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Introduction:

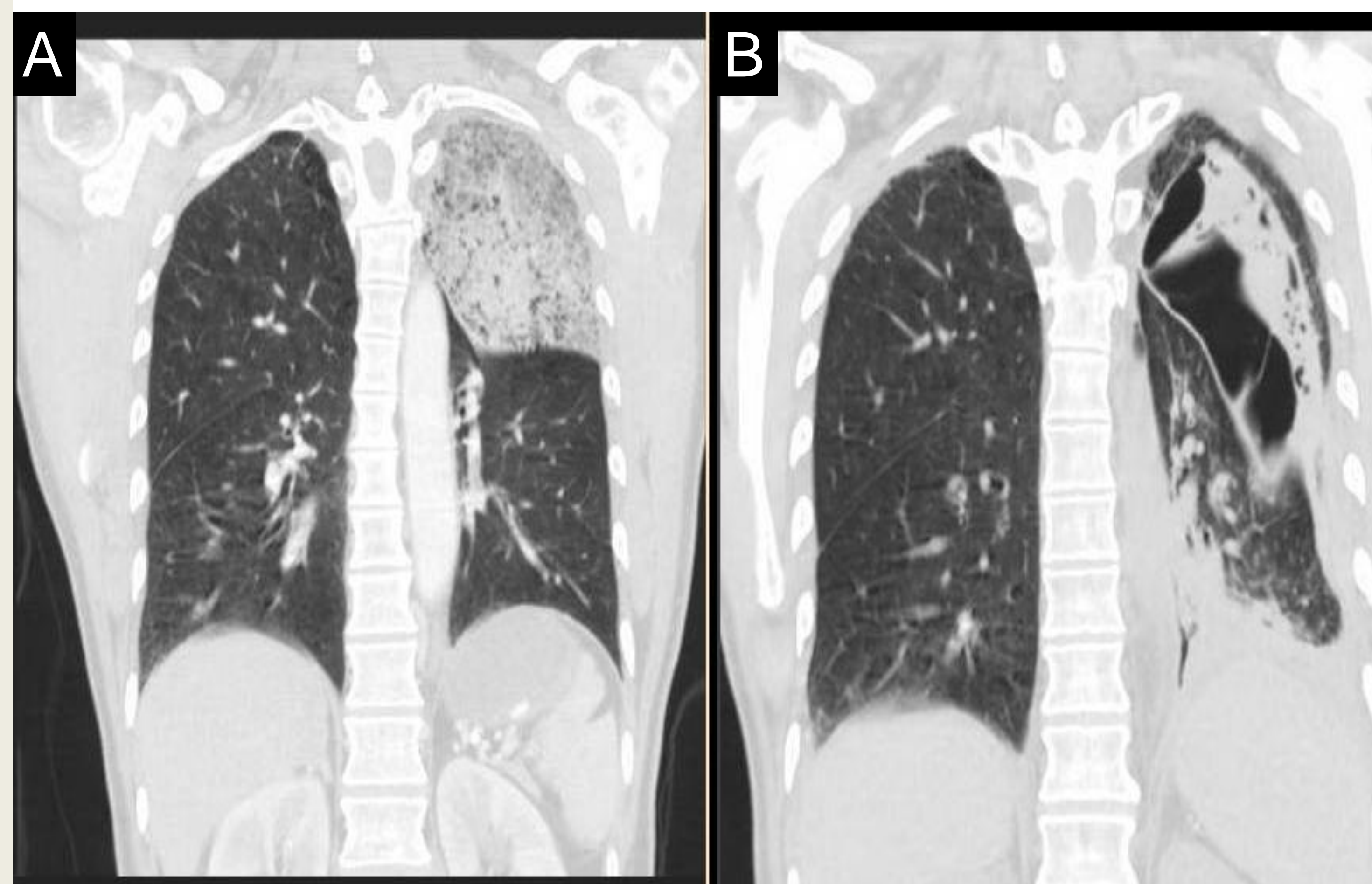
Staphylococcus aureus (SA) is a common cause of community acquired pneumonia (CAP). Both methicillin-sensitive *S. aureus* (MSSA) and methicillin-resistant *S. aureus* (MRSA) strains can cause rapidly progressive necrosis by secreting a cytotoxin called Panton-Valentine leukocidin (PVL), but reported cases are largely related to MRSA strains¹. PVL-induced necrotizing pneumonia (NP) may further serve as a precipitating event for spontaneous secondary pneumothorax (SSP). We report a case of MSSA -community acquired pneumonia complicated with recurrent SSP.

Case Description:

A 54-year-old male with no past medical history presented with left-sided pleuritic chest pain associated with shortness of breath. At presentation he was hypotensive and tachycardia, but afebrile. Oxygen saturation was 94% on 4L/m. Physical exam revealed rapid, shallow breathing with wheeze in the left lung. Labs revealed neutrophilic leukocytosis and thrombocytopenia with bandemia. Chest x-ray showed left upper lobe haziness. Chest CT revealed left upper lobe consolidation and no pneumothorax (Figure 1A). Blood gas showed hypoxemia with normal pH. Urine antigen and influenza tests were negative. He was started on IV hydration and combination antibiotic therapy. The following day he was found in respiratory distress with worsening desaturation. Blood gas revealed severe respiratory and metabolic acidosis. He was intubated.

Follow-up x-ray showed worsening left-sided opacity and antimicrobial coverage was broadened accordingly. Bronchoscopy with BAL grew MSSA; antibiotic therapy was de-escalated. Pleural fluid boluses were consistent with empyema. Pigtail catheter was placed. Follow-up CT was in favor of necrosis inside the consolidation (Figure 1B). He improved clinically. Repeated imaging studies revealed left-sided SSP. Chest tube was placed with re-expansion of the lung. He had a prolonged hospital stay with weakness necessitating transfer to rehabilitation facility. While awaiting disposition, he developed another SSP on the left side.

Figure 1:



Coronal chest CT views (A) with IV contrast on presentation showing left upper lobe consolidation and (B) with out contrast on follow-up revealing a necrotic cavity.

Discussion:

The mortality associated with CAP in ICU patients is reported to be 50%² and can be even higher with progression to necrosis. SA-producing PVL toxin causing NP can be highly lethal even in immunocompetent patients. SSP can complicate the course of NP. In this setting, SSP can be associated with extension of bacterial infection into the pleura and development of empyema. Management of SSP is complicated due to underlying lung disease, which increases the likelihood of persistent air leakage and further expansion of the pneumothorax. Hence, most patients with SSP will require a pleural drainage. A high clinical suspicion of NP early in the course of disease is crucial in guiding appropriate antimicrobial therapy to minimize complications such as SSP. Laboratory PVL toxin would be helpful in guiding therapy and anticipating a complicated course in MSSA pneumonia.

References:

1. Lina, G., Piemont, Y., Godail-Gamot, F., Bes, M., Peter, M.-O., Gauduchon, V., ... Etienne, J. (1999). Involvement of Pantone-Valentine Leukocidin--Producing *Staphylococcus aureus* in Primary Skin Infections and Pneumonia. *Clinical Infectious Diseases*, 29(5), 1128–1132.
2. Restrepo MI, Faverio P, Anzueto A. Long-term prognosis in community-acquired pneumonia. *Current opinion in infectious diseases*. 2013;26(2):151-158.