NEW YORK INSTITUTE OF TECHNOLOGY
College of Osteopathic Medicine at Arkansas State University

# 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

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## INTRODUCTION

While pericardial effusion is a well-documented phenomenon in patients with rheumatoid arthritis, very little has been noted in literature about the development of pyopericardium 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 three-hour history of chest and left arm pain. Initial troponin I level was normal, and electrocardiogram (ECG) showed no ischemic changes. Transthoracic echocardiogram (TTE) showed reduced left ventricular function and wall motion abnormalities. Coronary angiogram subsequently revealed multi-vessel coronary artery disease. He 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 aseptic 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[1]. It occurs in five in 1,000 adults with a higher prevalence in people assigned female at birth[1,2]. 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[2]. Patients can also have extra-articular manifestations which are often cardiovascular in nature, including pericarditis, pericardial effusion, myocardial disease, vasculitis, valvular disease, and atherosclerosis[1]. In recent years, however, advances in treatment have led to improved outcomes. Current standards of care for pharmacologic treatment include initiation of a conventional synthetic disease-modifying antirheumatic drug (DMARD), subsequent addition of a biologic DMARD, then a targeted synthetic DMARD until remission is reflected in lab monitoring and patient presentation[2].

### PERICARDITIS

Pericarditis is a condition in which the pericardium becomes inflamed due to an immune response of various infectious or aseptic 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 evaluations in emergency rooms are due to pericarditis, most being idiopathic. Only 14% of cases have either a viral or bacterial infectious etiology. 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[3].

## HOSPITAL COURSE

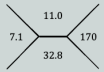
### ADMISSION

**Vital Signs:** Temperature 98.4°F, Heart Rate 75 beats per minute, Respiratory Rate 20 breaths per minute, Blood Pressure 168/85 mmHg, Oxygen Saturation 96% on room air

**Admission ECG:** Normal sinus rhythm with no acute ischemic changes

**Physical Exam:** Alert and oriented. Mucous membrane moist without jugular venous distention 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.

**Labs:**



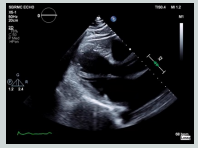


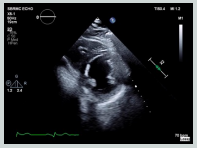
Triglycerides 52
Cholesterol 150
LDL 100
HDL 40

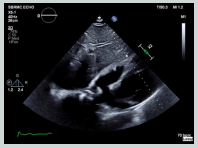
Troponin < 0.012
ProBNP 471

### REMAINING HOSPITALIZATION

**Imaging:** TTE demonstrating hyperechoic pericardium in the parasternal long and short axis and subcostal four chamber views, which was not noted by the initial interpreting physician when reading the preoperative study.


Parasternal Long Axis View


Parasternal Short Axis View


Subcostal Four Chamber View

**Surgical Course:** Murky fluid and obvious pericarditis discovered on surgical opening of the pericardium. Intraoperative Infectious Disease consultant recommended fluid gram staining which showed marked purulence but no bacteria, so the surgeon proceeded with bypass grafting but elected to not proceed with needed left atrial appendage (LAA) clipping due to concern for infection risk.

**Post-Operative Course:** Patient was continued on empiric antibiotic therapy until pericardial fluid cultures returned showing no growth. He had an uneventful post-operative recovery and was discharged home without incident.

**Follow Up:** Patient recovered as expected without signs of postoperative infection or impaired healing. Certolizumab pegol 400 mg subcutaneously every 4 weeks was added to his treatment regimen with sulfasalazine by his primary rheumatologist.

## UNIQUE ASPECTS OF CASE

After a sweeping review of existing literature, it does not seem that intraoperative discovery of aseptic 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 euvolemic. There exist only a few reports that discuss pyopericardium and pericarditis with RA. The cases published note discovery at the point in which the pericarditis has become constrictive, causing heart failure[4]. Pericardial effusion is a well-documented complication of RA, but less than 10% of RA patients develop pericarditis[5]. A 20-year single-center retrospective review of 33 patients showed purulent pericarditis to be almost always related to infection and often only discovered post-mortem[6].

## OSTEOPATHIC CONSIDERATIONS

Patients with pericardial effusion and pericarditis may benefit from osteopathic approaches to treatment to 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:

- **Biomechanical:** Identify and treat dysfunctions along the T1 to T6 segments and the upper ribs[7].
- **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 C6 and C7 vertebrae and the respiratory diaphragm given their attachments to the pericardium[7,8].
- **Neurological:** 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 occipitoatlantal junction, and cardiac viscerosomatics from T1 to T6[7,8].
- **Metabolic:** Provide counseling to maintain proper exercise and diet to optimize healing and avoid triggers of inflammation[7].
- **Behavioral:** Ensuring adequate management of allostatic load, including environmental stressors, life events, and management of current illness will better facilitate improved outcomes overall[7]

In terms of follow up care, continuing to be followed by an osteopathic physician with ongoing implementation of osteopathic manipulative treatments (OMT) could better facilitate long-term remission of his RA and aid in preventing recurrence of the pyopericardium.

## 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 hyperechoic pericardium, but this nonspecific finding 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 surprise findings intraoperatively. Unfortunately, due to the unexpected nature of his pyopericardium, the 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 aseptic 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 purulent pericarditis is often asymptomatic, it is likely underdiagnosed. Maintaining clinical suspicion during preoperative workup in patients with RA is critical.

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