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Community Acquired Multi Drug Resistant Pneumonia Due To Acinetobacter Baumannii Infection – A Case Report.

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Abstract

Acinetobacter baumannii has been attracting increasing attention in India and worldwide, due to their increasing prevalence of multi drug resistance. Initially though restricted to intensive care units with the increasing prevalence has spread to other areas in hospital with reports from community settings also. The available literature about Acinetobacter suggests that the pathogen has the potential to cause community-acquired infections ranging from pneumonia, meningitis, soft tissue infections and also ocular infections.

Herein we describe the present a case who was admitted with community acquired pneumonia (CAP) secondary to multidrug resistance (MDR) Acinetobacter with sepsis. She improved on last-line antibiotics, and was discharged home.

Introduction

A.baumannii is a gram-negative short bacilli and is a normal commensal bacteria on human skin. Acinetobacter's are considered to be ubiquitous microorganisms, since they are found frequently in soil, water, and dry environments and have also been isolated from the hospital environment, foods, and animals. *Acinetobacter* species are probably the group of gram-negative bacteria that are natural residents of human skin. The recent studies report a colonization rate of 42.5% in healthy individuals and 75% in patients which is higher than the previously described colonization rate of 25% in healthy individuals (1,2). Acinetobacter infections are most commonly reported from ICU's and the most common clinical forms are as ventilator associated pneumonia and blood stream infections. Community acquired infections are reported in literature in the form of case series and reports. The most commonly reported infections were pneumonia's and bacteremia's, followed by meningitis, skin and soft tissue infections, ocular and native valve endocarditis (3). There have been very few reports of MDR community acquired pneumonias.

Case Study

A 80-year-old female patient, known case of hypertension, Ischemic heart disease (IHD undergone CABG) and recently detected compressive myelopathy of the lower spine with bilateral lower limb paresis of unknown cause. MRI of spine and CT guided biopsy done 6 months ago was inconclusive. She was admitted to our institution with complaints of fever for two days, loose stools (2-3 episodes) since two days and breathlessness since morning. On arrival in the emergency room, she was found to be drowsy with a GCS of 10/15. She was found to have a heart rate of 126/min, blood pressure of 100/70

mm hg, SpO_2 of 74% on room air. On examination patient was drowsy obeyed simple commands with lower limb power of 2/6 and upper limb 6/6. Respiratory system revealed bilateral rhonchi in mammary area, bilateral increased VF and infra-axillary crepitations, right more than left side. Other systems were essentially normal.

She was admitted to ICU with the provisional diagnosis of LRTI with sepsis. The initial work-up revealed (Table-1) low sodium (111 meq/lt), high creatinine (3.47mg/dl), Chest x-ray suggestive of right sided pneumonia covering the whole lung (Fig 1), metabolic acidosis on ABG and a total count of 16,930/cumm. The patient was started on NIV (Non-invasive ventilation), antibiotics (Meropenem and Azithromycin) and supportive management. She was started on 3% saline at 15ml/hr in view of the hyponatremia. Due to the patient's raised creatinine levels and bilateral grade I-II nephropathy on ultrasound, nephrology opinion was taken, continuing supportive management was suggested. Over the next 48 to 72 hours patient showed improvement in the form of reduced NIV support and FiO_2 requirements.

Her urine output improved and creatinine reduced to 1.64 from 3.47. Cardiology opinion was taken in view of previous history of CABG and was optimized on the cardiac drugs. Her sputum culture sent on Day 1 showed mixed flora. The second sample sent on Day 3 in view of the purulent copious sputum and, minimal improvement on chest x-ray showed Colistin only sensitive (COS) - Acinetobacter baumannii. During the course of the patient's ICU stay a neurosurgery reference was taken for her myelopathy, a pulmonary opinion was suggested to rule out malignancy. Pulmonary opined, and the HRCT done showed moderate pleural effusion and right consolidation with bronchiectatic changes and no evidence of tumor or metastases. A Colistin regimen with intravenous Colistin 3miu tid and nebulised Colistin 1miu tid for 7 to 10 days resulted in drastic improvement in the chest x-ray and oxygen requirement of the patient. Patient was discharged on day 16.

Discussion

Community-acquired pneumonia (CAP) is a one of the frequent infectious disease, ranking as the fifth leading cause of death globally.(4) According to the 2007 Infectious Diseases Society of America (IDSA)/American Thoracic Society (ATS) guidelines gram-negative bacteria (GNB) was the leading cause of CAP in patients admitted to the intensive care unit (ICU) for pneumonia.(5)

Acinetobacter infections are generally restricted to hospitals, especially to intensive care units (ICU). Community acquired infections due to Acinetobacter is uncommon but the most frequent among the community acquired infections is pneumonia.(3) It is frequently seen in tropical or subtropical climate, especially from Australia and Asia and in patients with comorbidities.(6) The frequent and indiscriminate use of antibiotics has increased the incidence of Acinetobacter infections and also antibiotic resistance.

Acinetobacter is unique in its ability to accumulate diverse mechanisms of resistance and emergence of strains that are resistant to all commercially available antibiotics. In a survey by Centers for Disease Control and Prevention (CDC), rates of carbapenem resistance in 3601 isolates of acinetobacter increased from 9% in 1995 to 40% in 2004.(7)

CAP due to MDR acinetobacter infections is rarely reported in India. They are characterized by aggressive pneumonia with high case fatality rates (>50%). The presence of comorbidities such as diabetes mellitus, COPD, renal disease, previous usage of antibiotics, cancer, chronic smokers, alcoholics and increasing age is known to increase the risk.(8) Community acquired strains of A. baumannii are more susceptible to antibiotics than

hospital acquired strains. Though the available literature in India claims that the incidence is less than 5-10%, the increasing multidrug resistance among gram negative infections has made it difficult to treat this group of patients as many of them present at a very late stage due to which they present with florid features of sepsis, shock and multi-organ failure, increasing the mortality to as high as 50%.

In regards to treatment, most strains of CAP-AB are susceptible to Ampicillin/Sulbactam and Quinolones, resistant to third generation cephalosporins due to AB Amp C B-Lactamases. The MDR acinetobacters are generally treated with carbapenems in combination with a polymyxin or tetracycline or glycylcycline (Tigecycline). The response to tigecycline is dependent on the relevant interpretive break points but on the whole noted to have a favorable response in minocycline resistant, multidrug-resistant and imipenem-resistant isolates. In the imipenem resistant ones there are multiple reports of failure. World wide acinetobacter response to aminoglycosides and fluroquinolones has been 60% and 44% respectively with lower rates for MDR variant. (9)

Our patient was elderly with multiple co-morbidities and was partially bedridden (required assistance for activities of daily living) due to myelopathy.

In conclusion, in this era of multi-drug resistance a failed initial course of antibiotics should prompt the physician to consider alternatives, including multi-drug resistance, based on the patient risk factors. An early and prompt initiation of high end antibiotics and appropriate de-escalation should ensure a good outcome. An effective antibiotic stewardship program implemented in every organization would help in curtailing resistance which is on the increase year on year..

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Conflict of interest - NIL

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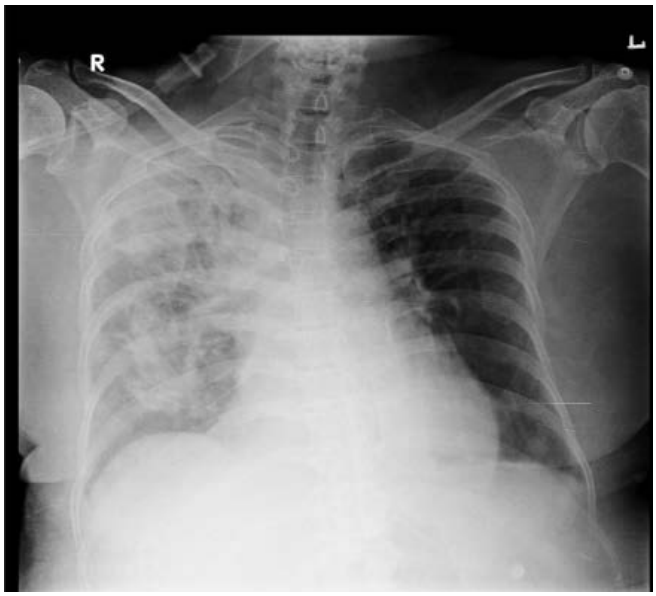

Figure 1 – Day 1 – Prominent right sided infiltrates

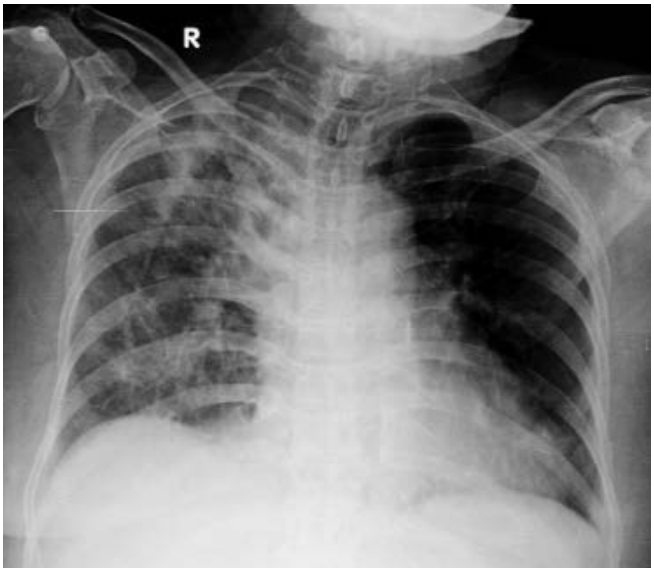

Figure 2 – Day 14 – Clearence of infection

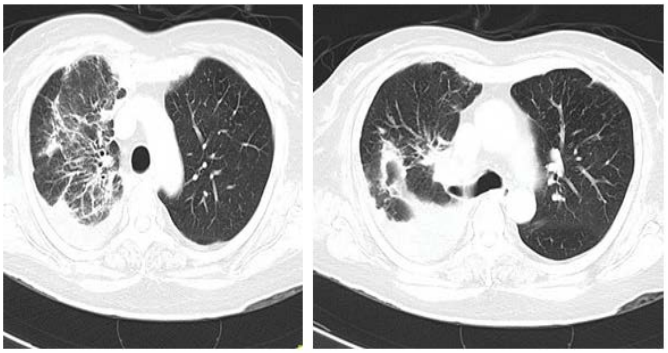

Fig 3 a & b – CT chest on Day 9 – Right sided resolving pneumonia with ground glass opacity.

	INV	DAY 1	DAY 2	DAY 4	DAY 5	DAY 9	DAY 10
1	Hemoglobin (gm/dl)	10.7					
2	PCV	33					
3	TLC (cells/mm^3)	16930					9670
4	Platelet count(cells/mm^3	221000					
5	Serum creatinine (mg/dl)	3.74	3.47	1.64	1.04	0.56	
6	BUN (mg/dl)	69.1	75.1	51.6	31.1	8.7	
7	Uric Acid (mg/dl)	6.8				2.8	
8	Sodium(mEq/l)	111.5	115.9	132	135.4	122.8	126.6
9	Potassium (mEq/l)	5.8	5.1	3.2	4.5	4.5	4.7
10	Chloride (mEq/l)	80.2	84.6	100.7	102.4	91.7	91.6
11	Urine Albumin	Nil					
12	Urine Sugar	Nil					
13	Urine WBC	18-20					
14	Urine RBC	1-2					
15	Urine Sodium mmol/L	26.4					
16	Ser Calcium(mg/dl)	8.1				7.5	
17	Ser Phosphorus (mg/dl)	6.4				3.0	
18	Ser TSH (microIU/ml)	2.98					
19	Free T4 (ng/dl)						
20	Cortisol, random	21.10					
21	Total Bil (mg/dl)	0.56					
22	Direct Bil (mg/dl)	0.89					
23	Total Protein (g/dl)	5.5					
24	Albumin (g/dl)	2.1					
25	Ser Globulin (g/dl)	3.4					
26	A/G Ratio	0.6					
27	D-Dimer		0.5				
28	HbA1C		6.6				

29	Peripheral Smear		Anemia with leucocytosis				