CASE REPORT

Unusual case of a lung abscess

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gaurigodbole@doctors.org.uk**SUMMARY**

A 56-year-old Caucasian lady presented with a short history of pleuritic chest pain on the background of a 2-month history of fever, chills, 10-kg weight loss and cough with brown sputum after a laparoscopic cholecystectomy. She had persistent eosinophilia and was diagnosed with a lung abscess as seen on chest x-ray. She did not respond to standard intravenous broad spectrum antibacterial medication and her chest CT scan showed a moderate pleural collection in continuity with the abscess. She also underwent bronchoscopy, the microscopy of the bronchial washings revealing eggs of the trematode *Fasciola*. The bacterial and fungal cultures of the washings were sterile. She had visited Turkey in the previous year but did not remember consuming any watercress or aquatic plants. She was successfully treated with two doses of the antiparasitic agent triclabendazole. Ectopic *Fasciola* can be a rare cause of a lung abscess.

BACKGROUND

Hepatic *Fasciola* is common parasitic infection worldwide, acquired from eating contaminated aquatic vegetables. It can present with gastritis, hepatitis or cholangitis, although some patients can be asymptomatic. It is very rare that ectopic infections present as a lung abscess or empyema. A patient with eosinophilia and no other risk factors for a bacterial lung abscess who is failing standard therapy should prompt attending physicians to the possibility of parasitic infection.

CASE PRESENTATION

A 56-year-old Caucasian lady attended casualty complaining of right-sided, sharp, non-radiating chest pain aggravated by deep breaths and movement. She also had a history of shortness of breath on minimal exertion, a low-grade fever with chills, a cough with foul-tasting sputum and 10 kg weight loss over the preceding 2 months. Eight days previously, she had been seen in the respiratory clinic for the above-mentioned symptoms, all of which started after she underwent a laparoscopic cholecystectomy and cholangiogram for multiple symptomatic gallstones. There was no other relevant past or family history. She consumed 20 units of alcohol per week and did not smoke. She had been started on a short course of co-amoxiclav by her general practitioner which helped reduce the amount and frequency of her sputum production.

When she was examined in the respiratory clinic, she was afebrile, not tachycardic or tachypnoeic and her chest had bilateral air entry without additional sounds. The heart sounds were normal, the abdomen was soft and non-tender, the

cholecystectomy scar was well-healed and there was no evidence of hepatosplenomegaly.

Investigations showed a normal haemoglobin 13.9 g/dl, white cell count $8.3 \times 10^9/l$, neutrophils $4.28 \times 10^9/l$, lymphocytes $1.74 \times 10^9/l$, monocytes $0.56 \times 10^9/l$ and eosinophils $1.66 \times 10^9/l$, erythrocyte sedimentation rate 18 and C reactive protein <3.

A chest radiograph (x-ray) (figure 1) revealed a large cavitating mass with an air fluid level in the right upper lobe suggestive of a lung abscess.

She was keen to avoid hospital admission in order to attend her daughter's wedding, and therefore, after extensive discussion, she was started on a 6-week course of oral antibiotics with a plan to repeat her chest x-ray in 1 week. Sputum samples were requested and an appointment was made for outpatient bronchoscopy.

Unfortunately, she became acutely unwell and presented to casualty as documented above. There were no new findings on clinical examination. Blood tests showed an elevated white cell count of $16.0 \times 10^9/l$, neutrophils $12.42 \times 10^9/l$ and eosinophils $1.23 \times 10^9/l$. She was given appropriate analgesia and remained comfortable awaiting the prearranged bronchoscopy. The chest x-ray revealed that the cavitating mass in the right upper lobe had increased in size and therefore the patient was started on intravenous piperacillin-tazobactam and metronidazole.

The bronchoscopy was performed without complication, the airways were predominantly normal except the posterior right upper lobe from which a small amount of purulent material was seen; microscopy of the bronchial washings revealed a moderate number of pus cells, no acid-fast bacilli and the bacterial and fungal cultures were negative. Other investigations including antglomerular

Figure 1 Chest x-ray.

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Figure 2 CT scan of the chest.

basement membrane antibodies, antinuclear cytoplasmic antibody screen and antinuclear antibodies were all negative.

However, her pleuritic chest pain got progressively worse, and although she remained afebrile, her physical examination revealed decreased air entry in the right base. CT of the chest (figure 2) showed the right upper lobe cavitating lesion with an air fluid level and a moderate right pleural effusion, suggesting a rupture of the abscess into the pleural cavity. A large bore chest drain was inserted with thick yellow fluid being drained from the pleural space. The pleural fluid pH was 6.9 and the fluid was sent for microbiological tests. In the meantime, the histopathology of the bronchial alveolar lavage was available; it showed the presence of eosinophils and Charcot-Leyden crystals and parasite eggs. There was no evidence of neoplasia. The samples were forwarded to the parasitology reference laboratory for further review.

The patient's travel history was revisited at this point, and she admitted to visiting Turkey 1 year previously and Vietnam/Thailand 4 years prior to the onset of illness. She could not remember any unusual dietary choices during her travel.

The previous hepatic imaging was also reviewed; the liver scan had shown gallstones with a normal biliary tract, as well as numerous hyperechoic areas in the left lobe, suggestive of multiple liver cysts; the laparoscopic image of the liver is shown (figure 3). The histology of the excised gallbladder revealed no evidence of parasitic infection, but had evidence of cholecystitis with transmural eosinophilia and cholesterosis. Specialist advice was sought from the local infectious diseases team and the differential diagnosis for parasitic infections causing lung abscess discussed. Stool samples and parasitic serology were sent and a course of diloxanide furaoate was added to her antimicrobial regimen to treat any intestinal amoebic cysts.

Figure 3 Hepatic surface with cystic nodular lesions (laparoscopic view).

Figure 4 Operculated eggs of *Fasciola* sp seen in the bronchial fluid (direct microscopy).

INVESTIGATIONS

The microscopy of the bronchial lavage reported by the parasitology reference laboratory showed multiple yellowish brown, operculated eggs measuring approximately $140\ \mu\text{m} \times 80\ \mu\text{m}$; pathognomonic for *Fasciola* sp (figures 4 and 5).

The *Fasciola* serology (IFAT for IgG detection) was positive at a titre of 1:256 (cut-off 1:40).

Strongyloides, amoebic and filarial serology were negative.

Stool microscopy examination for ova, cysts and parasites was normal.

DIFFERENTIAL DIAGNOSIS

Parasitic infections causing eosinophilia and a lung abscess include

- *Paragonimus* (lung fluke). A known cause of eosinophilia and a lung abscess in travellers to South East Asia and West Africa. The ova can be detected in the sputum; however, they are smaller ($80\text{--}120 \times 45\text{--}70\ \mu\text{m}$) and have a thicker shell in comparison to the ova of *Fasciola* sp.¹
- *Echinococcus* (pulmonary hydatid). Primarily cystic disease with secondary bacterial infection.

(*Entamoeba histolytica* or invasive amoebiasis can cause liver abscess but is not associated with eosinophilia and *Strongyloides stercoralis* causes eosinophilia but not a lung abscess).

Figure 5 Eggs of *Fasciola* sp.

TREATMENT

The patient was treated for ectopic *Fasciola* infection with oral triclabendazole, and two doses of 10 mg/kg were given 24 h apart. Hyoscine was given prior to the antiparasitic agent to prevent biliary colic which is seen in cases with a heavy load of flukes in the liver.

OUTCOME AND FOLLOW-UP

The pleural effusion reaccumulated and a chest drain was reinserted. The pleural fluid revealed reactive mesothelial cells, acute and chronic inflammatory cells with excess eosinophils, no parasitic eggs and no evidence of malignancy. The features were consistent with a reactive effusion secondary to a parasitic infection. Piperacillin-tazobactam was continued to treat any coexistent bacterial infection. However, after two doses of triclabendazole, the patient soon responded and the chest drain was removed. She remains clinically well at present. The *Fasciola* serology was repeated after 2 months of treatment and was still positive at the same titre.

DISCUSSION

Fasciola is a common trematode infection found worldwide,^{1 2} including the UK; the two predominant species causing human infection include *Fasciola hepatica* and *Fasciola gigantica*. It was the first trematode to be described in 1379 by de Brie. *F hepatica* (the sheep liver fluke) is found across temperate and subtropical countries (Europe, the Middle East and Central and South America) and *F gigantica* (mainly cattle fluke) is more common in South East Asia and Africa, and the two can coexist. Humans are the accidental hosts; infection occurs due to consumption of watercress and other aquatic plants growing in water contaminated with animal faeces. Unembryonated eggs are laid into the water by infected sheep, the eggs embryonate in water and the larvae enter the snails (intermediate host). The cercariae leave the snail and encyst to form metacercariae which attach to the aquatic vegetation, which is then consumed by sheep and sometimes humans. The metacercariae excyst in the duodenum. In the acute stage or hepatic stage, the parasite larvae then pass through the wall of the intestines into the peritoneal cavity, through the hepatic capsule, into the liver. The larvae reside in the hepatic parenchyma (hepatic stage) and cause cavitation, cysts and hepatic tracts. The host usually suffers from fever on and off, right upper quadrant pain, urticaria and tiredness. They have anaemia, eosinophilia (prominent feature) and may have transaminitis. The larvae then migrate to the bile ducts (biliary stage) and can cause severe inflammation of the biliary tree, causing repeated attacks of cholecystitis or cholangitis. Some patients may be asymptomatic for years in this stage, whereas others may have recurrent attacks.³ The larvae mature to form flukes which lay eggs, which are then excreted in bile and eventually the stools. Rarely, the flukes migrate outside the liver and may reside in other organs (ectopic fascioliasis) like the abdominal wall, lungs, heart, brain, skin, muscles and the genitourinary system.^{1 3 4} *F hepatica* more commonly cause ectopic infection.⁴ Other complications include pancreatitis, cirrhosis and portal hypertension.³

A large number of patients may be asymptomatic and many may present years after exposure.

Most patients have eosinophilia and a few may have hepatitis.

Diagnosis is based on finding characteristic ellipsoidal, yellowish brown, operculated eggs in the stool (130–150 µm×60–90 µm) on microscopy or by visualisation of the flukes in the biliary tract during endoscopic retrograde cholangiopancreatography. Eggs of

both species look similar and cannot be differentiated on microscopy.¹

Fasciola serology (immunofluorescence antibody test for *Fasciola* IgG) is usually positive in the immunocompetent patients.^{1 3}

Imaging of the liver may show ‘tunnels and caves’ in peripheral parts of the liver due to the tracts made by the flukes³ in the hepatic phase. These migrating trematodes produce micro-abscesses which produce hypoechoic nodules and cystic lesions on ultrasound, and nodular or linear hypodensities on CT which enhance homogeneously with dye. Although the nodular lesions are non-specific, the linear tracks are diagnostic of hepatic fascioliasis. In the biliary stage, ultrasound or CT may show biliary dilation and bile duct wall thickening suggesting biliary fascioliasis.⁵

Treatment consists of 1–2 doses of triclabendazole 10 mg/kg orally.¹ Keiser *et al*⁶ found an egg clearance rate of 92–100% after two doses of triclabendazole.⁷ Other agents which may be used are nitazoxanide¹ or bithionol.^{3 7} An antispasmodic agent is usually given prior to the treatment which prevents an attack of biliary colic, and the agent can be precipitated when the parasites die within the biliary tract. Antibiotics may be necessary to treat secondary bacterial infection in cholecystitis and cholangitis. Stools should be rechecked 4 weeks after treatment to confirm clearance of infection.

Our patient was a traveller to Europe, but did not remember consuming specific vegetables or exposure to contaminated water. She initially presented with hepatobiliary symptoms and the hepatic surface showed non-specific nodules or cysts on laparoscopy. She had eosinophilia and possibly developed a lung abscess as the flukes migrated into the lung. The lung parenchyma is far more porous than the liver, giving the flukes an ample chance to form a pulmonary inflammatory mass or an abscess. The rupture of the lung abscess into the pleural space led to a further relapse. Symptoms dramatically resolved following treatment with triclabendazole.

The presentation of a lung abscess in a previously fit and healthy patient without obvious risk factors of age, smoking, intravenous drug use, alcoholism, poor dentition, aspiration risk or any features of infective endocarditis should make one explore other possibilities including parasitic infections. A large number of people might have travelled to various parts of the world with obvious known and unknown risks. A high index of suspicion is required to reach the correct diagnosis.

Learning points

- ▶ A high suspicion for parasitic infection should be maintained in patients with lung abscess not responding to conventional therapy in the context of eosinophilia.
- ▶ A careful travel and dietary history should be obtained from all patients.
- ▶ What was initially thought to be a complication of a fairly standard surgical/anaesthetic procedure actually turned out to be an uncommon pathology; always seek specialist advice in complicated cases with recurrent symptoms and review all previous imaging and pathology.

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