinated healthcare workers
- Neonates born in areas of the UK with high TB prevalence (>40/100,000 cases per year)

How do you interpret Mrs Ahmed’s test results?

So far tests have not confirmed the diagnosis of TB as her three sputum samples are smear-negative and her Mantoux test is negative.

Are these test results surprising?

Her history and chest X-ray are strongly indicative of TB. The results are surprising but TB still remains the most likely diagnosis. False negative sputum microscopy can occur if the bacterial load is low or the sample is taken poorly. A false negative Mantoux test could indicate underlying immunosuppression (e.g. HIV).

What other investigations would help to make a diagnosis?

- Computed tomography (CT) scan of her chest to characterize the abnormality seen on X-ray and exclude less likely diagnoses including cancer and pulmonary embolus
- Sputum induction, i.e. physiotherapy to induce coughing after inhalation of hypertonic saline to obtain samples from deep in the lung

Mrs Ahmed’s chest CT confirms consolidation and cavitation in the right upper lobe (Fig. 86), consistent with pulmonary TB. She also has mediastinal lymph node enlargement. Sputum induction was unsuccessful. Bronchoscopic appearances were unremarkable, but microscopy of washings from the right upper lobe identified M. tuberculosis, which was fully sensitive to antituberculous treatment when cultured.

After bronchoscopy Mrs Ahmed commences quadruple antituberculous therapy with rifampicin, isoniazid, pyrazinamide and ethambutol.

What else must be done now a diagnosis of tuberculosis has been made?

- Tuberculosis is a notifiable disease, which means that there is a statutory requirement to inform the local ‘proper officer’, usually the Consultant in Communicable Disease Control, of any cases that have occurred. This information is collected by the Health Protection Agency and allows detection of TB outbreaks and monitoring of TB management
- People close to Mrs Ahmed may have become infected with M. tuberculosis. Her contacts must now be identified, traced, tested and treated as necessary

Mrs Ahmed is counselled for an HIV test which is found to be positive.
What is the significance of a positive HIV test?

A positive HIV test indicates that Mrs Ahmed has antibodies to and is infected with the human immunodeficiency virus (HIV).

HIV is a retrovirus that infects immune cells, particularly the CD4 (or T helper) lymphocyte. It replicates in and damages these cells:
- Directly during the release of virus from the cell
- By inducing apoptosis
- By marking the cell for destruction by other immune cells

As a consequence of HIV the CD4 count is reduced and this impairs cell-mediated immunity, increasing susceptibility of the affected person to infection with opportunistic pathogens and to tumour growth. Mrs Ahmed may have reactivated latent TB as her cell-mediated immunity became impaired by HIV.

How might Mrs Ahmed have contracted HIV?

HIV infection is very common in sub-Saharan Africa, although prevalence rates in Somalia, where Mrs Ahmed comes from, are relatively low. HIV is spread from person to person by contact with body fluids. The most common modes of transmission are:
- Sexual contact
- Parenteral exposure to infected needles or blood products
- Vertical transmission from mother to infant during delivery or breastfeeding

Why was Mrs Ahmed counselled before undergoing an HIV test?

When HIV was first discovered in the 1980s it was untreatable and the only benefit in knowing the diagnosis was reduced risk of transmission of virus to others. By contrast, there were many disadvantages to testing including concerns around confidentiality, insurance and legal issues as well as the negative emotional and social consequences of a positive result.

Testing has become more important since the development of highly active antiretroviral therapy (HAART), which prolongs survival and reduces morbidity from HIV. However, a positive HIV test can still have considerable physical, emotional and social implications and counselling before testing is still practiced.

How does HIV affect the respiratory system?

The effects of HIV on the respiratory system depend on the stage of the illness (Table 11). Once decline in cell-mediated immunity has begun the major respiratory infections associated with HIV are TB and pneumocystis pneumonia (Box 109).

Outcome. Mrs Ahmed is referred to an infectious diseases consultant who takes over her care. Her TB is treated first before she is given HAART for her HIV because of potential drug interactions. She improves rapidly on antituberculosis quadruple therapy and is discharged home to continue treatment. Contact and family testing for TB and HIV are planned.

Box 109 Pneumocystis pneumonia

Cause
Pneumocystis pneumonia is caused by the fungal organism Pneumocystis jirovecii (not P. carinii, a similar organism which affects rats). Despite this it is usually known as Pneumocystis carinii pneumonia (PCP). Pneumocystis is widespread in the environment and is not a pathogen to healthy individuals, but causes lung infection in people who are immunocompromised.

Pathology
Infection causes thickening of the alveoli and alveolar septae. This causes severe hypoxia which can be fatal if untreated.

Clinical features
Patients experience fever, cough and breathlessness. On examination they may be extremely hypoxic without chest signs or with inspiratory crepitations and wheezes.

Diagnosis
Successful diagnosis is more likely if clinicians maintain a high index of suspicion for PCP in immunocompromised patients.

A chest X-ray may show perihilar haziness resulting from alveolar thickening (Fig. 87).

Diagnosis is confirmed by visualization of the organism by microscopy of induced sputum or bronchial washings.

Treatment
PCP is treated with co-trimoxazole, a combination of trimethoprim and sulfamethoxazole antibiotics. Alternative drugs include pentamidine, trimethoprim-dapsone, clindamycin, primaquine and atovaquone.

Patients with hypoxia and moderate to severe PCP are given corticosteroids to reduce alveolar inflammation and hasten recovery.
Table 11 Respiratory effects of HIV infection.

<table>
<thead>
<tr>
<th>Illness stage</th>
<th>Respiratory effect</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acute HIV infection</strong></td>
<td>Flu-like illness with pharyngitis, lymphadenopathy and fever</td>
</tr>
<tr>
<td>Initial infection with seroconversion</td>
<td></td>
</tr>
<tr>
<td><strong>Latency</strong></td>
<td></td>
</tr>
<tr>
<td>Stage I*</td>
<td>No effect</td>
</tr>
<tr>
<td>Stable CD4 count and HIV viral load</td>
<td></td>
</tr>
<tr>
<td><strong>Stage II</strong></td>
<td>Recurrent upper respiratory tract infections including sinusitis, pharyngitis, bronchitis</td>
</tr>
<tr>
<td><strong>Stage III</strong></td>
<td>Pulmonary tuberculosis</td>
</tr>
<tr>
<td>Progressive decline in CD4 count</td>
<td></td>
</tr>
<tr>
<td>and increase in HIV load</td>
<td></td>
</tr>
<tr>
<td><strong>Stage IV</strong></td>
<td>Opportunistic lung infections including candidiasis, pneumocystis pneumonia</td>
</tr>
<tr>
<td></td>
<td>AIDS</td>
</tr>
<tr>
<td></td>
<td>Kaposi’s sarcoma</td>
</tr>
</tbody>
</table>

*World Health Organization (WHO) classification.

Mrs Ahmed is referred for urgent assessment of fever, weight loss and haemoptysis. Her chest X-ray demonstrates consolidation and cavitation in the right upper lobe. As TB seems likely she is admitted to a side room to avoid spread of infection. Initial investigations do not confirm TB and her negative tuberculin test raises the possibility of a false negative result because of coexistent HIV infection. Subsequently, *M. tuberculosis* is seen in bronchial washings and she is started on quadruple anti-tuberculous treatment. She is also found to be HIV positive and is referred to the infectious diseases consultant for consideration of HAART.

Figure 87 *Pneumocystis* pneumonia.
Tuberculosis should be suspected in all patients presenting with night sweats, weight loss or haemoptysis

If a patient is suspected of having active pulmonary TB they should be cared for in a side room to prevent cross-infection

Risk factors for TB include:

- Previous exposure
- Impaired immunity, reactivating previous infection

Initial diagnostic tests for pulmonary TB should include:

- Chest X-ray
- Three early morning sputum samples for microscopy and culture
- Tuberculin skin testing

If these are unhelpful other useful tests for pulmonary TB include:

- Induced sputum for microscopy and culture
- Fibreoptic bronchoscopy with bronchial washings for microscopy and culture

Once the diagnosis of pulmonary TB has been made:

- The patient should be started on appropriate treatment
- The case must be notified to the Health Protection Agency via the local officer
- Contact tracing should be performed to identify, screen and treat close contacts as necessary

Co-infection with HIV and *M. tuberculosis* is common and HIV testing should be considered in all patients with TB

HAART prolongs life and reduces morbidity in patients with HIV

The most common pulmonary manifestations of AIDS are TB and pneumocystis pneumonia