CASE REPORT

Case series: Fulminant community-acquired *Acinetobacter* pneumonia

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SUMMARY

***Acinetobacter* infection, especially the drug-resistant strain, is a common cause of nosocomial infection. However, community-acquired *Acinetobacter* infection is uncommon. We reported three cases of community-acquired *Acinetobacter* pneumonia. All three cases had histories of regular home-brewed alcohol consumption presented with severe acute respiratory symptoms requiring ventilatory support and had low total white cell count. They succumbed to the illness within 2 to 10 days of admission. They had positive blood or endotracheal aspirate cultures of sensitive-strain *Acinetobacter sp.* which was only sensitive to high dose sulbactam. Early recognition and correct antibiotic can help reduce mortality.**

INTRODUCTION

Acinetobacter is found naturally in soil and water, especially in the temperate climate countries, and it's a rare coloniser of the human skin and throat.[1] This pathogen, especially the drug-resistant strain, commonly causes nosocomial infection, especially in catheter-related and ventilator-related infection and causes significantly increased mortality and morbidity of patients.[2]

Community-acquired *Acinetobacter* pneumonia (CaAP) is uncommon. It now has increasing importance as it is associated with high mortality of 40-60%, especially in patients with co-morbidities. The identifiable risk factor is excessive alcohol consumption.[3] We report three cases of community-acquired *Acinetobacter* pneumonia where the patients died.

CASE DESCRIPTION

Case 1

The patient a 65-year-old man, who was a chronic smoker and consumes regular home-brewed alcohol *(tuak)*. He had underlying Chronic Obstructive Pulmonary Disease (COPD). He presented with a two-week history of productive cough with fever, right sided pleuritic chest pain and worsening shortness of breath. He was in septic shock, tachypnoeic and febrile on examination. Chest X-ray (CXR) showed evidence of right lobar pneumonia. He had low total white cell count of 1.16K, renal impairment and severe metabolic and respiratory acidosis. He was empirically treated with intravenous (IV) ceftriaxone 2gram (g) once a day (OD) and azithromycin 500mg OD. He deteriorated rapidly and despite ventilatory support, he succumbed to his illness on day-2 of admission. His blood culture grew sensitive-strain of *Acinetobacter spp.*

Case 2

The second patient a 49-year-old previously healthy man was a chronic smoker and regular home-brewed alcohol consumer. He presented with a two-day history of fever, cough, abdominal pain and vomiting. He was tachypnoeic and in septic shock. His total white cell was 1.4K, and he also had severe metabolic and respiratory acidosis, renal impairment, coagulopathy with severe liver impairment. CXR showed left lung lobar pneumonia. He deteriorated rapidly and needed ventilatory support. He was empirically treated with IV ceftriaxone 2g OD and azithromycin 500mg OD, which was rapidly upgraded to IV meropenem 1g 8hourly. His blood and endotracheal culture grew sensitive-strain *Acinetobacter baumanii*. Antibiotic was changed to IV ampicillin-sulbactam 3g 6 hourly, but he succumbed to the illness on day 10 of admission due to multi-organ failure.

Case 3

The third patient was a 54-year-old previously healthy man, who was a chronic smoker and regular home-brewed alcohol consumer. He presented with a three-day history of fever, cough and left pleuritic chest pain. He was tachypnoeic and in septic shock. He had low total white cell count of 2.0K, severe metabolic and respiratory acidosis and renal impairment. CXR showed left lobar pneumonia. He was empirically treated with IV ceftriaxone 2g OD. His condition deteriorated rapidly which required ventilatory support. He succumbed to his illness on day 2 of admission. His blood culture grew sensitive-strain *Acinetobacter spp.*

DISCUSSION

In this series, all three cases presented with acute onset of fever, cough and shortness of breath. Two patients had pleuritic chest pain. They were in septic shock, severe metabolic and respiratory acidosis and renal impairment. All had significant histories of regular home-brewed alcohol consumption. Despite prompt treatment all of them succumbed rapidly to the illness.

Acinetobacter spp. is present in community in soil and water causing opportunistic infection, for example in wound

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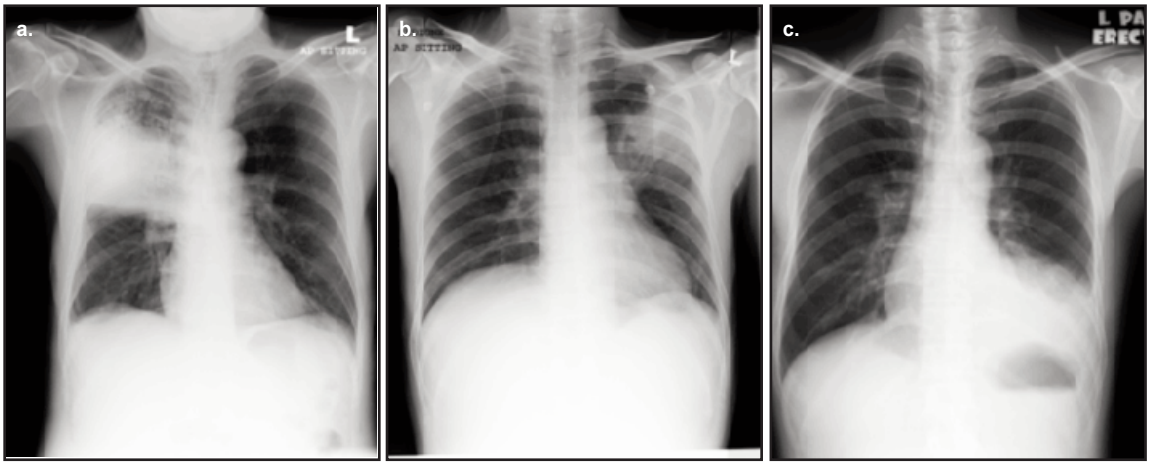
Table I: Blood culture reports

	Case 1 22 November 2014	Case 2 18 June 2015	Case 3 7 July 2015
Pathogen	*Acinetobacter spp.*	*Acinetobacter baumannii*	*Acinetobacter spp.*
Amikacin 30µg	Sensitive	Sensitive	Sensitive
Cefoperazone/Sulbactam 30µg	Sensitive	Sensitive	Sensitive
Gentamicin 10µg	Sensitive	Sensitive	Sensitive
Netilmicin 30µg	Sensitive	Sensitive	Sensitive
Ampicilin/Sulbactam 10/1	Sensitive	Sensitive	Sensitive
Piperacillin/Tazobactom	Sensitive	Sensitive	Sensitive



CXR of Case 1 CXR of Case 2 CXR of Case 3

Fig. 1: A,1 B, 1 C: Chest X-Ray of Case 1, 2 and 3.

infection.[1,3] CaAP is typically rapid in onset and progress. It is associated with respiratory failure, hemodynamic instability and septic shock.

Acinetobacter spp. is known to survive prolonged periods of dry and iron-deficient environment which makes it a successful pathogen.[3] Increasing numbers of case reports regarding fulminant community acquired *Acinetobacter* pneumonia are seen, especially in subtropical countries during warmer and humid season, for example, Singapore, Taiwan, Hong Kong and Northern Australia.[3]

The major risk factors that affects the outcomes of CaAP include underlying COPD, diabetes mellitus, renal impairment, heavy smoking and excessive alcohol consumption.[3] Local home-brewed alcoholic beverages carries a risk of contamination if the process of production was not monitored carefully. A recent report revealed alveolar macrophages are important in combating *Acinetobacter spp.* by phagocytosis via microtubules and microfilaments and produce high level of nitric oxide.4 Alcohol causes impaired immune system via a few mechanisms, including causing impaired phagocytosis by alveolar macrophages, reducing nitric oxide synthase activity and altering cytokines production.[4] Impairment in cytokines production lead to hazardous complications, such as septic shock and impaired adaptive immune cellular recruitment.[4]

The choice of antibiotic in the management of community acquired *Acinetobacter* pneumonia is crucial. International guideline suggested broad spectrum antibiotics as empirical antibiotic cover in severe pneumonia, which is beta-lactam (ampicilin/sulbactam or third generation cephalosporin) with macrolides.[2] Sulbactam is different from other beta-lactam inhibitors as it has significant intrinsic antimicrobial ability against *Acinetobacter*, its action is via binding to Penicilin-binding protein-2.[3] CaAP is caused by sulbactam-susceptible *Acinetobacter*, and IV sulbactam will be the antibiotic of choice for all community-acquired *Acinetobacter* infection.[3] The recommended dose of ampicillin/sulbactam dose is 8g/4g IV infusion over 4 hours every 8 hourly.[5] The delay in administering appropriate antibiotic is likely to be one of the major contributor for the mortality in this illness.

CONCLUSION

Acinetobacter spp. can cause life-threatening infection which rapidly progressing into community acquired pneumonia. Risk of infection is often associated with consumption of home-brewed alcohol beverages. Community-acquired lobar pneumonia with low total white cell count and regular alcohol consumption of home brewed alcohol should prompt the physicians to consider this diagnosis.

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