AEROMONAS ENDOCARDITIS IN A PATIENT WITH CHRONIC HEPATITIS-B INFECTION

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ABSTRACT

Aeromonas, a genus of gram-negative bacteria normally found in water and soil, is well established as a pathogen in the animal kingdom. Often considered as a pathogen of low virulence, its role in human infections has recently been recognised. Aeromonas infections in humans range from cellulitis to septicaemia. Endocarditis is rare (7). We describe here a patient with a chronic liver disease with aeromonas bacteremia and endocarditis.

Keywords: Aeromonas Endocarditis, Aeromonas bacteremia

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INTRODUCTION

Aeromonas species are seldom recognised as the cause of human infections. Previously, Aeromonas, a member of the family Vibrionaceae, were considered opportunistic pathogens of low virulence causing serious infections only in immunocompromised hosts. Recently they have been recognised as primary pathogens as well (4, 12). Aeromonas species have been associated with a wide range of human infections, the severity of which depends on the immune status of the host. Infections range from wound infection with or without fulminant myositis(5), osteomyelitis, meningitis, gastroenteritis (1, 6, 9) and septicaemia in immunocompromised hosts (3, 8, 10, 11, 13). A case of endocarditis in a cirrhotic patient has also been reported (12).

Aeromonas species are gram-negative facultative anaerobic rods that ferment carbohydrates with the production of acid and gas and give a positive oxidase reaction. Four species are commonly mentioned in the literature: *Aeromonas hydrophila, A. sobria, A. caviae and A. salmonicida.* The latter is associated with fish furunculosis and is an important pathogen in the fishing industry.

The majority of human Aeromonas isolates have been *Aeromonas hydrophila.*

The primary habitat of Aeromonas strains is in nonfaecal sewage with high organic content. They may be isolated from lakes,water taps and hospital water supplies and for this reason human disease is frequently related to water exposure (3, 5). Although Aeromonas is not considered part of the normal faecal flora, positive stool cultures have been obtained from asymptomatic individuals (8). This probably represents transient colonisation from contaminated water supplies. In terms of clinical significance, the most important form of Aeromonas infection is sepsis. We describe here a patient whom we believe had Aeromonas endocarditis.

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CASE REPORT

Mr TKC, a 57-year old Chinese retired mechanic was admitted to Tan Tock Seng hospital on 17 June 1987 for the complaints of high fever with chills and rigors and joint pains for two days duration.

He had been passing frequent loose stools for about two weeks prior to this. There was no history of abdominal pain or bleeding from the gastrointestinal tract. Two days before admission, he developed a high swinging fever with chills and rigors and noticed pain and swelling over the left ankle and right shoulder. He also had a mild cough productive of whitish phlegm. There was no history of other joint pains, change in urinary habits, vomiting or rash. He did not notice any breathlessness or orthopnoea. The patient had no history of similar illness before.

In January 1986, the patient complained of lethargy and generalised malaise. He consulted a general practitioner who noticed that he was jaundiced. Subsequent investigations by a gastroenterologist in another hospital showed that he had chronic Hepatitis B infection and Thalassaemia B minor. The cardiovascular examination then did not reveal any murmur. The HBsAg was positive. Anti-HBc IgM, HBeAg and anti-HAV IgM were negative. Total protein was 7.7 gm/dl, albumin 3.9 gm/dl, bilirubin 2.1 mg/dl, alkaline phosphatase 138u/l, SGPT 98 u/l and SGOT 113 u/l. Alpha-foeto protein was negative. CT scan of the abdomen showed a normal sized liver with an irregular surface suggestive of cirrhoses. The spleen was enlarged. He refused consent for a liver biopsy. He had no history of liver disease prior to this. He used to be a social drinker but he stopped the habit about two years ago. He was a chronic smoker.

There was no history of hypertension, diabetes mellitus or ischaemic heart disease. There was no family history of any blood or liver disorder.

The temperature was 40 degrees Celsius, the pulse was 120pm and the respiration 25pm. The blood pressure was 130/80 mmHg. On examination, the general condition was fair and the patient was alert and able to converse. He was mildly jaundiced and pale. Palmar erythema and spider naevi on the chest were present. There was no asterixis. The jugular venous pressure was raised and bilateral ankle oedema was present. No lymphadenopathy was detected. Fine inspiratory crackles were heard at both lung bases. The heart rhythm was regular and there was a grade 2/6 systolic murmur heard along the left sternal border. S_3 was not heard. Abdominal examination revealed a soft, non-tender but slightly distended abdomen. There was shifting dullness and the spleen was palpable 4 cm below the costal

margin. The liver was not palpable nor were the kidneys ballotable. Both the left ankle and the right sternoclavicular joints were swollen, inflamed and tender. Joint movements were limited because of pain. The other joints were normal. There was no clubbing, splinter or conjunctival haemorrhages. Fundoscopy was normal.

Investigations revealed a haemoglobin of 8.3 gm/dl. The white cell count was 16.3×10^9/L with 90% polymorphs, 8% lymphocytes, 1% monocytes and 1% eosinophils. Platelet count was 120×10^9/L and the reticulocyte count was 3%. The peripheral blood film showed a moderate hypochromia and microcytosis with some target cells present. No early whites were seen. Haemoglobin electrophoresis showed a HbA_2 of 5.1% (2.3 – 3.3) and the alkaline resistant Hb was 1.0% (0.1 – 1.7). The erythrocyte sedimentation rate was 70mm in the 1st hour. Prothrombin time was 14 seconds with a control of 13 seconds and partial thromboplastin time was 41 seconds with a control of 38 seconds. The urea was 29 mg/dl, creatinine 1.3 mg/dl, sodium 127 mmol/dl, potassium 3.9 mmol/dl, chloride 100 mmol/dl and glucose 125 mg/dl. Uric acid was 6.4 mg/dl (3.9 – 8.3). Urinalysis was normal. A portable CXR and ECG were normal. Blood culture grew Aeromonas species sensitive to gentamicin, tetracycline, trimethoprimsulphamethoxazole and ceftriaxone, and resistant to ampicillin and cephalothin. Serum immunoglobulins showed IgG was 2452 mg/dl (760 – 1600), IgA 472 mg/dl (70-380) and IgM 119mg/dl (30-160).

TREATMENT AND PROGRESS

A diagnosis of infective endocarditis was made and the patient started on intravenous ceftriaxone 1 gm b.i.d. Gentamicin 60 mg t.i.d. was added on day 4 of admission as the fever had still not subsided. These 2 antibiotics were continued after the blood culture and senstivity results were known. The fever settled after about one week of antibiotics and gentamicin was taken off at the end of two weeks. Ceftriaxone was reduced to 1 gm o.m. on day 14 and was continued till day 28 of admission. Except for a short period of low grade fever which was attributed to phlebitis due to the intravenous line, the patient improved progressively. The arthritis subsided and the erythrocyte sedimentation rate and white cell count dropped to normal. 2D echocardiogram of the heart was done on day 2 (Technicare EDP 1200) and day 33 (ATL MK 500 Echo System) of admission. The valves appeared normal and no vegetations were detected. The patient was discharged on 21 July 1987 after a stay of 34 days in hospital.

FOLLOW UP

The patient was subsequently seen at the outpatient clinic. Apart from mild pedal oedema and anaemia he was well and had no complaints. He was last seen on 23/09/87, two months after discharge. He was readmitted again on 30/09/87 because of progressive abdominal distension and abdominal pain. He was anorexic, nauseated and developed a fever on the same day. Temperature was 37 degrees celsius, pulse was 108 and blood pressure was 80/60. On examination, the patient appeared ill but was conscious and able to answer questions. He was jaundiced and pale but there was no asterixis. Fine crackles were heard at both lung bases. Heart sounds were regular and there was a grade 2/6 systolic murmur over the left sternal edge. Abdominal examination revealed a distended abdomen and fluid thrill was present. The liver and spleen were not palpable and bowel sounds were absent. Electrocardiograph did not show any myocardial infarction and a peritoneal tap revealed yellowish aspirate with an amylase content of 92 u/l. The white cell count was 3.4×10^9/L and the haemoglobin was 6.6 gm/dl. Prothrombin time was 16 seconds with a control of 12 seconds and activated partial thromboplastin time was 37 seconds with a control of 26 seconds. He was treated for septicaemic shock and was resuscitated with intravenous colloid infusion and started on intravenous ceftriaxone, gentamicin and dopamine. Two specimens of blood did not grow any organisms and the peritoneal fluid grew citrobacter species (possibly a contaminant). The hypotension persisted and the patient collapsed and died 24 hours after admission.

DISCUSSION

Human infections with Aeromonas may ensue through trauma (3), after ingestion or exposure to contaminated water (3, 10, 12). Sepsis usually originates from an endogenous source in an immunocompromised host (8, 13). Most authors have reported that the gastrointestinal tract is the usual source of entry into the bloodstream. Liver cirrhoses and malignancies have been most frequently associated with Aeromonas bacteria. Approximately 71% of the published cases have occured in such patients (8, 12). Haematological malignancies have been present in 40% of patients with Aeromonas bacteremia, cirrhoses in 17% and solid tumours in 14%. Although not common Aeromonas bacteria has been reported in noncompromised hosts (4, 12). The cumulative sepsis-related mortality for patients with Aeromonas bacteremia is approximately 57% (12). The symptoms of Aeromonas septicaemia resembled those of other gram-negative organisms. Most blood isolates have been *Aeromonas hydrophila*. Unfortunately, the particular species was not identified in our patient.

It is interesting to note that in our patient, the initial illness was a form of gastroenteritis. He had complained of frequent loose stools before presenting with symptoms of bacteremia. Therefore, the portal of entry into the blood was most likely through the gastrointestinal tract. He had chronic Hepatitis B infection with ascites and splenomegaly and it has been proposed that in cirrhotics, bacteremia from the gut is favoured by defective hepatic filtration. Serum immunoglobulins were not low in our patient and this seems to be in agreement with most patients in which immunological studies have failed to reveal defects in immunoglobulins or other specific host defences. Our patient had endocarditis, the diagnosis of which was based on the fever, cardiac murmur, embolic phenomena (sternoclavicular and ankle arthritis) and the positive blood culture. The Aeromonas species grown from blood were sensitive to gentamicin, tetracycline, trimethoprimsulphamethoxazole and ceftriaxone and were resistant to ampicillin and cephalothin. This was in agreement with most other reports (10, 12, 13). Our patient responded favourably to the combination of gentamicin and ceftriaxone. Apart from a brief period of low grade fever midway during treatment which was attributed to phlebitis, there were no complications observed throughout the treatment. He was discharged on day 34 of admission and was well for the next 2 months. The final event was another septicaemic bout with hypotension. Blood culture was negative on that occasion. Despite the appropriate measures taken, he died on 1 October 1987,10 weeks after discharge from hospital. Consent for autopsy was not obtained.

SUMMARY

Our patient with chronic liver disease and Thalassaemia B minor, represented an example of a susceptible host to Aeromonas infections. Bacteremia in such patients

have been well described but endocarditis seems rare (12, 13). In our patient, the portal of entry into the blood was most probably through the gastrointestinal tract. There was a good response to the antibiotics given. He died 10 weeks later of another bout of sepsis which was unrelated to the first admission.

This report serves to demonstrate that Aeromonas species are capable of causing serious human infections. Although it was described as early as 1954 by Hill (14), its importance is not usually recognised. With the advent of Acquired Immune Deficiency Syndrome and the relative ease of contact with contaminated water supplies, the role of Aeromonas in human infections may attain more importance.

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