Troponin Leak: A Puzzle

Introduction: Elevated troponin in patients hospitalized with non-cardiac systemic illnesses is commonly attributed to demand ischemia. Clinicians should be careful not to always dismiss elevated troponin in such cases as type 2 myocardial ischemia (MI). We present a case of incidental type 1 MI in a patient admitted for pneumococcal pneumonia.

Case presentation: A 79-year-old female admitted with complaints of generalized weakness. Initial workup showed leukocytosis, elevated procalcitonin, and a right lower lobe consolidation on chest X-Ray; initial troponin was 0.1 ng/mL, which then peaked at 10 ng/mL. No ischemic changes were seen on electrocardiogram, and she remained without chest pain or shortness of breath.

Treatment: The patient underwent coronary angiography revealing 90% mid-LAD occlusion with successful drug-eluting stent placement in mid LAD. The patient was then started on goal-directed medical therapy.

Discussion: Demand ischemia with elevated troponin is common in the setting of infection and sepsis, especially in those with a previous history of coronary artery disease. However, we should consider the possibility of type 1 MI in the setting of localized infection, even in those patients without significant risk factors. Corrales-Medina et al. reported a higher incidence of cardiac events in patients with pneumonia who were also found to have advanced age, history of prior cardiac arrhythmias, BUN >29 mg/dL, sodium < 130 mmol/L, hematocrit < 30%, and those with new pleural effusions. These signs and symptoms associated with a significant troponin elevation in a patient with acute infection should raise suspicion for a type 1 MI and may warrant a more in-depth investigation. Pneumonia from any cause has been associated with acute coronary syndromes, with the highest risk being during acute infection. Musher et al. found a rate of myocardial infarction of 7 to 8% among patients who were hospitalized for pneumococcal pneumonia. Other infections like influenza and urinary tract infections have been also associated with increased risk for adverse cardiac events. These infectious processes continue to place patients at an increased morbidity and mortality risk for up to 10 years when compared to the normal population. This is believed to be a downstream effect seen from the activation of the inflammatory pathway via interleukins and catecholamines.