Melioidosis and Peritoneal Dialysis Related Peritonitis

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SUMMARY

We report a case of melioidosis presenting as peritonitis in a patient on continuous ambulatory peritoneal dialysis (CAPD). A 47-year-old man, a lorry driver, with end-stage renal disease due to diabetes mellitus on CAPD presented in PD-related peritonitis. He was started on intraperitoneal cloxacillin and ceftazidime, and changed to intraperitoneal vancomycin and meropenam after day 5 due to nonresponse. *Burkholderia pseudomallei* was identified from the dialysate culture. He was treated with intraperitoneal meropenam for two weeks, and IV ceftazidime for 4 weeks. He responded, and the Tenckhoff catheter was not removed. He was discharged well and continued on oral sulfamethoxazole/trimethoprim for six months. This patient had done his PD exchanges in a lorry.

KEY WORDS: Peritoneal dialysis, melioidosis, peritonitis, CAPD

INTRODUCTION

Melioidosis is caused by *Burkholderia pseudomallei*, a motile aerobic, non-spore forming gram negative bacilli. It is an environmental saprophyte that causes melioidosis. Melioidosis is considered endemic in southeast-Asia¹. Three modes of acquisition are recognized: inhalation, ingestion and inoculation. It has also been recognized that melioidosis is an opportunistic infection, occurring in patients with underlying disease(s), such as diabetes mellitus^{1,2}. Melioidosis has various clinical presentations, from benign soft tissue infection to fulminant septicaemia. The lungs seem to be the commonest affected organ. We report a case of melioidosis presenting as peritonitis in a patient on continuous ambulatory peritoneal dialysis (CAPD).

CASE REPORT

JT was a 47-year-old man who developed end-stage renal disease due to diabetes mellitus. He also suffered from hypertension. He worked as a lorry driver. He was started on CAPD in June 2011. The CAPD was interrupted in August 2011 due to Tenckhoff catheter migration. He was put on haemodialysis temporarily, and partial omentectomy and revision of Tenckhoff catheter was performed. He was restarted back on CAPD in September 2011.

Despite achieving good Kt/V (>1.7), with reasonable daily ultrafiltration of about 800 - 1000 ml/day, and a urine output of >200 ml/day, he had poor fluid balance status, blood

pressure control, low serum albumin and low haemoglobin (due to poor compliance to using erythropoietin). His blood sugar was also poorly controlled.

He presented in December 2011 with abdominal pain, nausea and vomiting, fever, and cloudy peritoneal dialysate. The exit site did not reveal any sign of infection. He was treated as PD related peritonitis, and was started on intraperitoneal cloxacillin and ceftazidime. There was initial response in terms of clinical symptoms and signs as well as the dialysate cell count, but later the dialysate remained cloudy. The antibiotics were then changed to intraperitoneal vancomycin and meropenam after day 5, and the fluid cell count remained above 100/uL. *Burkholderia pseudomallei* was then identified from the PD fluid culture.

He was then treated with intraperitoneal meropenam for two weeks, and IV ceftazidime for 4 weeks. His PD peritonitis responded, with the fluid became clear, and the white blood cell count in the PD fluid dropped to normal range. The Tenckhoff catheter was not removed. Subsequent PD fluid cultures were negative. There was no other sites of infection or abscess.

He developed bilateral vitreous haemorrhage due to proliferative diabetic retinopathy during the hospitalization. His wife then took the role to assist him to continue the CAPD.

He was discharged from the hospital after completion of the antibiotics, and was continued on oral Bactrim® (sulfamethoxazole/trimethoprim) (two tabs bd) for six months. He admitted later that he performed his PD exchanges in the lorry that he was driving for at work.

DISCUSSION

A few clinical risk factors were summarized by Cheng and Currie¹. In our patient, the fact that he did the PD exchanges inside a lorry may have contributed to exposure of the organism into the peritoneal cavity, and being male, having diabetes mellitus and chronic kidney disease were his other obvious risk factors.

Pneumonia is the most common presentation of melioidosis, followed by genitourinary, skin or soft tissue infection, splenic or liver abscesses, parotid, and prostate abscess¹⁻³. From the literature search, only one case of melioidosis presenting as PD-related peritonitis has been described⁴.

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Treatment consists of an intensive phase, followed by a prolonged course of eradication phase ¹. Intravenous ceftazidime has been the antibiotic of choice in our center for melioidosis. We changed the IP ceftazidime to IP meropenam before we knew the diagnosis, and the patient seemed to be responding after the switch. Therefore we persisted with two weeks of IP meropenam, together with four weeks intravenous ceftazidime (as the culture demonstrated the sensitivity of the organism to ceftazidime).

CONCLUSION

This case highlighted the importance of microbiologic identification, as melioidosis is rarely identified in PD-related peritonitis. This is also important as melioidosis requires a subsequent three to six month regime of oral antibiotic(s) to reduce the risk of relapse/recurrence of the infection.

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