

Figure 2: Gastroscopy showing diffuse suppurative ulcers in the duodenum


Figure 3: Gastric contents showed a larva of Strongyloides stercolaris

## Discussion

Diagnosis of UGIB due to S. stercolaris infection is difficult and easily missed given its rarity. In this case, the diagnosis was made after two weeks of hematemesis and three gastroscopy attempts. The identification of a larva of S. stercolaris is a crucial diagnostic clue and can be isolated from the gastric contents microscopically. Detection of its eggs in stool examination is also helpful.

There are no pathognomonic gastroscopic findings. A report from Malaysia showed edematous mucosa at the prepyloric area. ${ }^{5}$ A bleeding gastric ulcer with eggs and larvae identified in the mucosal layer was also reported. In our patient the gastroscopic findings were non-specific but showed ulcers throughout the upper gastrointestinal area from stomach to the duodenum. Suppurative ulcers may occur due to
superimposed bacterial infection. Eosinophilic infiltration may be another clue for gastric strongyloidiasis.

Even though there is no specific clinical manifestation for gastric strongyloidiasis, some suggestive features are important. Most previous reports of UBIG caused by S. stercolaris are always severe. ${ }^{2,3}$ In addition, the patients also always have co-morbid conditions such as corticosteroid use, malignancy, organ transplant or HIV infection. Physicians in tropical areas should be aware of S. stercolaris associated UGIB given its difficult diagnosis, non-specific gastroscopic findings and severity of UGIB.

## WATTANA SUKEEPAISARNJAROEN KITTISAK SAWANYAWISUTH

Correspondence: Dr Kittisak Sawanyawisuth Department of Medicine, Faculty of Medicine, and Research Center in Back, Neck, Other Joint Pain and Human Performance (BNOJPH),

Khon Kaen University,
Khon Kaen 40002, Thailand
Email: kittisak@kku.ac.th

## References

1. Anantaphruti MT, Nuamtanong S, Muennoo C, Sanguankiat S, Pubampen S. Strongyloides stercoralis infection and chronological changes of other soil-transmitted helminthiases in an endemic area of southern Thailand. Southeast Asian J Trop Med Public Health. 2000;31:378-82.
2. Bollela VR, Feliciano C, Teixeira AC, Junqueira AC, Rossi MA. Fulminant gastrointestinal hemorrhage due to Strongyloides stercoralis hyperinfection in an AIDS patient. Rev Soc Bras Med Trop. 2013;46:111-3.
3. Bhatt BD, Cappell MS, Smilow PC, Das KM. Recurrent massive upper gastrointestinal hemorrhage due to Strongyloides stercoralis infection. Am J Gastroenterol. 1990;85:1034-6.
4. Dees A, Batenburg PL, Umar HM, Menon RS, Verweij J. Strongyloides stercoralis associated with a bleeding gastric ulcer. Gut. 1990;31:1414-5.
5. Shekhar KC, Krishnan R, Pathmanathan R, Fook CS. Gastric strongyloidiasis in a Malaysian patient. Southeast Asian J Trop Med Public Health. 1997;28:158-60.

Melioidosis: an unusual cause of isolated liver abscess

## Introduction

Liver abscess is a common clinical problem encountered in the tropics. In India, most cases are due to amebiasis or are pyogenic, secondary to portal pyemia. In appropriate clinical settings, anti-amebic therapy or empirical antibiotic therapy is instituted. If there is no clinical improvement, image guided abscess drainage or aspiration is done for microbiological analysis and treatment is modified accordingly. We describe two such patients who had failed to improve with initial empirical therapy for amebiasis or pyogenic liver abscess and were diagnosed to have melioidosis.

## Case reports

Case 1

A 51-year-old diabetic man from Orissa presented with a history of low-grade fever, progressive left-lower-quadrant abdominal pain for three months. He was treated in another hospital for pyogenic liver abscess with intravenous piperacillin/ tazobactam based on blood culture report positive for Pseudomonas spp. Abdominal examination revealed non-tender hepatosplenomegaly. Blood investigations revealed anemia (hemoglobin: $9 \mathrm{gm} / \mathrm{dl}$ ), normal WBC count $\left(6,000 / \mathrm{mm}^{3}\right)$, raised erythrocyte sedimentation rate ( 80 mm in 1 hr ), hypoalbuminemia $(2.5 \mathrm{gm} / \mathrm{dl})$, and elevated alkaline phosphatase ( $888 \mathrm{U} / \mathrm{L}$ ). Remaining liver function tests were within normal limits. The chest radiograph was normal. CECT abdomen showed the presence of multiple liver abscesses with subdiaphragmatic collections (Figures 1 \& 2). Blood culture grew Burkholderia pseudomallei. Ceftazidime was instituted as per antimicrobial sensitivity. In view of the subdiaphragmatic collection, laparoscopic drainage of the liver abscesses was done. The patient gradually recovered and was discharged on cotrimoxazole for 6 months.

## Case 2

A 50-year-old farmer from Tirupathi, with diabetes mellitus, was admitted with fever, abdominal pain and vomiting for two months. He was treated at another hospital for liver abscess with intravenous metronidazole and USG guided abscess drainage was done with little improvement. On examination, he had tachycardia with hypotension and tenderness in the right hypochondrium. Investigations revealed anemia, leukocytosis, low platelet count, altered LFT (increased AST, GGTP and ALP,


Figure 1: CT abdomen showing multiple cystic lesions with peripheral enhancement in the right lobe of liver. The largest cyst located in segment 8 , measures about 7 cm in diameter


Figure 2: CT abdomen showing a segment 5 lesion, which has ruptured into the anterior abdominal wall forming a collection in the intramuscular compartment. One of the segment 8 lesions also appears to have ruptured through the superior capsule of liver forming a small subcapsular collection
and hypoalbuminemia). Chest radiograph showed right lower zone non-homogenous opacity. USG abdomen showed a mixed echogenic heterogeneous lesion with multiple anechoic to hypoechoic areas within the right lobe of liver. Splenomegaly with portal and superior mesenteric venous thrombosis was also noted (Figure 3). USG guided aspiration of the liver lesion was sent for culture. He was initially resuscitated with intravenous fluids, inotropes, and empirical intravenous cefaperazone/ sulbactam. Pus culture grew Burkholderia pseudomallei. The antibiotic was changed to ceftazidime and


Figure 3: USG abdomen showing multiple hypoechoeic lesions located predominantly in the right lobe of the liver, with the largest measuring $2.7 \times 2.6 \mathrm{~cm}$ in size
cotrimoxazole. He gradually improved and was advised to continue cotrimoxazole for 6 months.

## Discussion

Burkholderia pseudomallei (formerly, Pseudomonas pseudomallei) is a bipolar-staining, Gram-negative aerobic bacillus and is found in the tropics, mainly between latitudes $20^{\circ}$ north and $20^{\circ}$ south. ${ }^{1}$ It was first recognised in Burma in 1912 by Whitemore and Krishnaswami. ${ }^{2,3}$ The organism enters the human host through a preexisting skin abrasion or by inhalation and ingestion. It resides in the soil and water in locations such ascultivated fields, drains, gardens and playgrounds. ${ }^{4}$ Melioidosis occurs more frequently in patients with underlying diseases, such as diabetes mellitus, chronic renal failure, alcoholism, malignancy or haematological disorders, or thosewho are immunosuppressed as the result of either disease ortherapy. In patients without underlying disease, occupational exposure to soil is an important risk factor. ${ }^{5,6}$ Both our patients were diabetic and farmers.

The clinical manifestations of melioidosis range from acute localized forms, acute septicemia to chronic forms. Septicemia is the most common presentation. The lung is the most commonly affected organ. ${ }^{1.5}$ The spleen is the most common intra-abdominal organ infected by melioidosis, followed by the liver and kidney. ${ }^{6}$ Many reports of melioidosis with abscessesat unusual sites such as the neck ${ }^{7}$ parotid, ${ }^{8}$ have been published from India.Visceral organ abscesses appear on ultrasoundas multiple and small hypoechoic lesions, target lesions and multiloculated lesions.On CT, the "necklace sign" or
the" honeycomb sign" with multiloculated lesions are characteristic of melioidotic abscesses. ${ }^{9}$ Concurrent liver and splenic abscesses are more likely to be associated with melioidosis than other micro organisms. Definitive diagnosis is made by culture and isolation of the organism from blood or infected organ. ${ }^{4,9}$ The treatment of choice is intravenous ceftazidime (or) meropenem 6-8 hourly followed by 3-6 months of co-trimoxazole (or) doxycycline therapy. The mortality rate in the acute septicemic form exceeds $50 \%$ if patients do not receive appropriate treatment. ${ }^{4,6}$

In summary, melioidosis should be thought of and evaluated for in patients with underlying risk factors who present with multiple liver abscesses. If appropriate treatment is not instituted, there is high risk of mortality.

## PIRAMANAYAGAM PARAMASIVAN

Correspondence: Dr. PiramanayagamParamasivan Department of Gastroenterology, Apollo Hospitals, Chennai - 600006, India
Email: piraman2000@yahoo.co.in

## References

1. Cheng AC, Currie BJ. Melioidosis: epidemiology, pathophysiology, and management. Clin Microbiol Rev. 2005;18:383-416.
2. Whitmore A. An Account of a Glanders-like Disease occurring in Rangoon. J Hyg (Lond). 1913;13:1-34.
3. Whitmore A, Krishnaswami CS. An account of the discoveryof a hitherto underscribed infective disease occurring among the populationof Rangoon. Ind Med Gazette. 1912;47:262-7.
4. Limmathurotsakul D, Peacock SJ. Melioidosis: a clinical overview. Br Med Bull. 2011;99:125-39.
5. Currie BJ, Jacups SP, Cheng AC, Fisher DA, Anstey NM, Huffam SE, et al. Melioidosis epidemiology and risk factors from a prospective whole-population study in northern Australia. Trop Med Int Health. 2004;9:1167-74.
6. Currie BJ, Ward L, Cheng AC. The epidemiology and clinical spectrum of melioidosis: 540 cases from the 20 year Darwin prospective study. PLoSNegl Trop Dis. 2010;4:e900.
7. Mathew S, Perakath B, Mathew G, Sitaram V, Nair A, Lalitha MK, et al. Surgical presentation of melioidosis in India. Natl Med J India. 1999;12:59-61.
8. Shivbalan S, Reddy N, Tiru V, Thomas K. Systemic melioidosis presenting as suppurativeparotitis. Indian Pediatr. 2010;47:799-801.
9. Apisarnthanarak P, Thairatananon A, Muangsomboon K, Lu DS, Mundy LM, Apisarnthanarak A. Computed tomography characteristics of hepatic and splenic abscesses associated with melioidosis: a 7 -year study. J Med Imaging Radiat Oncol. 2011;55:176-82.
