A young man with bilateral Hydro-Pneumothorax and Empyema following Community Acquired Pneumonia due to Methicillin Resistant Staphylococcus Aureus

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The Author declares that there is no conflict of interest.

Abstract

Staphylococcus Aureus is though, uncommon but important cause of community acquired Pneumonia because it carries high mortality. Staphylococcus Aureus is provided with many virulent factors and has the ability to change genetic structure to become resistance to antibiotics. Methicillin resistant *Staphylococcus Aureus* (MRSA) used to be the common cause of nosocomial pneumonia. But now it is an important cause of community acquired pneumonia in healthy young patients carrying high mortality. We report here a case of severe pneumonia caused by MRSA in a young patient complicated by bilateral hydro pneumothoraxes and empyema needing intercostal drainage and decortication.

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Introduction

Staphylococcus Aureus is an important cause of community acquired pneumonia (CAP) though less frequent than *Streptococcus Pneumonia* and *Mycoplasma Pneumonia*. It accounted for 1-5% of all community acquired pneumonia and usually followed influenza like illness.[1] It is second or third most common cause in patients with community acquired pneumonia requiring ICU admission. It accounts for 20-40 % of nosocomial Pneumonia and ventilator associated pneumonia.[2] It carries high mortality and morbidity because it is armed with variety of toxins and also because of resistance to several classes of antibiotics. It has acquired several genes that confer resistance to several classes of antibiotics like mecA that confers resistance to methicillin and beta lactam. Methicillin Resistance Staphylococcus Aureus (MRSA) is important cause of skin and soft tissue infection.[3] It is major cause of nosocomial Pneumonia, ventilator associated pneumonia and community acquired Pneumonia, all associated with high mortality. Many cases of pneumonia have been reported in the community caused by MRSA –known as CA –MRSA since 1990.[4] This strain of Staphylococcus Aureus is different from hospital /health care associated MRSA causing Pneumonia.[5] CA-MRSA causes severe necrotizing Pneumonia in young health individual wit high mortality.

CA-MRSA Pneumonia is defined by center for disease control and prevention (CDC) as illness compatible with CAP, in which MRSA was cultured from sputum or blood in an outpatient setting or < 48 hours after admission to hospital, and with none of the following health care risk; recent hospitalization, surgery, dialysis, or residence in long –term health care facility < 1 year before the onset of illness; and permanent indwelling catheter or percutaneous medical device.

Case report

A previously fit 16 yeas Afghan boy was admitted from ER in Rehman Medical Institute (RMI), Hayatabad Peshawar to MICU withe history of fever, cough, hemoptysis and breathlessness for the last 10 on 22/4/2019. Before coming to RMI, he was admitted in hospital in Afghanistan where he was diagnosed to have septicemia and pneumonia. His blood culture grew staphycoccus aures. He was treated with inj. Vancomycin and inj. Meropenem. There was no history of flu like illness before developing pneumonia. No history of injury or wound. He also complained of pain the R thigh.

O/E ill looking, tachypnoiec, Pyrexial temperature

102°F, BP 90/60

Chest examination revealed bilateral crackles and occasional wheeze

Abdomen was soft, no visceromegaly.

Skin examination did not reveal wound, injection mark, and rash

Pneumonia, ARDS and septicemia were diagnosed and he was admitted in medical ICU.

Investigations

Chest X-ray showed bilateral consolidation.

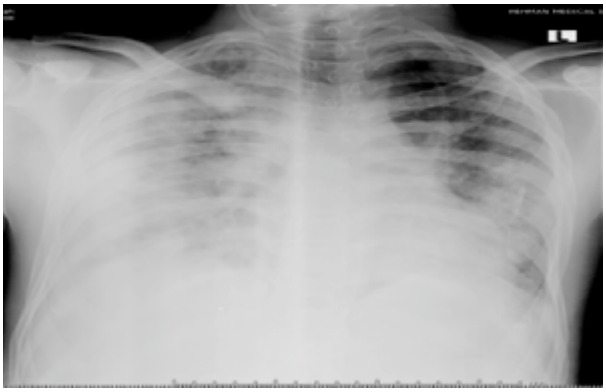


Figure 1. chest X-ray bilateral multilobar consolidation

Complete Blood Count: Hb 10.9g/dl, WBC 30.73x10^9, Platelets 216x10^9, MCV 74.8fl, Neutrophil 83.7%, Lymphocytes 8.4%, Monocytes 7.5%, Eosinophil 0.1%, Basophil 0.3%

Peripheral smear: RBCs show mild Anisocytosis, Micocytosis, White Blood Cells, Neutrophil, Leukocytosis,

CRP: 47.07mg/dl (<0.5)

VBG: pH 7-443, PCO_2 44.0mmHg, pO_2 22.4 mm Hg, HCO3 29.4mmol/L, Urea 30mg/dl, creatinine 0.59 mg/dl

Serum Electrolyte: Sodium 128 .4mmol/ L Potassium 4.79mmol/L, Chloride 93.5mmol/L bicarbonate 27.4 mmol/L

HIV: Ab Negative

HBsAg: Negative Anti HCV antibodies; Non- reactive

Blood C/S: Yielded no growth in RMI

Blood C/S: Staphylococcus Aureus in Afghanistan

Sputum Gram: Gram +ive Cocci seen

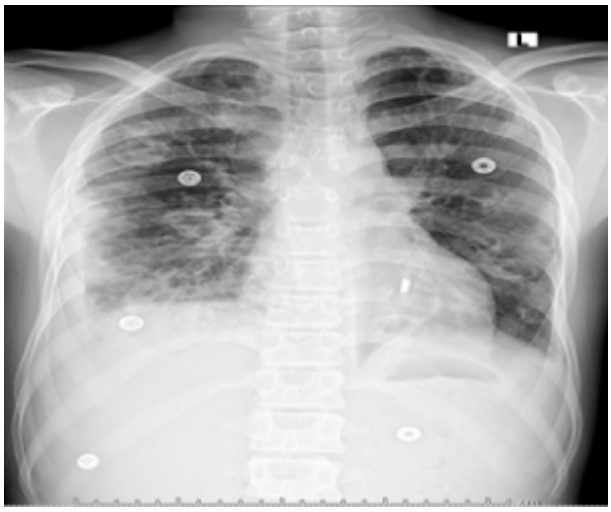
Sputum culture: Normal throat flora

Sputum cytology: Smear revealed moderate cellularity and consists of lymphocytes and multiple fragments superficial squamous epithelium. No malignant cells seen

Treatment: Started on inj. Imipenim 500mg TID IV and Vancomycin Inf. 500mg TID IV, Oxygen, bronchodialator and steroids

He was shifted to Medical C ward on April 25, 2019 when his condition improved and at the request of his father because of financial issues.

On April 27, 2019 noticed to have pleural rub on Left side of the chest and advised repeat chest X-ray in the radiology department. . It showed multiple cavitating lesion and R sided pleural effusion.



Fig; 2 Chest x-ray showing R sided pleural effusion and widespread bilateral cavitating lesions.

On 28th the patient developed R sided chest pain and breathlessness. Chest examination revealed increased resonance on percussion note. Chest X ray revealed R hydro-pneumothorax .

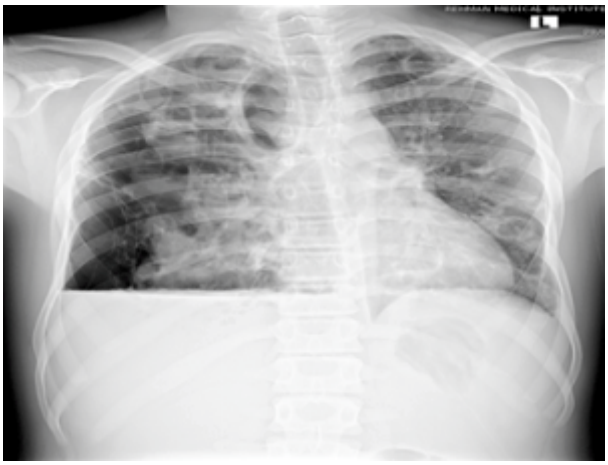


Figure 3: chest x-ray showing R sided air-fluid level, R sided consolidation and cavitating lesions on the L side.

ICD was put in the R pleural cavity and about 900 ml fluid was drained. Vancomycin was stopped and was stared on linezolid infusion the patient was referred to thoracic surgeon.

On 30th, became breathless at rest and also complained of cough and sputum .chest examination revealed resonant percussion note on the Left side of the chest and decreased intensity of breath sound. L pneumothorax was suspected and advised chest X-ray Chest X-ray showed L hydro-pneumothorax. ICD put in L pleural cavity.

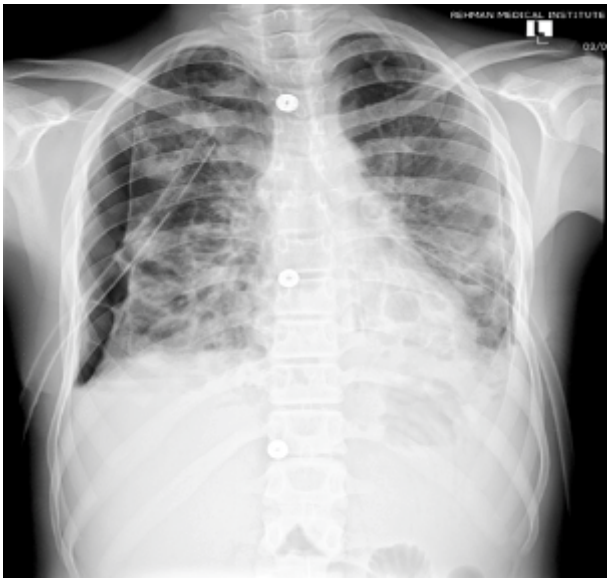


Figure 4: chest x-ray showing bilateral consolidation, cavitating lesions, bilateral pneumothoraxes and bilateral ICD tubes.

On 1st June pleural fluid C/S reveled MRSA so Amikacin was added to linezolid infusion

11thJune sputum C/S E. coli. So Amikacin was stopped and stared on Tienam Inj. And linezolid infusion was continued.

On June 12, 2019 he had thoracotomy for R side trapped lung for failure to expand because of thickened pleura and multiple abscesses. Decortication was done and 5 abscesses sites were drained.

Post operatively, he complained of chest pain at the operation site for which analgesic. He was gradually mobilized. He was discharged on will on June 15, 2019.

Discussion;

This case illustrates the devastating course of MRSA pneumonia in a previously young fit person. Pneumonia due to MRSA was complicated by bilateral hydro-pneumothoraxes and empyema despite antibiotics necessitating decortication. Previously MRSA was considered a hospital or health care related facilities pathogen but it is emerging as an independent pathogen in the community. The new strain of MRSA causing community acquired infection is known as CA-MRSA.[3] These new clones of MRSA carry virulent gene including Panton-Valentine leucocidin making infection more severe than usual. Also these infection occur in young healthy individual and children without prior history of contact with health care facilities, as in our case. Certain group of people are more prone to CA-MRSA infection like people living in barracks, military recruits, prisoners, IV drug addicts and homeless. The mortality in CA-MRSA varies between 20-60%.[6]

These patients present with fever, hemoptysis, breathlessness, hypotension and hypoxia.CA-MRSA pneumonia is rapidly progressive and frequently leads to septic shock. These care are often complicated by extensive lung necrosis, lung abscesses formation and empyema.[7] The chest x-ray usually shows multilobar cavitating alveolar infiltrates.[2]

The main characteristic reported in the literature for patients having MRSA pneumonia was severity of pneumonia. But the etiology of severe CAP is quite diverse. A combination of severe pneumonia following flu like illness in younger age gives a clue about the etiology of MRSA pneumonia. However several studies have shown that CA- MRSA pneumonia may occur in the absence of antecedent influenza or other viral respiratory infection.[8]

Few patient with MRSA pneumonia in the community receive appropriate antibiotic initially, because of low incidence of CA-MRSA pneumonia. However, the guidelines by the Canadian thoracic society and Canadian Infectious diseases Society recommend the use of vancomycin or linezolid in the case of hospitalized patients with severe community –acquired pneumonia.[9]

Vancomycin and linezolid are considered as the optimal option for CA-MRSA.[10] Linezolid is preferred over vancomycin because of less toxin release. CA-MRSA is also susceptible to fluroquinolone, clindamycin, trimethoprime-sulphamethoxazole and rifampicin.[1] The duration of treatment for non - bactraemic CA-MRSA pneumonia is 7-8 days and for bacteremia the duration of treatment is 14 days.

This case illustrate the importance of MRSA as a causative agent for community acquired pneumonia with many complications, especially in the previously young and fit individuals. Is there need for guidelines for treating community acquired pneumonia to suggest adding appropriate antibiotic against MRSA very early to avoid these complications?

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