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A challenging coexistence: community-acquired methicillin-resistant *Staphylococcus aureus* and *Mycobacterium tuberculosis*

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Abstract

Background Community-acquired Methicillin-resistant *Staphylococcus aureus* (CA-MRSA) usually emerges after a viral infection and causes severe disease in immunocompetent individuals. Concurrent infection with tuberculosis (TB) is generally very rare in immunocompetent patients. Our case is the first report of the coexistence of CA-MRSA and TB in an immunocompetent patient.

Case presentation A 24-year-old male patient of African origin, who has been living in Turkey for a year, was admitted to our hospital 3 months ago with fever, cough, and sputum complaints, which developed following symptoms of influenza infection. More intense bilateral infiltration and cavitary appearance were observed on the left in the chest radiography of the patient who did not respond to amoxicillin and gemifloxacin treatments. The patient's sputum culture showed MRSA growth, and his sputum acid-resistant bacteria (ARB) was reported as three positive. Vancomycin, isoniazid, rifampicin, pyrazinamide, and ethambutol treatments were started. Subsequently, *Mycobacterium Tuberculosis* growth was also detected in the mycobacteria culture. Vancomycin treatment was completed in 14 days. There was no growth in the control sputum culture. When the patient, who gave clinical and laboratory response, was admitted with increased shortness of breath complaint two months after discharge, it was observed that minimal spontaneous pneumothorax developed in the left lung, and it was decided to follow up without intervention. In the second month of tuberculosis treatment, sputum ARB and mycobacteria culture became negative, and the patient was switched to dual antituberculosis treatment (isoniazid, rifampicin), and his treatment is still ongoing.

Conclusions Mixed infections should be considered in case of non-response to treatment in patients with pneumonia. Mixed infections should also be followed closely as they may be more complicated.

Keywords CA-MRSA, Tuberculosis, Pneumothorax

Background

Staphylococcus aureus, a member of the Staphylococcaceae family, is a human pathogen that lives as a commensal in the armpit, pharynx, vagina, perineum, or eroded skin, with a rate of 30–50% in the anterior nasal mucosa [1]. *S. aureus* has been reported to colonize approximately 30% of the world's population [2]. Methicillin-resistant *S. aureus* (MRSA) can be a causative agent in both community-acquired (CA-MRSA)

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and hospital-acquired pneumonia [3]. The estimated CA-MRSA prevalence is approximately 0.51–0.64 per 100,000. Mortality in CA-MRSA pneumonia is significantly higher than in methicillin-sensitive strains and has been reported as 56–63% [4].

Many cases of CA-MRSA occur after a viral infection. Even in immunocompetent individuals, lung infiltration, necrosis, hemoptysis, and severe sepsis can cause septic embolism [5–7]. Concomitant infection with tuberculosis is generally very rare in immunocompetent patients. Our case is the first report of the coexistence of community-acquired MRSA and TB in an immunocompetent patient.

Case presentation

A 24-year-old male patient of African origin, who has been living in Turkey for a year, was admitted to our hospital 3 months ago with symptoms of fatigue, muscle pain, and sore throat, followed by fever, cough, and sputum. More intense bilateral infiltration and cavitary appearance were observed on the left in the chest radiography of the patient who did not respond to amoxicillin and gemifloxacin treatments (Fig. 1a). Sputum acid-resistant bacteria (ARB) results were found to be negative twice. The patient, whose complaints of fever, dyspnea, cough, and sputum continued to increase, was hospitalized and examined. There was no feature other than a fever of 38.2 °C and inspiratory rales in the upper zone of the left lung in physical examination. No BCG scar was observed. It was learned that the patient, who had no comorbidities, did not use medication, cigarettes, alcohol, or drugs and did not have TB. Anti-HIV test was negative. Laboratory tests of the patient were leukocyte 5.5 K/μL (normal 4.5–11 K/μL), lymphocyte: 0.57 K/μL (normal 1.1–5.1 K/μL) (10.4%), neutrophil: 4.61 K/μL (normal 1.8–7.3 K/μL) (83.8%), CRP 171 mg/L (normal 0–5 mg/L), AST 88 U/L (normal 5–34 U/L), ALT 58 U/L (normal 0–55 U/L), and kidney function tests were normal. Vancomycin treatment was started for the patient due to MRSA growth in the sputum culture. When the sputum ARB was sent again, three positives were detected, and the patient was started on isoniazid, rifampicin, pyrazinamide, and ethambutol treatment. *Mycobacterium tuberculosis* was also detected in the mycobacterial culture in the follow-up. Anti-tuberculosis drug resistance test was negative. Vancomycin treatment was completed in 14 days. There was no bacterial growth in the control sputum culture. The patient received clinical and laboratory responses and was discharged. After two months, the patient was admitted with complaints of increased dyspnea and a new spontaneous pneumothorax on the left was observed on chest radiography (Fig. 1b). Thorax computed tomography (CT) showed bilateral infiltration, necrotic parenchyma, and minimal pneumothorax on the left (Fig. 2). Since the patient's clinical condition was stable, it was decided to follow up without intervention. In the 2nd month of tuberculosis treatment, sputum ARB and mycobacteria culture became negative, drug resistance test was also negative dual antituberculosis treatment (isoniazid, rifampicin) was started and treatment is still ongoing.

Discussion

Whether or not pneumonia develops is determined by the host's immune response, the virulence of the infecting organism, and the presence of vaccination.

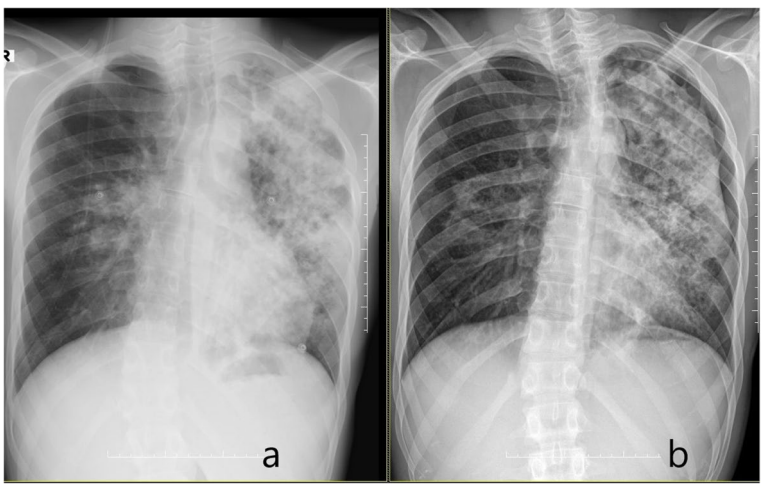

Fig. 1 **a** The chest X-ray reveals bilateral infiltration and cavitary appearance, more intense on the left. **b** The chest X-ray reveals a pneumothorax on the left compared to the previous radiograph

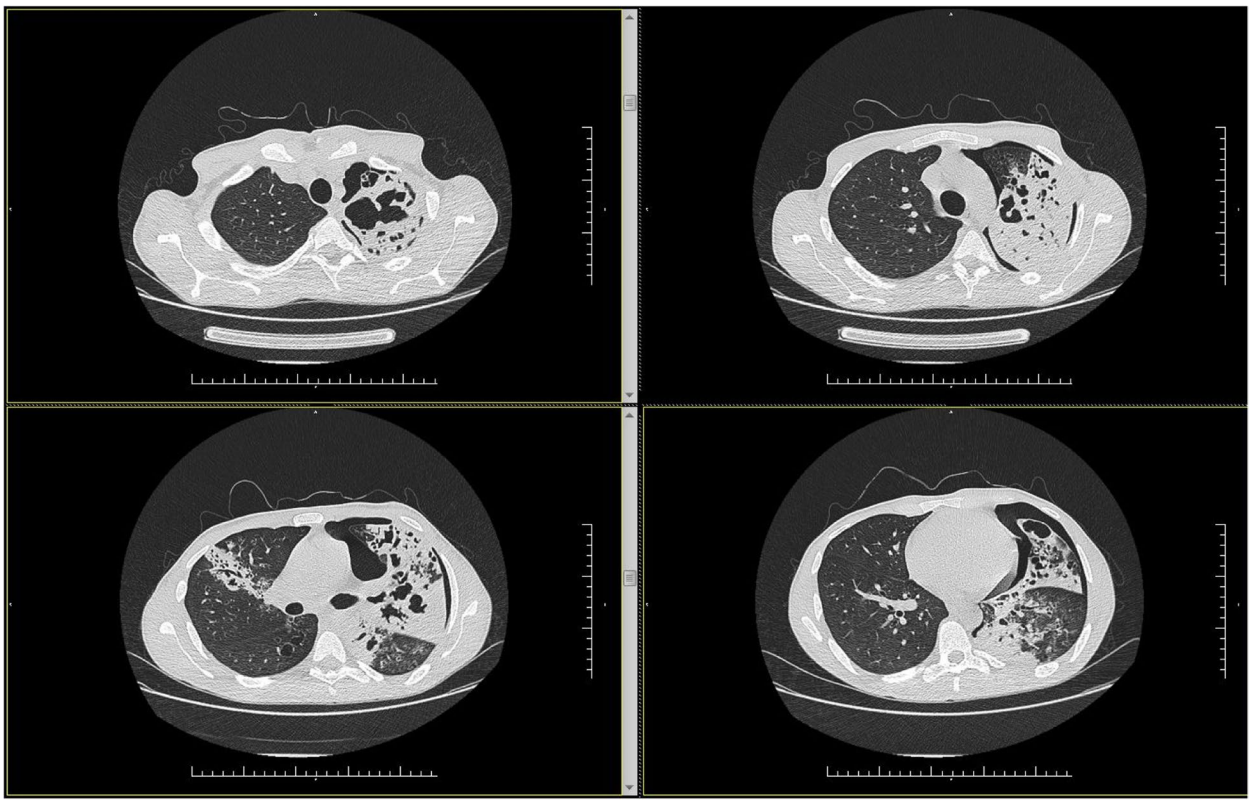

Fig. 2 Thorax CT reveals bilateral consolidation with cavities, more intense on the left, and minimal pneumothorax on the left

Comorbidities such as impaired cough reflex and mucociliary clearance, impaired local and humoral immunity, alcohol use and smoking, chronic obstructive pulmonary disease (COPD), congestive heart failure, chronic kidney disease, liver disease, and immunodeficiency conditions constitute a predisposition for the development of community-acquired pneumonia. CA-MRSA pneumonia, on the other hand, usually occurs after influenza infection in patients who have not previously been exposed to any health care [8]. Influenza can weaken T cell immunity and innate immune responses to secondary bacterial infections [9]. Thus, it can also pave the way for the reactivation of latent tuberculosis. In our case, both CA-MRSA and *Mycobacterium tuberculosis* were found to be the causative agent of pneumonia that developed following flu-like symptoms. We aimed to contribute to the literature by reporting this association in an immunocompetent patient for the first time.

The Centers for Disease Control and Prevention (CDC) definition of CA-MRSA infection includes the presence of a positive MRSA culture isolated within 48 h of admission, absence of indwelling catheters or medical devices permanently implanted in the skin, absence of a history of prior MRSA colonization and absence of a recent history of significant hospitalization [10]. Our case also meets these criteria.

The burden from staphylococcal infections in Africa is overshadowed by the common diseases HIV/AIDS, tuberculosis, and malaria [11]. Surveillance studies of bacterial infections in Africa indicate that *S. aureus* is a common pathogen in healthy adults immunocompromised individuals, and those with a genetic predisposition [12]. It has also been reported that MRSA prevalence can rise to 78.6% in African countries [13]. The fact that our patient was of African origin suggests that the possibility of being colonized with MRSA is high.

CA-MRSA pneumonia should be considered in the differential diagnosis, especially in healthy young individuals with rapidly progressive clinical features, including cavitary consolidation, bilateral infiltration, pleural effusion, and hemoptysis [14]. In patients with severe disease, MRSA can cause lung necrosis by producing various exotoxins, including Panton-Valentine leukocidin [15]. Panton-Valentine leukocidin toxin could not be tested in our case. We also think that in our case, accompanying TB also increased the necrotic/cavitary appearance in the lung.

Although parapneumonic effusion, empyema, necrotizing pneumonia, pneumothorax, and lung abscess

are more common as acute complications of bacterial pneumonia [16], pneumothorax may also develop. Spontaneous pneumothorax is caused by visceral pleural tear resulting from subpleural bleb or necrotic parenchymal process or hyperinflation of distal airways due to dynamic bronchiole obstruction resulting in check-valve and subsequent alveolar rupture [17]. Secondary spontaneous pneumothorax occurs due to an underlying lung disorder and, therefore, has a worse prognosis than primary spontaneous pneumothorax [18]. Pneumothorax developing in severe CA-MRSA pneumonia may develop secondary to barotrauma and underlying lung necrosis in intubated patients [19]. In some series, it has been reported that pneumothorax develops in 60% of patients with severe CA-MRSA pneumonia [20]. Spontaneous pneumothorax secondary to pulmonary TB may develop in rare cases of residual fibrosis with retractions and bullae [21]. The frequency of spontaneous pneumothorax in active pulmonary TB is approximately 1–2% [22]. In our case, the coexistence of both MRSA and TB may have led to the development of pneumothorax.

Multidrug resistance has been reported in CA-MRSA strains, but in general, these organisms are more drug-sensitive than similar hospital-acquired infections [23]. In our case, there was no multidrug resistance, and there was a response to treatment with Vancomycin.

Conclusion

Mixed infections should be considered in patients being followed up with a diagnosis of pneumonia, especially in cases where there is no response to treatment. Mixed infections also require close follow-up as they may be more complicated.

Abbreviations

CA-MRSA	Community-acquired Methicillin-resistant *Staphylococcus aureus*
TB	Tuberculosis
ARB	Acid-resistant bacteria
CRP	C-reactive protein
AST	Aspartate aminotransferase
ALT	Alanine aminotransferase
CT	Computed tomography
COPD	Chronic obstructive pulmonary disease

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Authors' contributions

EA wrote the initial draft of the manuscript. AS and AEG managed the diagnosis and treatment. EA, AS, and AEG approved the final version of the manuscript and agreed to be accountable for all aspects of the work. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Declarations

Ethics approval and consent to participate

Written and verbal informed consent was obtained from the patient for publication of this case report.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor of this journal.

Competing interests

The authors declare that they have no competing interests.

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