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CASE REPORT

Edwardsiella tarda septicemia with underlying multiple liver abscesses

RM Yousuf *MD*, SH How *M Med*, M Amran *M Radiology*, KT Hla *FRCS (Ed)*, A Shah *MSc*, and A Francis* *MSc (Microbiology)*

*Department of Internal Medicine, Kulliyyah of Medicine, International Islamic University Malaysia and *Department of Microbiology, Hospital Tengku Ampuan Afzan Kuantan.*

Abstract

Edwardsiella tarda has recently been described as a member of the family *Enterobacteriaceae*. The genus *Edwardsiella* contains three species; *E. hoshinae, E. ictaluri* and *E. tarda*. *Edwardsiella tarda* is the only species which has been recognised as pathogenic to humans, especially in those with an underlying disease. The most common presentation is watery diarrhoea. Extra intestinal infections have been reported infrequently. Humans seem to be infected or colonised with *Edwardsiella* through ingestion or inoculation of a wound. This report is of a patient with multiple liver abscesses due to *E. tarda* who later developed bacterial peritonitis and septicaemic shock.

Key words: *Edwardsiella tarda*, liver abscesses

INTRODUCTION

The genus *Edwardsiella* was first described by Ewing in 1965 and consisted of a single species, *Edwardsiella tarda*, until 1980-1981 when two other species, *Edwardsiella hoshinae* and *Edwardsiella ictaluri*, were added to the genus. *E. tarda* is the most common of the three, and is the only species which has recently been implicated in human disease. The organism is widely distributed in nature. It is common in tropical and subtropical environments and appears to be spread by contact with infected marine life, including ornamental fish and turtles, or by eating raw fish.[1]

E. tarda is an oxidase-negative, catalase-positive, facultative, anaerobic, motile, Gram-negative bacillus. Certain biochemical properties are useful in distinguishing *E. tarda* from other *Enterobacteriaceae* such as *Salmonella* and *Proteus* species. The non-lactose fermenting colonies of *E. tarda* produce hydrogen sulphide and indole but do not produce D-manitol, urease, oxidase and D-sorbital.

E. tarda causes illness in both humans and animals. The asymptomatic carrier state is rare but documented.[2] Humans are regarded as an occasional host, and are prone to suffer from serious disease. *E. tarda* most frequently causes gastroenteritis with acute watery diarrhoea[3], but dysentery-like presentations also occur.[4] We recently encountered a case of multiple liver abscesses complicated by peritonitis due to *E. tarda* infection without any predisposing illness.

CASE REPORT

A 27-year-old Indonesian male presented at the Accident and Emergency Department of the Hospital Tengku Ampuan Afzan, Kuantan, Pahang with a two-week history of fever, chills and rigors associated with right upper abdominal pain. The fever was intermittent and was associated with generalised body weakness. He had about four to five loose stools/day alternating with constipation but gave no history of vomiting or yellow discoloration of the eyes. He was admitted to the hospital for further investigation. He did not have any history of chronic illnesses like liver disease, diabetes mellitus or renal problems. No other co-worker living in the immediate environment suffered from a similar illness.

On examination, he looked ill and was drowsy and febrile, with a temperature of 37.8^{0}C, pulse

Address for correspondence and reprint requests: R M Yousuf, MD, Kulliyyah of Medicine, Department of Internal Medicine, International Islamic University Malaysia. P.O. Box 141, 27510 Kuantan, Pahang, Malaysia. Phone (O): 09-5132797; E mail: drmyrathor@yahoo.com; Fax: (609) 5133615.

rate of 112/minute, blood pressure of 110/70 mm Hg and respiratory rate of 26/minute. He was dehydrated with mild pallor and jaundice, but no lymphadenopathy was evident. His abdomen was distended with diffuse tenderness but no rigidity. Bowel sounds were sluggish. Chest examination revealed decreased air entry over the base of the right lung with bi-basal crepitations. Both heart sounds were heard with no added sounds. There was no neck stiffness and the musculo-skeletal examination was normal. A provisional diagnosis of liver abscesses was made and he was treated empirically with intravenous ceftazidime, metronidazole and intravenous fluids.

Initial investigations revealed a haemoglobin level of 9.2 g/dl, a white blood cell count of $12.3x10^9$ /L with neutrophils 86%. His platelet count was normal. Blood urea was raised (11.1 mmol/L), but serum creatinine was normal. Blood film for malarial parasites was negative. Liver function tests were remarkable with a total bilirubin of 24.3 μmol/L (direct 12.7 μmol/L, indirect 11.6 μmol/L), albumin 15.5 g/L, globulin 45.3g/L, alkaline phosphatase 516 U/L, alanine amino transferase (ALT) 492 IU/L, aspartate aminotransferase (AST) 284 IU/L, prothrombin time 17.5 with INR 1.43, activated partial thromboplastin time 34.7 sec, glucose 6.3 mmol/L. Renal function was normal after hydration. Serology for hepatitis B and C was negative. Chest X-ray showed raised right hemi-diaphragm with basal consolidation. The blood culture grew *E. tarda* which was sensitive to Ampicillin, gentamicin, Cefroxime, Cefperozone, Ceftriaxone and Ciprofloxacin. As a consequence of this finding, the antibiotic was changed to Ampicillin 2 gm 6 hourly.

By the third day following admission, the patient had improved clinically and was alert. However, his abdomen remained distended and was tender with sluggish bowel sounds. Ultrasonography (Fig.1) revealed multiple well-defined hypoechoic lesions in the liver.

In view of the ultrasonography finding, a CT scan of the abdomen was performed. This revealed multiple, well-circumscribed, hypodense, cystic-like lesions disseminated in both hepatic lobes (see Figs. 2a and b).

Ultrasound guided drainage of liver abscesses was performed and 25 ml of thick pus was drained, and sent for culture and sensitivity. A drain was inserted and connected to a bag. Stool examination revealed hook worm infection; no *E. tarda* was reported. Pus culture from the liver abscesses did not grow any bacteria.

The patient's abdomen distended further with very sluggish bowel sounds. A repeat CT scan of the abdomen was done (Figs. 3a and b). This showed an increase in the free fluid in the abdomen.

An exploratory laparotomy was performed which revealed substantial amounts of slough and pus in the peritoneal cavity with sections of bowel adherent to one another. Multiple loculated abscesses were found in the liver. There was a collection of pus in the pelvic cavity.

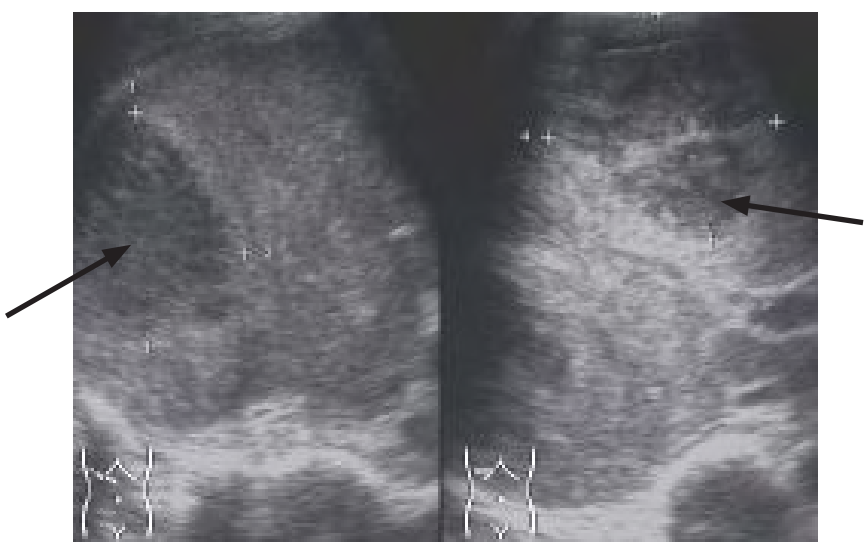

FIG. 1: Ultrasound of liver showing the presence of multiple, well-defined, hypoechoic lesions (arrows) with cavity debris in the right hepatic lobe

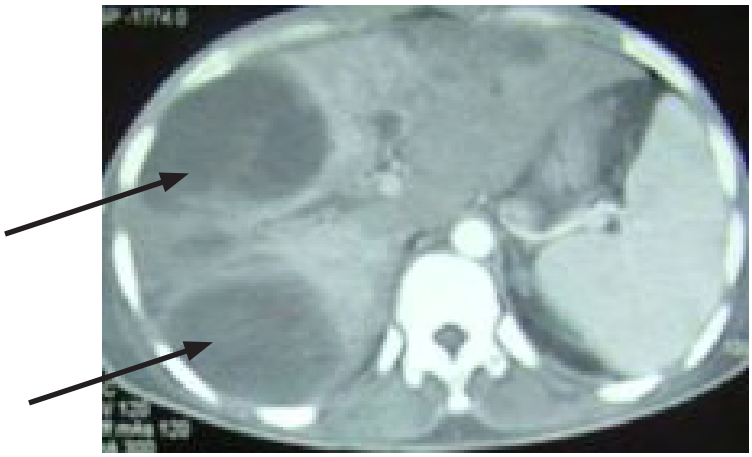

Figure 2(a)

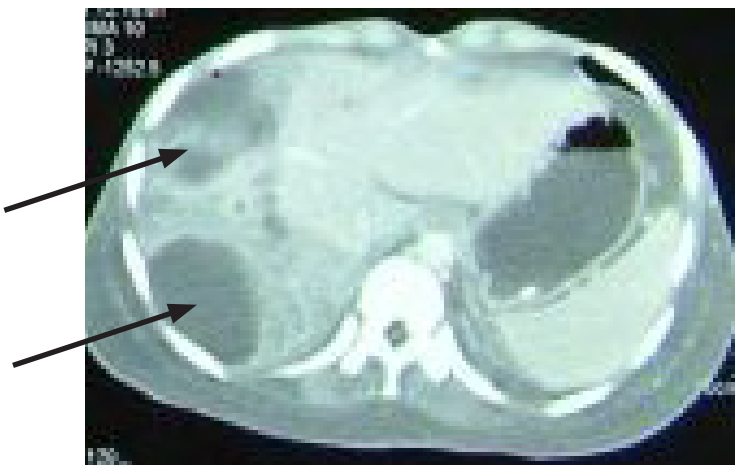

Figure 2(b)

FIG. 2: Post-contrast CT scan of the abdomen showing the liver during (a) the late arterial phase and (b) the portal venous phase showing the presence of multiple, well-circumscribed, hypo dense, cystic-like lesions (arrows) disseminated throughout both hepatic lobes, but predominantly in the right lobe. The lesions demonstrate peripheral enhancement. Both portal veins and inferior vena cava are patent. Minimal ascites is evident.

Drainage and peritoneal toilet was performed. Post-operatively, the patient went into shock and was treated in the intensive care unit. He was ventilated and the antibiotics were changed to intravenous amoxicillin-clavulanic acid and intravenous metronidazole. In spite of this, the patient deteriorated subsequently and died of septic shock two weeks later.

DISCUSSION

The most common manifestation of *E. tarda* infection is a gastrointestinal disease causing watery diarrhoea, but cases of invasive enterocolitis[4] have been reported suggesting that this pathogen can invade cells to spread systemically and cause tissue damage *in vivo*. Risk factors for *E. tarda* infections include exposure to aquatic environments or exposure to exotic animals (e.g., reptiles or amphibians), pre-existing liver disease, and dietary habits (e.g. ingestion of raw fish). A number of serious, extra-intestinal infections have been reported such as septicaemia with a mortality rate close to 50%,[5] meningitis, peritonitis, septic arthritis,[6] myo-necrosis,[7] tubo-ovarian abscess,[8] liver abscesses and wound infections.[9] Humans seem to be infected or colonised with *Edwardsiella* through either ingestion or inoculation of a

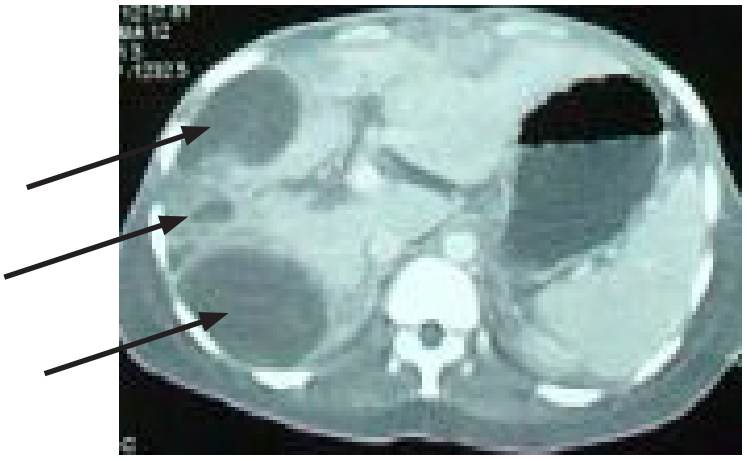

Figure 3(a)

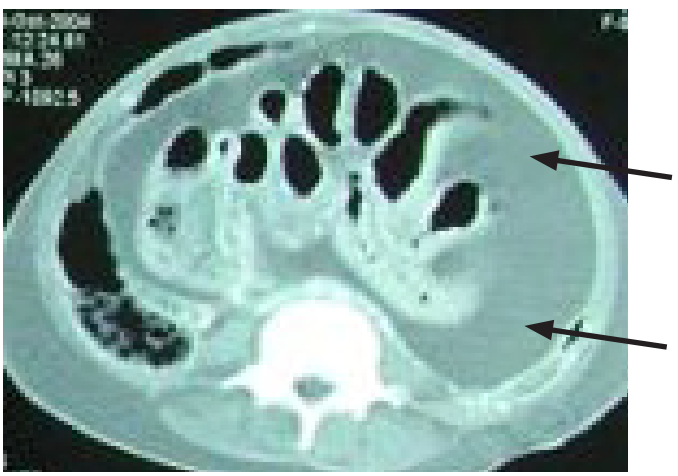

Figure 3(b)

FIG. 3: Post-contrast CT liver during portal venous phase. Figure 3a: the previously seen multiple, hypo dense, cystic-like lesions (arrows) have remained unchanged in size and appearance. Both portal veins and inferior vena cava are still patent. Figure 3b: there is now an increased amount of free fluid (arrows) in the abdomen and pelvis.

wound. Although the gut is probably the portal of entry in most cases of extra-intestinal infections, *E. tarda* has been isolated only rarely from the stools of such patients.

Our patient presented with an episode of gastroenteritis which was the most likely cause of his sepsis and multiple liver abscesses. He later developed bacterial peritonitis most likely due to liver abscesses that might have spontaneously ruptured into the peritoneal cavity before surgery. Stool culture failed to isolate *E. tarda* as he had received antibiotics for over a week.

Open surgical drainage along with antimicrobial chemotherapy has long been regarded as standard treatment for pyogenic liver abscesses. However, in many centres it is being replaced by percutaneous drainage under the guidance of computed tomography or ultrasound. Percutaneous drainage has been successful in patients with a single abscess, but the outcome has been less favourable in those with multiple abscesses.[10] Open surgical drainage is reserved for patients in whom treatment fails or who have complications.[11]

We initially performed percutaneous drainage in our patient. Pus was sterile on culture, possibly because he had been on antibiotics for more than one week. He had to undergo exploratory laparotomy due to development of peritonitis and further deterioration. Several researchers have obtained satisfactory results in selected patients with pyogenic liver abscess who received only medical therapy.[12, 13]

On review of the literature, we could trace only 4 reported cases of liver abscess due to *E. tarda*. Wilson and colleagues[5] reviewed 16 cases of serious infections due to *Edwardsiella tarda*, two of which had liver abscess. A 71-year-old Panamanian woman with liver abscesses had *E. tarda* isolated from a specimen of blood as well as from pus obtained by needle aspiration of the liver abscess. She died of septicaemia despite receiving prolonged antibiotic therapy. A 14-year-old female Nicaraguan immigrant to the United States presented with septicaemia due to *E. tarda*. Exploratory laparotomy revealed a liver abscess, which was drained. The pus revealed *E. tarda* on culture. The patient recovered after antibiotic therapy. From India, Koshi and Lalitha reported a case of liver abscess due to *E. tarda* in a patient who had hepatoma.[1] This patient died despite receiving antibiotics and undergoing surgical drainage. Zighelboim and associates from Baylor College of Medicine, Houston, Texas reported a case of multiple liver abscesses due to *E. tarda*, which was successfully managed with antibiotic therapy alone.[14] Of the total five cases of liver abscesses due to *E. tarda* including our case, the overall mortality was 60 % and most of the patients were treated by both drainage and antibiotics.

E. tarda is susceptible *in vitro* to a wide range of antibacterial agents.[15] Most strains of *E. tarda* are sensitive to Ampicillin, most β-lactam antibiotics, quinolones, chloramphenicol, tetracycline, and aminoglycosides. Our isolates showed the same pattern. Our patient received a variety of antibiotics and death was related to consequences of sepsis. Although extra-intestinal human infection with *E. tarda* has been reported infrequently, the recent identification of the first such case at our hospital suggests the need to consider such unusual pathogens in patients who present with febrile diarrhoea and consequent bacteraemia. Early empiric therapy for infections may prevent the isolation and recognition of *E. tarda* because of its susceptibility to numerous antibiotics. Therefore blood cultures should be taken before giving antibiotics.

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