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Cardiac tamponade revealing MPO-ANCA-associated vasculitis

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Introduction: ANCA-associated vasculitis (AAV) is a potentially life-threatening systemic small-vessel disease. The two major ANCA vasculitis subtypes based on the target antigens are proteinase 3 (PR3)-ANCA (associated with granulomatosis with polyangiitis) and myeloperoxidase (MPO)-ANCA (associated with microscopic polyangiitis (MPA) and eosinophilic granulomatosis with polyangiitis). Cardiac involvement occurs in approximately 10–20% of patients with MPA, but cardiac symptoms are an unusual initial presentation, and cardiac tamponade is particularly rare. Recognizing this presentation is important because prompt immunosuppressive treatment can prevent irreversible organ damage.[1,2]

Case report: 50-year-old man with inadequately controlled hypothyroidism on levothyroxine substitution therapy and previously detected nonspecific pulmonary nodules presented with progressive dyspnea and epigastric pain. Laboratory findings showed microcytic anemia, acute kidney injury (creatinine 188 µmol/L), elevated inflammatory markers (C-reactive protein 104 mg/L), active urine sediment, high thyroid-stimulating hormone (23 mIU/L) but with only mildly decreased free T4 (8,31 pmol/L). On admission, the patient was subfebrile, tachycardic, but still normotensive. Echocardiography demonstrated a large pericardial effusion with hemodynamic compromise (right ventricular diastolic collapse, and significant respiratory variation in mitral inflow) (**Figure 1**). Urgent pericardiocentesis was performed, and 1500 mL of serous fluid was drained. Initial treatment consisted of levothyroxine dose adjustment, antibiotics, colchicine, while nonsteroidal anti-inflammatory drugs were avoided given the potential for further kidney injury. Additional evaluation revealed nephrotic-range proteinuria, so a renal biopsy was performed demonstrating diffuse necrotizing glomerulonephritis. Combining clinical, histopathological, and serological findings, the final diagnosis of MPA was confirmed with positive titers for MPO-ANCA. Endoscopic evaluation of the gastrointestinal tract and bronchoscopy did not show significant pathological findings. The patient promptly received induction therapy with methylprednisolone (followed by a prednisone taper) and cyclophosphamide, with rituximab also planned for maintenance therapy. Inflammatory markers rapidly normalized and renal function stabilized. Three months after treatment initiation, MPO-ANCA became negative. At ten-month follow-up, proteinuria had markedly decreased, chest X-ray was normal, and the patient had no recurrent cardiac symptoms with only mild pericardial effusion (**Figure 2**).

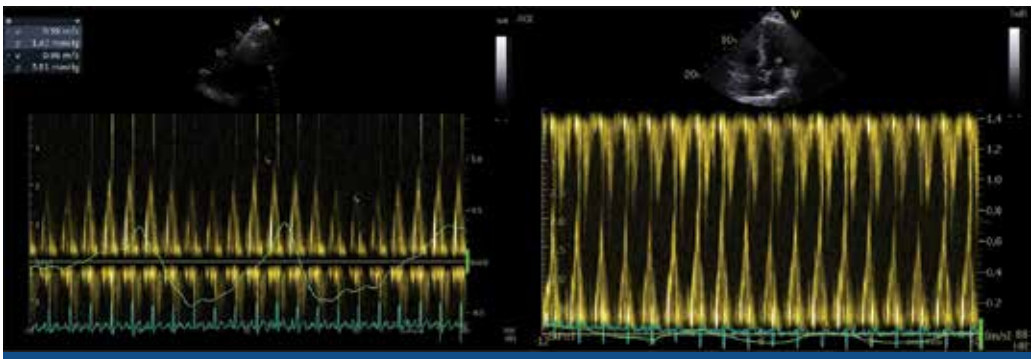


FIGURE 1. Mitral inflow during and after pericardial drainage.

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FIGURE 2. Mild pericardial effusion.

Conclusion: Because renal involvement is common in MPA, urinary abnormalities accompanying pericardial disease may provide a diagnostic clue. Final diagnosis relied on integrating the clinical presentation with systemic inflammation, serology and renal histopathology rather than considering the effusion in isolation or attributing it solely to hypothyroidism. Given the high mortality associated with untreated AAV, early recognition, rapid diagnostic evaluation, a multidisciplinary approach, and timely immunosuppressive treatment initiation are essential for improving outcomes.[2]

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