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CASE REPORT

Primary hepatic actinomycosis

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Summary Actinomycotic liver abscess was diagnosed in a 35-year-old alcoholic farmer from southern India with tender hepatomegaly and fever. CT of the abdomen revealed three coalescing hypodense lesions in the liver. The causative organism could be demonstrated on direct microscopy and cultured from the pus. Treatment with i.v. penicillin for 2 months and oral ampicillin for 5 months resulted in cure as evidenced by clinical improvement and radiological disappearance of the lesions.

1. Introduction

Actinomycosis is a chronic infection characterised by abscess formation, draining sinuses and tissue fibrosis. It is caused by microaerophilic or anaerobic strains of filamentous non-sporing organisms belonging to the genus *Actinomyces*. Cervicofacial, thoracic and abdominopelvic varieties of actinomycosis are commonly seen (Smego and Foglia, 1998). Hepatic actinomycosis is usually secondary to abdominal or thoracic infections with the organism. However, it may be a primary infection without a demonstrable lesion elsewhere (Kazmi and Rab, 1989). Actinomycosis primarily affecting the liver is rare and has a grave prognosis (Suvarna Kumari et al., 1970). Here we report a case of primary hepatic actinomycosis in an alcoholic farmer from southern India, who was cured of the disease.

2. Case report

A 35-year-old male farmer from Malur town, Kolar district, Karnataka, India, presented at R.L. Jalappa Hospital during November 2003 with fever of one and a half month's duration, pain in the abdomen, vomiting and loss of appetite for 7 days.

The patient was a chronic alcoholic who had consumed approximately 90 g of alcohol per day for the past 10 years. He had not undergone any abdominal surgery. There was no history of diarrhoea or dysentery. On examination, the patient was febrile (104 °F) with a pulse rate of 104/min. His blood pressure was 110/70 mmHg and he was pale. On systemic examination the liver was enlarged 8 cm below the right costal margin,

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soft in consistency and tender. The patient also had proximal muscle weakness in both lower limbs.

His total white blood cell count was 7900 cells/mm^3 (neutrophils, 79%; lymphocytes, 18%; eosinophils, 2%; monocytes, 1%). Haemoglobin was 10.4 g% and the erythrocyte sedimentation rate was 115 mm/h. Liver function tests showed an aspartate aminotransferase level of 77 IU/l, an alanine aminotransferase level of 130 IU/l, alkaline phosphatase 532 U/l and gamma glutamyl transferase 94 U/l; total protein was 6.2 g/dl, albumin 2.8 g/dl and globulin 3.4 g/dl (albumin/globulin ratio of 0.8). Chest radiography showed a raised right dome of the diaphragm. CT of the abdomen showed three hypodense coalescing lesions in the right lobe of the liver close to the surface and measuring 10 cm × 5 cm when taken together (Figure 1). Considering these findings, a provisional diagnosis of pyogenic liver abscess and alcohol-induced proximal myopathy was made. The patient was administered tinidazole 800 mg per day and cefotaxime 1 g twice a day i.v. along with paracetamol, antacids and vitamins.

On the fourth day of admission the patient continued to have spikes of fever and pain in the abdomen; tenderness increased in the right hypochondrium, with bulging of the abdominal wall. Ultrasound-guided percutaneous aspiration of the liver abscess was performed and approximately 200 ml of thick, greenish yellow, foul smelling pus containing dark brown granules was aspirated (Figure 2). Gram stain of the pus showed numerous pus cells and thin filamentous, non-acid fast, branching Gram-positive organisms, breaking into coccoid and bacillary fragments at places. The pus was inoculated onto blood agar, chocolate agar and MacConkey's agar and incubated aerobically. The sample was also inoculated into Thioglycollate broth and brain–heart infusion (BHI) broth, which were incubated anaerobically (Gaspac; Himedia Laboratories, Mumbai, India). Aerobic culture yielded no growth. After 8 days of incubation in the anaerobic jar, Thioglycollate broth and BHI broth showed growth as granular turbidity with fluffy balls. Gram staining of the broth culture showed Gram-positive branching filamentous organisms (Figure 2, insert). The broth cultures were subcultured onto BHI agar, which was incubated anaerobically; however, there was no growth. The organism was identified as *Actinomyces* species and a diagnosis of actinomycotic liver abscess was made in light of the above findings. Colonoscopy and upper gastrointestinal endoscopy were performed to detect primary actinomycotic lesions in the intestine; however, no such lesions could be detected.

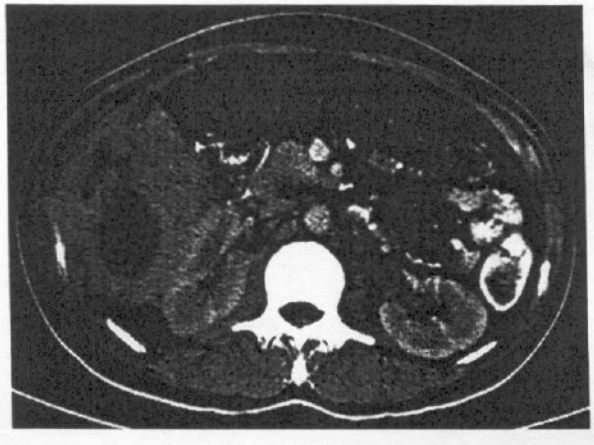

Figure 1 Contrast-enhanced CT of the abdomen showing the liver with three coalescing abscesses.

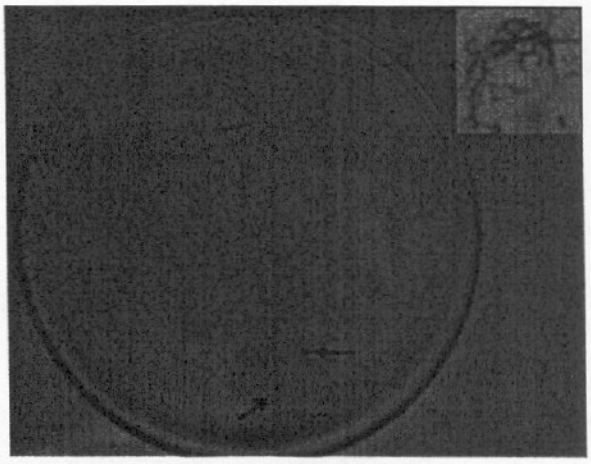

Figure 2 Petri dish containing pus aspirated from the abscess. Arrows indicate granules. Insert: microscopic picture of Gram-positive branching filamentous organism grown in broth culture.

The patient was administered crystalline penicillin 2 000 000 units i.v. every 2 h. He became afebrile within a week. Liver size and tenderness reduced in 3 weeks. The patient was discharged after a month's treatment and was advised to continue amoxicillin 2 g per day in divided doses.

The patient returned after a month with complaints of pain in the abdomen and a bulge over the right hypochondrium. Ultrasound examination showed a track extending from the liver abscess into the abdominal wall. Crystalline penicillin 2 000 000 units i.v. every 2 h was re-started and was continued for another month. The patient showed symptomatic improvement. Ultrasound examination showed disappearance of the track. He was discharged with advice to continue ampicillin 2 g/day in divided doses. After 4 months of treatment, an ultrasound examination revealed a normal liver. On further follow-up for 6 months, the patient continued to be asymptomatic.

3. Discussion

Actinomycotic abscess of the liver is a rare condition (Sherlock, 1997). It is usually seen in males in the fourth and fifth decades (Miyamoto and Fang, 1993). *Actinomyces* species are found as commensal flora in the gastrointestinal tract. They invade the adjacent tissues and bloodstream when normal anatomical barriers are disrupted. Conditions such as intravenous drug abuse, alcoholism, peptic ulcer, biliary tract disease and recent appendicitis predispose to hepatic actinomycosis. However, spread to the liver has also been documented without any disruption of tissue barriers (Miyamoto and Fang, 1993).

Our patient presented with tender hepatomegaly and fever. The ultrasound examination showed multiple hypoechoic coalescing lesions consistent with a diagnosis of pyogenic liver abscess. However, microscopic examination of the pus and culture established the actinomycotic aetiology. We could not demonstrate any primary lesion in the gastrointestinal tract, thus the diagnosis in our patient conforms to primary hepatic actinomycosis.

Despite extensive research, we could find only one case of primary hepatic actinomycosis reported from India (Suvarna Kumari et al., 1970). In India, amoebic liver abscess is more common and presents with clinical features similar to that of hepatic actinomycosis (Kazmi and Rab, 1989). It is possible that some of the cases of hepatic actinomycosis might have been misdiagnosed as amoebic liver abscess. Often, diagnosis of hepatic actinomycosis missed during life has been brought to light during autopsy (Kazmi and Rab, 1989; Suvarna Kumari et al., 1970). We emphasise that hepatic actinomycosis should be kept in mind as one of the causes for liver abscess.

Standard therapy for actinomycosis requires large doses of penicillin given parenterally for 1 month followed by oral administration for a prolonged period. As patients with hepatic actinomycosis tend to have long-term relapses, they need to be followed for a long time and cure confirmed (Miyamoto and Fang, 1993; Mohr et al., 1970). Lincomycin has been used successfully in patients allergic to penicillin (Mohr et al., 1970). Fortunately, our patient responded to i.v. penicillin followed by a prolonged course of oral ampicillin and was cured of the disease.

This report emphasises that hepatic actinomycosis needs to be considered in the differential diagnosis of liver abscess, despite its rarity and reported grave prognosis. If diagnosed promptly by microscopy and anaerobic culture, it is a curable condition amenable to penicillin therapy.

Conflicts of interest statement

The authors have no conflicts of interest concerning the work reported in this paper.

References

Kazmi, K.A., Rab, S.M., 1989. Primary hepatic actinomycosis: a diagnostic problem. Am. J. Trop. Med. Hyg. 40, 310–311.

Miyamoto, M.I., Fang, F.C., 1993. Pyogenic liver abscess involving *Actinomyces*: case report and review. Clin. Infect. Dis. 16, 303–309.

Mohr, J.A., Rhoades, E.R., Muchmore, H.G., 1970. Actinomycosis treated with lincomycin. JAMA 212, 2260–2262.

Sherlock, S., 1997. The liver in infections, in: Sherlock, S., Dooley, J. (Eds), Diseases of the Liver and Biliary System. Blackwell Science Ltd, UK, pp. 497–529.

Smego Jr, R.A., Foglia, G., 1998. Actinomycosis. Clin. Infect. Dis. 26, 1255–1263.

Suvarna Kumari, G., Leelanaidu, P.S., Reddy, C.R.M., Rao, N.R., Ramasubbaiah, Y., 1970. Actinomycosis of liver. J. Assoc. Physicians India 18, 375–377.