Rare Cases of Aortic Dissection Presenting with Acute Congestive Heart Failure: Implications for Surgical Timing

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Introduction

Stanford type A aortic dissection (AAD) is a surgical emergency with mortality rising by 1–2% per hour without intervention. While typically presenting with abrupt chest pain or malperfusion, atypical cases may manifest as acute congestive heart failure (CHF), often due to acute aortic regurgitation or coronary malperfusion. Such presentations are frequently mistaken for acute coronary syndrome (ACS), delaying diagnosis and exposing patients to harmful therapies such as dual antiplatelet agents. Malperfusion significantly worsens outcomes, increasing operative mortality from 5–6% in uncomplicated cases to 20–40% with multi-organ involvement. Early recognition, rapid imaging, and prompt surgery remain critical for survival.

Case presentation

A 53-year-old male presented to a peripheral hospital with acute dyspnea and severe pulmonary edema. He was hemodynamically unstable and initially treated for acute coronary syndrome (ACS) with decompensated heart failure. Dual antiplatelet therapy was administered, and urgent coronary angiography showed normal coronary arteries. Persistent chest pain and respiratory distress prompted CT angiography, which revealed a Stanford type A aortic dissection involving the ascending aorta and arch. The patient was urgently transferred to our tertiary center.

On arrival, he remained dyspneic with cardiomegaly and pulmonary edema on chest radiography. Echocardiography demonstrated concentric left ventricular hypertrophy with grade III diastolic dysfunction, elevated left atrial pressure, and a structurally normal trileaflet aortic valve with mild regurgitation.

Emergency hemiarch replacement using a Dacron graft was performed under moderate hypothermic circulatory arrest and selective cerebral perfusion. A large intimal tear was identified at the aortic arch, and the aortic valve was preserved. Postoperative recovery was uneventful and the patient was discharged in stable condition.

Discussion

AAD presenting with acute CHF is rare but clinically significant. The mechanisms include acute aortic regurgitation from annular distortion or flap prolapse, and less commonly, coronary malperfusion. In this patient, pulmonary edema and hemodynamic compromise dominated the clinical picture, leading to misdiagnosis as ACS. The administration of DAPT posed additional surgical risk, reflecting the consequences of diagnostic delay.

Coronary angiography remains a common initial investigation in suspected ACS, but in patients with pulmonary edema, chest pain, or unexplained hemodynamic instability, rapid echocardiography or CT should be considered to exclude AAD.

Surgical repair remains the definitive treatment. Hemiarch replacement under circulatory arrest is an established approach, with outcomes highly dependent on preoperative condition and timeliness of intervention. Patients presenting with CHF or requiring mechanical ventilation have been shown to carry higher perioperative mortality.

This case highlights the importance of maintaining diagnostic suspicion for AAD in any patient presenting with acute CHF and pulmonary edema, particularly when coronary anatomy is normal.

Conclusion

Stanford type A aortic dissection can rarely present with acute CHF, mimicking ACS and delaying diagnosis. In such patients, early imaging is crucial. Emergent surgical repair remains the only curative therapy. Prompt recognition and immediate surgical management are key to survival.

Keywords: Aortic dissection, Stanford type A, Congestive heart failure, Pulmonary edema, Hemiarch replacement