Silent Purulent Pericarditis in a Patient With Rheumatoid Arthritis
Discovered During Cardiac Surgery

Brandon Doty, RN, OMS-II — New York Institute of Technology College of Osteopathic Medicine – Jonesboro, AR
Bridget Lee, DO, PGY-2 – St. Bernards Medical Center – Jonesboro, AR
Ahmed S. Ahmed, MD, FACC – St. Bernards Medical Center – Jonesboro, AR

INTRODUCTION

While purulent pericarditis is a well-documented phenomenon in patients with rheumatoid arthritis, very little has been noted in literature about the development of pericarditis and pericarditis in patients when it is well-controlled. Improved screening modalities and increased awareness of this phenomenon would allow for earlier intervention, decreased healthcare cost, increased antibiotic stewardship, and improved patient outcomes overall. Osteopathic treatments could also aid in improved outcomes in this patient population.

CASE PRESENTATION

A 79-year-old male with a history of rheumatoid arthritis (RA) well-controlled on sulfasalazine presented with a ten-hour history of chest and left arm pain. Initial troponin I level was normal, and electrocardiogram (EKG) showed no ischemic changes. Transthoracic echocardiogram (TTE) showed reduced left ventricular function and mild aortic valve insufficiency. Coronary angiogram subsequently revealed multi-vessel coronary artery disease. The patient underwent coronary artery bypass grafting (CABG) several days later. Upon surgical opening of the pericardium, murky fluid and obvious pericarditis were discovered. Pericardial fluid culture and gram stain showed no bacteria but marked purulence. The infectious disease consultant determined the purulent pericarditis to be asymptomatic and likely related to RA despite the previous accepted remission status. Post-operative recovery was uneventful.

BACKGROUND

RHEUMATOID ARTHRITIS

Rheumatoid Arthritis (RA) is an autoimmune disease of the synovial membrane which destroys articular cartilage and nearby bone, often causing severe disability and pain. It occurs in five to six adults with a higher prevalence in women compared to males. Joint manifestations are caused by activation of endothelial cells and neovascularization of the synovial membranes with subsequent invasion with T-cells, B-cells and monocytes. Patients often experience extra-articular manifestations which are often cardiovascular in nature, including pericarditis, pleuritis, myocardial disease, vasculitis, vascular disease, and arthritis. Although RA has been present for several decades, unfortunately, advancement in treatment has failed to improve outcomes. Current standards of care for pharmacological treatment include initiation of a continuous conventional disease-modifying antirheumatic drug (DMARD), subsequent addition of a biological DMARD, or a targeted synthetic DMARD until remission is reflected in nDIL monitoring and patient presentation.

PERICARDITIS

Pericarditis is a condition in which the pericardium becomes inflamed due to an immune response of various infectious or inflammatory etiologies. It is often accompanied by chest pain that is relieved with forward lean and exacerbated by coughing or deep breathing, as well as ECG changes and pericardial effusion. Cause can be idiopathic or a manifestation of some systemic disorder, often autoimmune or inflammatory in nature. Around 5% of all chest pain patients have emergency room admission are due to pericarditis, most being idiopathic. Only 14% of cases have either a viral or bacterial infectious etiology. Classic treatments classic treatments include nonsteroidal anti-inflammatory drugs, colchicine, corticosteroids, and management of any underlying cause. The disease is typically self-limiting and not life-threatening.1

HOSPITAL COURSE

ADMISSION

Vital Signs: Temperature 98.4°F, Heart Rate 75 beats per minute, Respiratory Rate 20 breaths per minute, Blood Pressure 163/81 mmHg, Oxygen Saturation 96% on room air

Admission EKG: Normal sinus rhythm with no acute ischemic changes

Physical Exam: Alert and oriented. Mucous membrane moist without significant venous distortion or peripheral edema. Normal S1 & S2 heart sounds without S3 or S4 gallops, murmurs, clicks, or rubs. Lungs clear to auscultation bilaterally. Bowel sounds normoactive. No palpable lymphadenopathy. No joint tenderness or changes in range of motion.

Leukograms:

<table>
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<tr>
<th>WBC</th>
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<tbody>
<tr>
<td>RBC</td>
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<tr>
<td>Hgb</td>
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Triglycerides 52

LDL 310

HDL 40

Tryptophan 0.082

Protein 5.71

REMAINING HOSPITALIZATION

Imaging: TTE demonstrating hyperesophagus pericardium in the parasternal long and short axis and subcostal four chamber views, which was noted by the initial interpreting physician when reading the preoperative study.

UNIQUE ASPECTS OF CASE

After a sweeping review of existing literature, it does not seem that intraoperative discovery of asymptomatic purulent pericarditis in a seemingly well-managed RA patient is something that has been reported. Prior to surgery, the patient had a TTE, radiograph and computed tomography scan of the chest, and a coronary angiogram which revealed no underlying process because the pericardial fluid was everted. There exist only a few reports that discuss pericarditis and pericarditis and RA. The case published noted discovery at the point in which the pericarditis has become constricting, causing heart failure.4 Pericardial effusion is a well-documented complication of RA, but less than 10% of RA patients develops pericarditis. A 20-year single-center retrospective review of 35 patients showed purulent pericarditis to be almost always related to infection and often only discovered post-mortem.5

REFERENCES


OSTEOPATHIC CONSIDERATIONS

Patients with pericardial effusion and pericarditis may benefit from osteopathic approaches to treatment and aid in restoring homeostasis. Likewise, an early thorough osteopathic structural exam could have led to discovery of his condition prior to surgery. For this case, the following interventions would be indicated in adherence with the the Five Models of Osteopathic Treatment:

• Biomolecular: Identify and treat dysfunctions along the RA to RA segments and the upper ribs.
• Respiratory and Circulatory: Identify and treat changes in musculoskeletal tone along the thoracic spine, ribs, and diaphragm to allow for improved arterial and venous exchange and lymphatic outflow. Treat Chapman’s reflexes at the left intercostal space between ribs 2 and 3 and between T2 and T3 left-sided transverse processes. Special attention should be paid to the C5 and C7 vertebrae and the respiratory diaphragm given their attachments to the pericardium.2
• Neurologic: Recognize increased sympathetic tone with acute processes and work to restore homeostatic parasympathetic dominance, increasing coronary blood flow and lymphatic outflow. Identify and treat vagal reflexes at the C2 segment and the occipitocervical junction, and cardiac vasoconstrictors from T2 to T4.
• Metabolic: Provide counseling to maintain proper exercise and diet to optimize healing and avoid triggers of inflammation.
• Behavioral: Ensuring adequate management of allostatic load, including environmental stressors, lifestyle events, and management of current illness will better facilitate improved outcomes overall.

DISCUSSION

This patient’s presentation seemed to be consistent with someone having multivessel coronary artery disease needing surgical revascularization. He exhibited classic chest pain but lacked conventional risk factors for heart disease beyond his age, gender, and history of autoimmune disease. He had the usual pre-operative evaluation and imaging, but typical signs of pericarditis were absent. His TTE showed a hyperesophagus pericardium. In the noninvasive setting was only noted to be pertinent during a retrospective analysis of this case in preparation for our report. His case and presentation sparked little to no concern for complications or sequelae of surgical management post-operatively. Unfortunately, due to the unexpected nature of his pericarditis, planned LAA clipping was canceled. Given his history of paroxysmal atrial fibrillation, he was thus required to endure ongoing risk of oral anticoagulation until he was fully recovered from surgery and able to undergo LAA closure device placement at a later date. The patient was also subjected to several days of unnecessary broad-spectrum antibiotics until cultures returned negative. This may have been avoided if these chronic changes had been discovered prior to surgery.

CONCLUSIONS

Further review of the incidence rate of asymptomatic pericarditis in RA patients should be considered, as well as creation of protocols for providers to screen prior to invasive cardiac procedures. Furthermore, while there are reports of effective use of OMT in patients with pericarditis and pericardial effusion, randomized controlled trials with large sample sizes of OMT use could improve implementation of these techniques within this population. As pericarditis pericarditis is often asymptomatic, it is likely underestimated. Maintaining clinical suspicion during preoperative workups in patients with RA is critical.