Fatal *Campylobacter jejuni* infection in a patient splenectomised for thalassaemia

N Jackson, M Zaki, A R Rahman, M Nazim, M N Win, S Osman

**Abstract**

A 35 year old man with a fatal *Campylobacter jejuni* infection is described. He had HbE/β⁺ thalassaemia and had undergone splenectomy nine months previously for hypersplenism; he also had chronic hepatitis C infection. He presented with high grade fever but no gastrointestinal symptoms and rapidly progressed to septicemic shock and hepatic encephalopathy despite treatment with penicillin, gentamicin, and, later, chloramphenicol and ceftazidime. Only one case of *Campylobacter jejuni* septicaemia occurring post-splenectomy has been reported previously, also in an iron overloaded thalassaemia patient. Unusual Gram negative bacilli must be covered by the chosen antibiotic regimen when splenectomised thalassaemic patients present with high grade fever.

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Keywords: campylobacter; splenectomy; thalassaemia

The most common and serious infections in splenectomised patients are those due to *Streptococcus pneumoniae, Haemophilus influenzae* type b and *Neisseria meningitidis.* Current recommendations for prophylaxis in splenectomised individuals include routine immunisation against the first two of these organisms, and against the third when the patient is travelling to an endemic area. However, Gram negative bacilli and protozoal infections have also to be considered, especially in patients with other immune defects. We describe a very unusual case of overwhelming septicaemia due to *Campylobacter jejuni* in a splenectomised individual with iron overload and chronic liver disease.

**Case report**

A 35 year old Malay man was admitted with a two day history of generalised body aches and low grade fever, but no gastrointestinal upset. He had HbE/β⁺ thalassaemia for which he had been irregularly transfused, but he had not received iron chelation as this is not routinely available in Malaysia. He was known to be iron overloaded with impaired cardiac function, and to have chronic hepatitis with positive serological markers for hepatitis C. A splenectomy had been done nine months previously for hypersplenism, after which he had remained pale and jaundiced, but he had not required transfusion. Pneumococcal vaccine had not been available but he was taking oral phenoxymethylpenicillin 250 mg twice daily, with good compliance.

On examination at the time of this admission he was deeply jaundiced, his temperature was 40.5°C but he was alert. Blood pressure was 120/70 mm Hg and central nervous system examination was normal. There was evidence of marked derangement of liver function, but renal function was initially normal. Chest x ray showed cardiomegaly and mild pulmonary congestion. HIV antibody test was negative. He was treated with intravenous benzylpenicillin 4 MU every four hours, and gentamicin 80 mg every eight hours (equivalent to 5.6 mg/kg/day).

By the second day, his blood pressure was 90/50 mm Hg and he was drowsy and irritable but with no focal neurological signs or meningism. Chloramphenicol 1 g every six hours was added against the possibility of haemophilus meningitis. Later the same day, computed tomography of the brain showed slight effacement of sulci but no mass lesion or meningeal enhancement; an electroencephalogram showed encephalopathic changes; and cerebrospinal fluid examination was normal. No malarial parasites were seen on a blood film. A diagnosis of hepatic encephalopathy was made, and oral neomycin and lactulose were added.

On the third day, he became photophobic with neck stiffness, a positive Kernig’s sign, bilateral sustained clonus, and an extensor plantar reflex on the left side. Serum gentamicin concentration was subtherapeutic (pre-dose 0.9 mg/l, one hour post-dose 3.13 mg/l). However, in view of deteriorating renal function (blood urea had risen from 4.4 mmol/l on admission to 15.7 mmol/l), the dose was not increased; ceftazidime 2 g every 12 hours was added. On the fourth day, right-sided tonic-clonic fits occurred, he became more hypertensive and died. A postmortem examination was not performed.

Two blood cultures, taken on the day of admission, yielded motile Gram negative “S” shaped bacteria after five days of incubation. They were identified as *C. jejuni* based on the biochemical tests recommended by Karmali and Skirrow. The isolate was sensitive to gentamicin, chloramphenicol, and erythromycin, but resistant to penicillin and ampicillin.

**Discussion**

Although *C. jejuni* infection usually presents with acute enteritis, septicaemic cases without enteritis have been reported. The present patient died from hepatic encephalopathy caused by severe *C. jejuni* septicemia, meningitis being excluded by
Standardisation of polymerase chain reaction for the detection of *Salmonella typhi* in typhoid fever

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Abstract

To improve the diagnosis of *Salmonella typhi* infection, a polymerase chain reaction (PCR) assay was developed for the amplification of the dh flagellin gene of *S. typhi*. Primers were designed from dh flagellin gene sequence which will give an amplification product of 486 base pairs. In tests to study the specificity of the assay, no amplification was seen in non-salmonella strains or salmonella strains with flagellar gene other than “dh”. Sensitivity tests determined that 28 pg of *S. typhi* target DNA or 3 x 10^6 target bacteria could be detected by the PCR assay. Subsequently, the PCR technique was used for detection of *S. typhi* in blood or clot cultures from 84 patients clinically suspected of having typhoid fever, and from 20 healthy control subjects. Twenty five of 84 samples from clinically suspected cases were positive by PCR; four of which were culture positive. No amplification was seen in samples from patients who were culture negative for organisms other than *S. typhi* or from controls. The time taken for each sample for PCR analysis was less than 48 hours compared with five to three days for blood or clot culture. PCR appeared to be a promising diagnostic test for typhoid fever. (J Clin Pathol 1997;50:437–439)

Keywords: *Salmonella typhi*, polymerase chain reaction; typhoid fever

Typhoid fever, a septicaemic disease caused by *Salmonella typhi*, is a serious health problem in developing countries. Diagnosis of typhoid fever currently relies on blood culture and Widal’s test. Blood cultures are negative in 30–65% of cases with typhoid fever because of...
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