Silent danger at the cavoatrial junction: Fatal right atrial thrombus from a chemoport catheter

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Introduction/Objectives
Implantable chemoports are essential in oncology for durable venous access, but their long-term use carries risks beyond infection or mechanical dysfunction. Catheter-related right atrial thrombosis (CRAT) is a rare yet potentially fatal complication, often underestimated until catastrophic events occur. The pathogenesis is multifactorial, involving mechanical irritation of the atrial endocardium by the catheter tip, local turbulence and stasis, and the hypercoagulable milieu of cancer, renal failure, and sepsis. Recognition is critical, as CRAT carries significant morbidity and mortality.

Materials and Methods
A 44-year-old woman with end-stage renal failure on peritoneal dialysis and Stage III breast carcinoma in remission, previously treated with surgery, chemotherapy, radiotherapy, and chemoport insertion, presented with pneumonia and sepsis. Echocardiography revealed a right atrial mass measuring 2.0 × 0.5 cm, with differential diagnoses including thrombus, infective endocarditis, and myxoma. After optimizing anemia, renal parameters, and prior bacteraemia, she underwent sternotomy, excision of the mass, and chemoport removal.

Intraoperatively, the anterior right atrial wall was markedly thickened with myxomatous-appearing tissue. Residual yellowish tissue was excised, although no clot was seen attached to the catheter, which was removed intact. Histopathology confirmed fibrinous material and granulation tissue, supporting a diagnosis of catheter-related thrombus with chronic endocardial injury.

Postoperatively, she initially stabilized but developed coagulopathy, diffuse oozing, and progressive multiorgan failure. Despite massive transfusion, two surgical re-explorations, continuous renal replacement therapy, and escalating inotropes, she deteriorated and succumbed on postoperative day 5 from severe septic shock and multiorgan failure.

Results/Discussion
This case highlights the insidious nature of CRAT. Although frequently silent, it can precipitate sepsis, pulmonary embolism, or hemodynamic collapse. The mechanism is largely preventable: improper catheter tip placement within the atrium predisposes to persistent endocardial trauma and thrombus formation. Guidelines recommend positioning the tip at the cavoatrial junction, reducing atrial wall contact while maintaining central access.

In this patient, several high-risk factors converged: end-stage renal failure requiring dialysis, previous malignancy, immunosuppression, and recent bacteraemia. These comorbidities created a pro-thrombotic, pro-inflammatory state, magnifying the effects of catheter irritation. Surgical excision was chosen due to diagnostic uncertainty and embolic risk. However, her postoperative decline illustrates the vulnerability of such patients, where technically successful surgery may be overshadowed by systemic infection, coagulopathy, and organ dysfunction.

CRAT remains under-recognized, with no standardized management. Treatment strategies range from anticoagulation and thrombolysis to surgical excision, guided by thrombus size, mobility, comorbidities, and patient stability. Multidisciplinary input from cardiology, oncology, nephrology, and cardiothoracic surgery is essential in tailoring care. Prevention remains paramount, with correct catheter placement and vigilant surveillance in high-risk groups.

Conclusion
CRAT is a rare but devastating complication of long-term chemoport use. This case underscores the importance of correct catheter tip placement at the cavoatrial junction to minimize endocardial trauma, alongside vigilant monitoring in patients with risk factors such as renal failure, sepsis, and malignancy. Early recognition and multidisciplinary management are critical, yet outcomes may remain poor in fragile patients. Greater awareness and proactive prevention are essential to reduce morbidity and mortality in this vulnerable population.