CASE REPORT

Community Acquired Acinetobacter Infection

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SUMMARY

A young boy presented with fever for 2 weeks of which no source could be identified on history. Examination showed hepatomegaly. Investigations showed hepatomegaly and mild spleenomegaly and acinetobacter growth was seen on blood cultures. On initial treatment, patient started having multiple episodes of loose stools due to drug induced colitis, findings of which were also seen on CT Abdomen. This community acquired acinetobacter infection responded to meropenem and repeat blood cultures also showed clearance of acinetobacter from blood stream along with resolution of fever.
Keywords: Acinetobacter infection, hepatomegaly, spleenomegaly

INTRODUCTION

Acinetobacter is a gram negative coccobacillus that has emerged from an organism of questionable pathogenicity to an important infectious agent to the hospitals world-wide[1]. The organism can accumulate multiple mechanisms of resistance, which can lead to appearence of strains that are resistant to most of the available antibiotics[2]. Acinetobacter can colonize GI tract, skin wounds and respiratory tract[3]. In humans, Acinetobacter can colonize skin, wounds, and the GI and respiratory tracts[4]. The most common clinical manifestations are VAP(ventilator associated pneumonia) and bloodstream infections[5]. It may be difficult to distinguish between true infection and colonization[6]. In addition, colonization is a risk factor for subsequent infection.

CASE REPORT

A 19 years old male from northern Punjab was admitted in the ward due to persistent fever from past 2 weeks. Initially fever was low-grade and intermittent but after few days fever became high-grade and recorded up to 103˚F. It was not associated with rigors and chills. Fever was relieved by antipyretics and cold sponging. There was no accompanying, anorexia, night sweats, weight loss or history of Tuberculosis contact.

Two-days before admission fever became high-grade and continuous. It was not accompanied by sore throat, cough, shortness of breath, dysuria, burning micturation, abdominal pain, constipation, diarrhea, yellowish discoloration of eyes, blurring of vision, neck stiffness, focal weakness, fits, ear discharge, otalgia, joints pains, oral,ulcers, skin rash. Initial workup of fever went uneventful. Examination was uneventful except for multiple readings of high-grade continuous fever and hepatomegaly. Initially, results of laboratory tests performed showed: W.B.C= $5.6\times10^9/l$(N.R 4-11), neutrophils 70%, lymphocytes 25%, monocytes 3% , eosinophil 2%, E.S.R=25mm/1sthr. (male: 17-50=<10mm), CRP=24(<6). Anti HIV, urine R/E, MP slide, NS1 antigen, Typhidot IgM were negative.

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Ultra sound of abdomen showed liver of size 16.9cm with smooth surface and starry sky appearance. No focal parenchymal defect and spleen of size 14.2cm with no focal lesion. CT Scan Abdomen was done after few days which showed hepatospleenomegaly diffuse circumferential wall thickening involving distal ileum, caecum, ascending, transverse and descending colon. No proximal small bowel dilatation seen. Regional lymphadenopathy seen largest node measuring 1.5cm. Meanwhile repeated CDC shows: W.B.C = 3.6(4-11), platelet=$158\times10^9/l$ (150-400), neutrophils 70% ,lymphocytes=28% monocytes=1% , eosinophil=1%.

Patient was initially given Artemether+lumefantrine along with ceftriaxone, piperacillin and tazobactam. Patient remained febrile for next 48 hours and started passing multiple episodes of loose stools, which were watery, non-bloody not aggrevated or releived by any specific thing. After which we discontinued above mentioned medicines and started Tab. Metronidazole along with azithromycin and continued these medication for next 48 hours which resulted in improvement of diarrhea. Then Blood culture report was received which was sent on admission, it showed growth of Acinobacter which was sensitive to meropenem, piperacillin+ azobactem, levofloxacin and gentamicin. We started meropenem and continued previous medications, which was resulted in decrease in fever in next 48 hours. Patient became afebrile after 48 hours of starting meropenem. After 10 days of meropenem repeated blood culture showed no growth and patient was discharged and called on follow-up.

DISCUSSION

To our knowledge this is 1st case report of community acquired acinetobacter infection in Lahore. Community acquired acinetobacter infection has been reported in Australia and Asia[3,4,6]. In Australia community acquired pneumonia occurs more frequently during wet season. Acinetobacter is usually considered opportunistic nosocomial pathogen. The epidemiological profile tells that it has low virulence and disease is largely dependent on significant impairment of immunological system of host. The evidence is now mounting that acinetobacter is not only a nosocomial pathogen and is also capable of

causing significant clinical disease even in the absence of well known nosocomial resist factor. In a recent review, we have seen almost 80 cases of community acquired acinetobacter infection. It also suggests that co-morbidities such as renal dysfunction, COPD and Diabetes Mellitus are predisposing factors. Interestingly, smoking and alcoholism are also associated with increased chances of having acinetobacter infection. However, this reported case of community acquired acinetobacter infection do not suggest significant co-morbidities. High-grade fever, tachycardia, leucopenia, deranged transaminases, radiological evidence of colitis and hemoglobinuria were suggestive of acinetobacter induced sepsis. Acinetobacter is an important nosocomial pathogen and its forcible that this organism is going to evolve into community- acquired pathogen too.

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