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Dissecting Complexity : Pneumonia and Failure

Chronic Aortic Dissection Complicated by Necrotizing Pneumonia and Heart Failure: A Multisystem Challenge

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Introduction

Aortic dissection, particularly Stanford Type A, is a life-threatening condition typically requiring urgent surgical repair. However, chronic presentations with stable anatomy may be managed conservatively in select cases. When accompanied by cardiopulmonary comorbidities and systemic infection, management becomes particularly challenging. This report discusses a diagnostically and therapeutically complex case, emphasizing the role of imaging, vigilance for complications, and coordinated care.

Case Presentation

A 44-year-old non-smoking male with a background of bronchial asthma, severe obstructive sleep apnea (OSA), hypertension, and dyslipidemia presented with a chronic cough since September 2024. The cough was initially productive of whitish sputum and later progressed to hemoptysis. Over the next two weeks, he developed worsening dyspnea, orthopnea, pleuritic chest pain, and reduced effort tolerance, consistent with NYHA Class II heart failure.

Upon re-presentation, he was febrile (38.7°C), tachycardic (101 bpm), and mildly hypertensive (150/68 mmHg), with oxygen saturation of 98% on 3L/min nasal cannula. Lung ultrasound revealed bilateral B-lines suggestive of pulmonary congestion. Bedside echocardiography showed moderate left ventricular systolic dysfunction (EF ~40%) without aortic root dilation. Chest radiography showed cardiomegaly and bilateral pulmonary infiltrates. Laboratory findings included leukocytosis (WBC 13 ×10⁹/L), elevated CRP (4.2 mg/L), troponin I (41 ng/L), and creatinine 144.8 µmol/L. Arterial blood gases revealed primary respiratory alkalosis. He was admitted with suspected decompensated heart failure, partially treated pneumonia, hepatic congestion, and AKI.

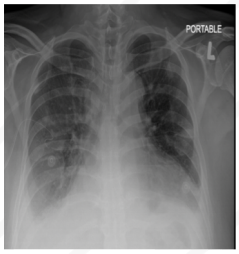

Figure 1 : CXR

During hospitalization, intermittent fever prompted antibiotic escalation from IV piperacillin-tazobactam to IV ceftriaxone, then IV ceftazidime. Inflammatory markers rose (CRP 7.48 mg/L, WBC 17.4 ×10⁹/L). Blood cultures grew *Staphylococcus lugdunensis*. Repeat echocardiography showed EF 42% with global hypokinesia and moderate-to-severe aortic regurgitation. CT pulmonary angiography excluded pulmonary embolism but revealed an ascending aorta measuring 4.6 cm, prompting a CT aortic angiogram.

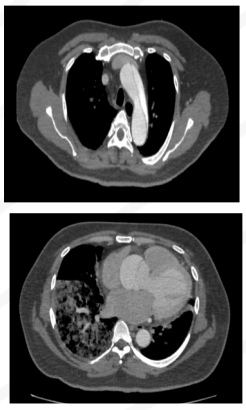

Figure 2, 3 : CTA shows Type A dissection with ascending aortic aneurysm.

A chronic Stanford Type A (DeBakey Type I) aortic dissection was identified, extending from the aortic root into the brachiocephalic trunk, carotid, and subclavian arteries, and down to T9–T10. Follow-up CTAs showed fusiform ascending aortic dilation (up to 5.5 cm), a stable intimal flap, and no active leak. Concurrent imaging revealed evolving necrotizing pneumonia, worsening right pleural effusion, and lung collapse.

Conservative management was recommended due to dissection chronicity and comorbidities. He received IV antibiotics, pleural drainage via pigtail catheter, and sustained low-efficiency dialysis (SLEDD) for AKI. Heart failure was managed with beta-blockers, diuretics, and fluid restriction. He was discharged on home oxygen with multidisciplinary follow-up in cardiology, respiratory, nephrology, and infectious disease. Serial imaging showed no dissection progression.

Discussion

This case exemplifies the complexity of managing chronic Stanford Type A aortic dissection complicated by necrotizing pneumonia, decompensated heart failure, and Staphylococcus lugdunensis bacteremia. Management involved escalation of antimicrobial therapy, judicious use of diuretics and beta-blockers, and temporary deferral of SGLT2 inhibitors. Acute kidney injury necessitated renal replacement therapy. Conservative monitoring of aortic pathology via serial CT angiography and multidisciplinary coordination were pivotal in achieving clinical stabilization and optimizing patient outcomes.

Conclusion

This case underscores the interplay of infection, cardiovascular, and renal dysfunction, highlighting the need for timely intervention, individualized therapy, and multidisciplinary care to optimize outcomes in complex patients with overlapping comorbidities.

References

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