ACUTE CARDIAC TAMPONADE IN A 58-YEAR-OLD MALE WITH POSTSTREPTOCOCCAL GLOMERULONEPHRITIS

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Abstract

Pericarditis in conjunction with nephritis is an uncommon clinical scenario with a broad differential diagnosis. We present the case of a 58-year-old male who developed nephritis and pericardial effusion with tamponade physiology. In the following, we discuss the differential diagnosis for concomitant nephritis and pericarditis and discuss the work-up performed on our patient. We also review the epidemiology of postinfectious glomerulonephritis in adults and describe previous cases of *Streptococcus pyogenes* pericarditis in the literature.

Case Report

A 58-year-old male with diabetes and chronic kidney disease presented with a history of malaise, fatigue, and sore throat. He also had experienced intermittent nausea, vomiting, and diarrhea over the prior 2 weeks and reported subjective fevers and chills. Physical examination was unremarkable. Basic metabolic panel demonstrated acute kidney injury (AKI).

The patient was administered intravenous saline. A renal ultrasound demonstrated normal anatomy, and *Clostridium difficile* antigen was negative. He was started on ceftriaxone and metronidazole for bacterial intestinal overgrowth. Emesis and diarrhea improved after 24 hours of antibiotics; however, renal function continued to worsen. The patient developed volume overload and ultimately required hemodialysis. Transthoracic echocardiogram showed low normal left ventricular systolic function with a trivial pericardial effusion. Urinalysis revealed proteinuria, hematuria, and pyuria. Urine culture was negative. Complement 3 level was low at 47.6 mg/dL (lower limit 90 mg/dL). Complement 4 level was normal. Both erythrocyte sedimentation rate and C-reactive protein were markedly elevated. Antinuclear antibody, antineutrophil cytoplasmic antibody, anti-double-stranded DNA, anti-Ro, anti-La, and antiglomerular basement membrane antibody were negative. Serum protein electrophoresis and urine protein electrophoresis were negative. Creatinine kinase was within normal limits. Hepatitis B and C antibodies were nonreactive, as were HIV antibodies, rheumatoid factor, and cryoglobulins. Serum IgA was 582 mg/dL (upper limit 400 mg/dL) and IgG was 3190 mg/dL (upper limit 1523 mg/dL). Antistreptolysin O titer was 2484 IU/mL (upper limit of normal 200 IU/mL). The patient was diagnosed with poststreptococcal glomerulonephritis and continued to tolerate dialysis well. Before discharge, the patient developed intractable hiccups. Chest X-ray revealed only resolving pulmonary edema. He was discharged with plans for outpatient chest and abdominal computed tomography (CT) to further evaluate his hiccups.

The following day, he presented to the emergency department with acute dyspnea after routine hemodialysis. He had tachypnea and hypoxia that responded to supplemental oxygen. Systolic blood pressure decreased by 10 mm Hg with inspiration. Chest CT demonstrated a moderate pericardial effusion. A repeat transthoracic echocardiogram (online video) showed a pericardial effusion with right atrial and right ventricular diastolic collapse. The patient underwent urgent pericardiocentesis with removal of 700 mL of sanguineous fluid. Bacterial, fungal, mycobacterial cultures, and cytology were negative. His blood urea nitrogen was 39 mg/dL on re-presentation to the hospital. Nine months later, the patient has had no recurrence of pericardial effusion. He has recovered kidney function and no longer requires hemodialysis.

Discussion

Pericarditis in conjunction with nephritis presents as an interesting clinical picture, leading one to consider a robust list of differential diagnoses that includes autoimmune processes, malignancy, uremia, and infection. We considered a broad differential for our patient and believe the cause for pericarditis and nephritis was infection with *Streptococcus pyogenes*.

Systemic lupus erythematosus (SLE) was considered since approximately 50% of patients will develop nephritis within the first 10 years of their disease and about 25% will experience symptomatic pericarditis.[1,2] Wegener's granulomatosis was also entertained. Although Wegener's is often described as a pulmonary renal syndrome, pericarditis is sometimes observed with this autoimmune disorder as well. Our patient, however, did not meet diagnostic criteria for SLE or Wegener's.[3,4] Patients also should be evaluated for malignancy since adenocarcinoma is a known cause of pericardial effusion and nephritis.[5] In addition, uremia can generate a similar clinical picture. Evaluation of our patient revealed no signs of malignancy. At the time of dialysis initiation, the patient had a known trivial pericardial effusion with no evidence of tamponade. He was regularly receiving dialysis which would have been expected to reduce the size of the effusion had it been uremic in nature. Therefore uremic pericarditis was unlikely.

Our patient had both a clinical history and laboratory findings consistent with *Streptococcus pyogenes* infection. Postinfectious glomerulonephritis (PIGN), although originally considered a disease of childhood, has become more prevalent in adults in the last three decades and is typically caused by a nephritogenic strain of streptococci. Unlike younger patients, affected adults often have

predisposing conditions. In the United States, being immune-compromised, namely from diabetes or malignancy, has been identified as a risk factor. An age of 65 or older has also been associated with increased risk, as has male gender and alcoholism. Although childhood cases of PIGN rarely require hemodialysis, renal dysfunction tends to be more severe in adults. Almost 50% of adult cases require temporary hemodialysis, and up to 33% require permanent hemodialysis. The most common extrarenal manifestation is congestive heart failure.[6,7]

In contrast to PIGN, *Streptococcus pyogenes* is not a common cause of bacterial pericarditis.[8] A literature review revealed eight prior cases of purulent pericarditis caused by *Streptococcus pyogenes* and one case of constrictive pericarditis.[9,10] Cases of myopericarditis have also been reported.[11,12] Only one previous case report of cardiac tamponade secondary to *Streptococcus pyogenes* infection could be found.[13] All reported cases were in children and young adults.

Conclusion

Streptococcus pyogenes nephritis in conjunction with pericarditis is an uncommon diagnosis, particularly in adults. Cases with tamponade appear to be infrequent. We present an interesting case of a 58-year-old male with poststreptococcal glomerulonephritis and streptococcal infection leading to cardiac tamponade. Although gram stain and cultures of pericardial fluid were sterile in the setting of recent antibiotic use, we feel strongly that our patient's effusion was in fact related to *Streptococcus pyogenes*. No other infectious, inflammatory, or malignant sources were identified. Our case demonstrates the challenges of diagnosing this uncommon condition in an unusual patient demographic.

Conflict of Interest Disclosure: The authors have completed and submitted the *Methodist DeBakey Cardiovascular Journal* Conflict of Interest Statement and none were reported.

Keywords: postinfectious glomerulonephritis, pericarditis, cardiac tamponade, acute kidney failure

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