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Thrombocytosis in a patient with healthcare-associated pneumonia combined with severe sepsis

Qasim Malik[1], **Qi Shi**[1], **Lisa Thomas**[2], **Suraj Thangada**, **Bo Jiang**[1], **Louis Crevecoeur**[1], **Dinesh Keerty**[1] and **Sahana Pai Dongerkary**[1]
[1]The Wright Center for Graduate Medical Education, USA
[2]Hematology and Oncology Associates of Northeastern Pennsylvania, USA

Introduction: Healthcare-associated pneumonia (HCAP) is the second most common type of pneumonia. Mortality rates associated with HCAP have been reported as high as ~20%. Recent clinical data show that thrombocytosis may be a predictive factor of mortality with community-acquired pneumonia (CAP). However, thrombocytosis in patients with HCAP has been less reported.

Case presentation: A 62-year-old male with a history of chronic obstructive pulmonary disease (COPD) was admitted with shortness of breath and dry cough for about 1 week. 2 weeks before presentation, patient was hospitalized and treated for COPD exacerbation. Physical examination indicated a temperature of 98F and a blood pressure of 110/60 mmHg. His respiratory rate was 28 breaths/min, pulse was 110/min, and O2 saturation was 93% at room air. Examination of his chest revealed crackles at the base of left lung. His WBC count was 38.23K/μL with 93% neutrophils, Platelet count was 962K/μL, BUN was 109 mg/dl, and Creatinine was 1.8 mg/dl. AST/AST were 454/344 Unit/L. The total bilirubin was 3.7mg/dl. Lipase was 4332 units/L. Chest X-ray showed left lower lobe infiltrate. Patient was initially treated with empiric antibiotics, including intravenous levaquin, Zosyn, and vaconmycin for 7 days. Since patient's symptoms had improved, he was treated with Zosyn alone for another 3 days; meanwhile, blood cultures were negative. However, the patient was unable to produce a sputum sample. Repeated platelet, WBC, BUN/Creatinine, and liver function were normal before discharge, and JAK2V617F was negative.

Discussion: Reactive thrombocytosis can be frequently observed as a marker of normal reaction of infection, as platelets can undergo chemotaxis and are able to release numerous proinflammatory molecules. Recent clinical data show that in patient with CAP, there is an association between levels of inflammatory cytokines and severity of disease. Also, thrombocytosis at the time of hospital admission predicted worse clinical outcome in CAP, but it is also independently associated with increased length of hospitalization. HCAPs have demonstrated the increased risk for colonization and infection with multidrug-resistant pathogens, and increased morbidity and mortality. However, the role of platelets in the outcomes of patients with HCAP has been less studied.

Conclusion: Our case presents HCAP combined with severe sepsis and thrombocytosis at the time of hospital admission that suggests thrombocytosis can be an important clinical feature for severe HCAP, which increases the length of hospitalization.

malikq@thewrightcenter.org