Case Report

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Case Report

Trousseau's Syndrome and Marantic Endocarditis in a Patient with Pulmonary Adenocarcinoma: A Case Report and Review of Literature

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Abstract

Background: Trousseau's syndrome, characterized by recurrent thromboembolic events and non-bacterial thrombotic endocarditis, represents a severe paraneoplastic condition associated with poor prognosis in cancer patients**. Methods:** We describe a case of a 63-year-old male presenting with ischemic stroke and mitral valve marantic endocarditis, ultimately diagnosed with pulmonary adenocarcinoma. The case description is followed by a brief review of the current literature on the condition. **Discussion and Conclusion:** This case highlights the complexity of diagnosing and managing Trousseau's syndrome. Early recognition, appropriate anticoagulation strategies, and the need for multidisciplinary management are crucial to improve the outcomes and the quality of life of cancer patients.

Keywords: Trousseau's syndrome; venous thromboembolism; marantic endocarditis; cancer

1. Introduction

Thromboembolic events represent the second leading cause of death among patients with malignant neoplasms [1] and may even constitute the first clinical manifestation of an underlying cancer [2]. The combination of recurrent venous and/or arterial thromboses in association with non-bacterial (marantic) endocarditis is known as Trousseau's syndrome. This condition was first described in 1865 by Armand Trousseau [3], who self-diagnosed it two years later, shortly before his death due to gastric cancer [4]. Trousseau's syndrome is associated with poor prognosis and reduced quality of life in cancer patients [5]. However, specific histological subtypes and targeted oncologic therapies may influence clinical outcomes [5] making early recognition and diagnosis essential. Here, we report the case of a 63-year-old man diagnosed with Trousseau's syndrome, followed by a brief review of the current literature on this condition.

2. Ethical Statement and Patient Consent

All procedures at our institution are performed following the ethical standards set by institutional and national research committees and the 1975 Helsinki Declaration and its amendments. Our institution does not require ethical approval for reporting individual cases or case series. The patient's consent was obtained for publication of this report and images.

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3. Case Report

In March 2022, a 63-year-old Caucasian male patient presented to our Emergency Department (ED) with dysphagia and dysarthria, which had begun approximately seven hours earlier. Upon arrival, he was alert, aphasic, with a right-sided facial nerve deficit, and intact Mingazzini signs. The National Institutes of health Stroke Scale (NIHSS) score was 6, while the modified Rankin Scale score was 3. Cardiac auscultation revealed a regular rhythm with a 3/6 systolic murmur. The remainder of the physical examination was unremarkable.

At admission, the patient's body weight was 75 kg, with a body mass index (BMI) of 26 kg/m^2. Laboratory tests showed a white blood cell (WBC) count of 9,670/μL (reference range: 4,000–10,000/μL), hemoglobin of 11 g/dL (13–17 g/dL), platelet count of 170,000/μL (150,000–400,000/μL), markedly elevated D-dimer at 21,268 EEU (0–500 EEU), serum creatinine of 0.76 mg/dL (0.6–1.2 mg/dL), and a negative C-reactive protein (CRP) test (<5 mg/L). Autoimmune markers including antinuclear antibodies (ANA), extractable nuclear antigens (ENA), anticardiolipin antibodies, and β2-microglobulin were negative.

Past medical history included hypertension, hyperuricemia, and follicular lymphoma, initially treated with the CHOP + bendamustine regimen and later with Rituximab. One month earlier, enlarged lymph nodes with poorly defined margins raised suspicion for disease recurrence, but treatment was deferred due to the absence of GELF criteria[6]. Two weeks before the presentation, the patient developed left femoropopliteal deep vein thrombosis and started edoxaban 60 mg daily. His other chronic medications included ramipril and allopurinol. The patient was also a heavy smoker (35 pack-years).

A cranial CT scan and CT angiography revealed ischemic lesions in the left post-central insular gyrus and the left occipital gyrus, along with a complete occlusion of the left middle cerebral artery (*Figure 1*). No indication for endovascular treatment was found. Follow-up cranial CT scans excluded hemorrhagic infarctions. Anticoagulation with edoxaban was continued. No arrhythmias were detected on Holter ECG.

Transthoracic echocardiography (TTE) showed normal left ventricular systolic function with severe mitral regurgitation but no evidence of endocavitary masses. A transesophageal echocardiogram (TOE) ruled out a patent foramen ovale and identified endocarditis of the mitral valve, with distal thickening of both mitral leaflets (maximum 3 mm) resulting in multiple, predominantly eccentric, regurgitant jets, causing severe mitral regurgitation (*Figures 2*), with no indications for urgent/emergency cardiac surgery. Considering findings suggestive of endocarditis and coexisting aspiration pneumonia, empirical antibiotic therapy was initiated with ceftriaxone, vancomycin, and clarithromycin. However, the patient remained afebrile, with negative blood cultures, WBC counts 6390 /μL and stable CRP levels between 10-12 mg/L (likely related to the pneumonia). Serial procalcitonin (PCT) levels were always negative.

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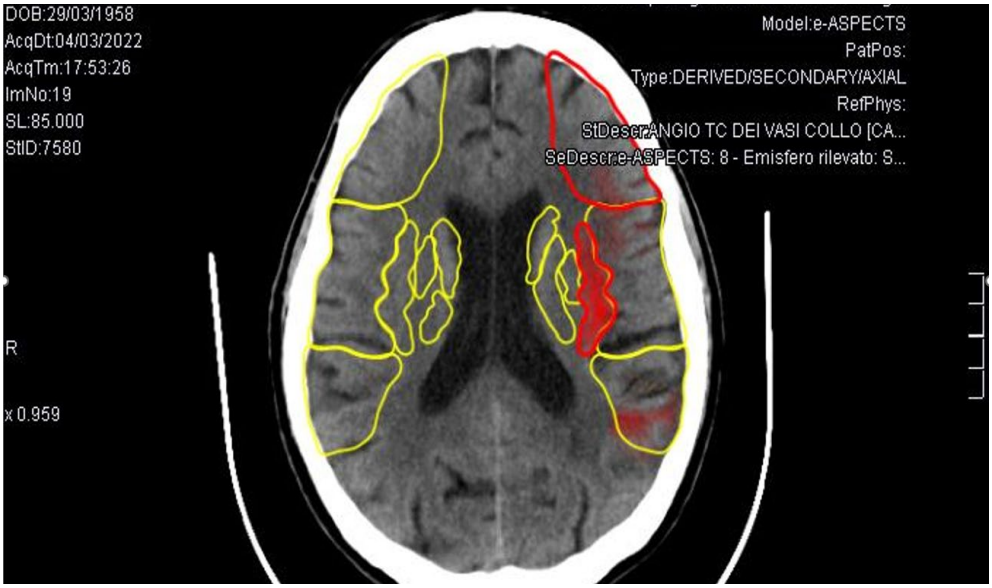

Figure 1. Cranial TC scan: ischemic area in the left post-central insular gyrus and the left occipital gyrus.

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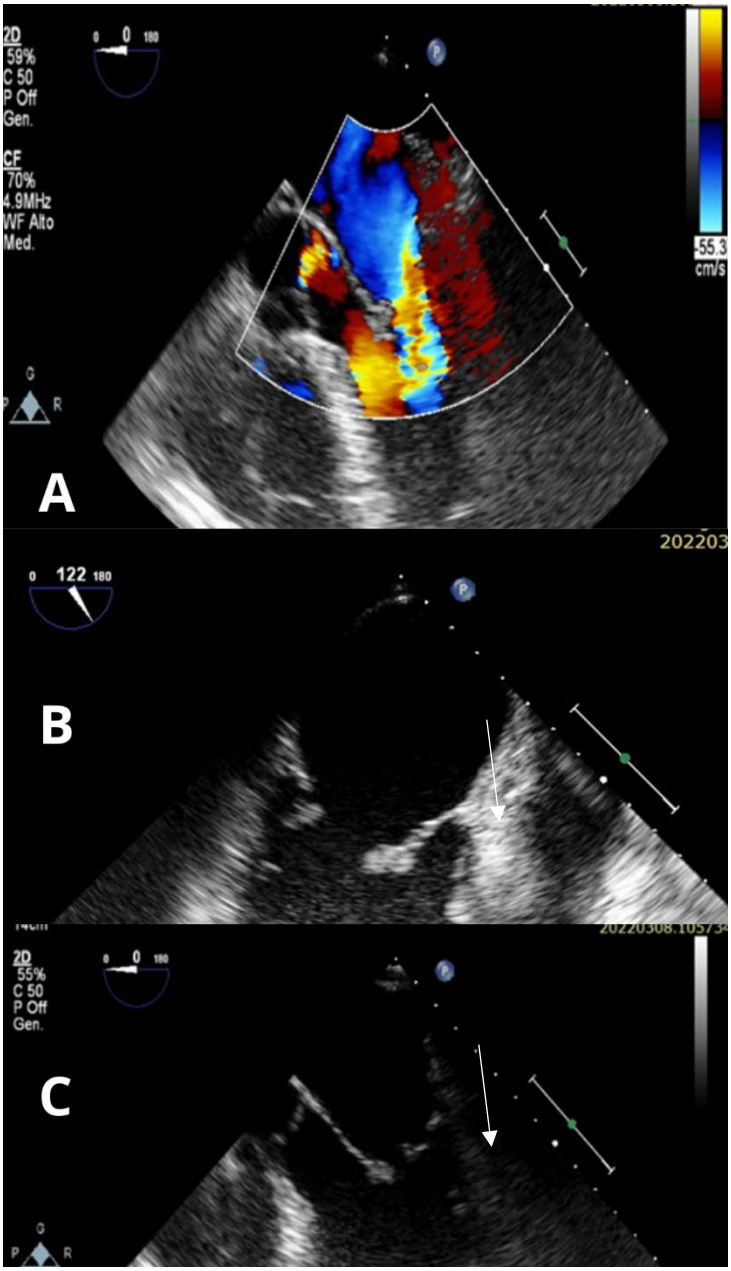

Figure 2. A, B and C): TOE: Distal thickening of both mitral leaflets (white arrows), with a maximum thickness of three millimeters, impairing proper systolic coaptation and resulting in multiple, predominantly eccentric, regurgitant jets, overall consistent with severe mitral regurgitation. Findings suggestive of mitral valve endocarditis.

Therefore, given the negative blood cultures and absence of fever or other minor criteria, the patient did not meet the Duke's criteria for infective endocarditis[7]. Suspecting lymphoma recurrence, a CT scan of the neck, chest, and abdomen was performed. The examination revealed ischemic-infarct areas in the spleen and a tissue mass with contrast enhancement in the left hilar para-aortic region causing narrowing of the bronchial structures (*Figure 3*). Consequently, bronchoscopy was performed with biopsy of the left upper lobe bronchus.

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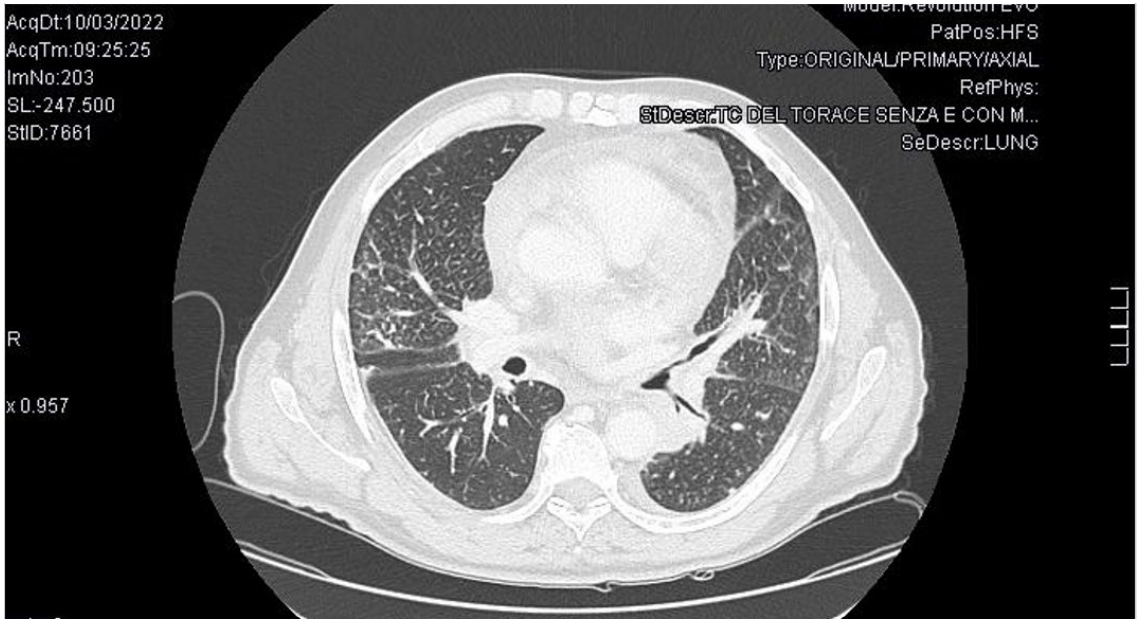

Figure 3. Chest CT scan: a tissue mass in the left hilar para-aortic region causing narrowing of the bronchial structures.

Soon after, the patient developed thrombosis of the proximal left axillary artery, right axillary vein at a PICC (Peripherally Inserted Central Catheters) line site. Edoxaban was replaced by LMWH at an anticoagulant dosage. The pulmonary biopsy report revealed the presence of pulmonary adenocarcinoma. The patient passed away two months later due to progression of malignancy.

4. Discussion

According to the ESC Cardioncology guidelines [1] malignancies increase the risk of venous thromboembolism (VTE) by fivefold and the risk of arterial thromboembolism by twofold. In the treatment and secondary prevention of venous thromboembolism in cancer patients, the guidelines recommend LMWH. LMWH is generally preferred to unfractioned heparin (UFH) because LMWH does not require blood test monitoring and hospitalization. LMWH was also more effective than warfarin in reducing the risk of VTE without increasing the risk of bleeding in patients with high thrombotic risk [8,9]. Direct acting oral anticoagulants (DOACs) such as edoxaban , rivaroxaban or apixaban [10–12] are potential alternatives (*Table 1*).

In head-to-head trials with LMWH, all three DOACs were non-inferior in treating venous thromboembolism; however, only apixaban, in the CARAVAGGIO trial, did not increase the bleeding risk. Consequently, the 2023 update of the ASCO guidelines [13] included apixaban amog the preferred DOACs. In the API-CAT trial [14] reduced-dose apixaban (2.5 mg twice daily) was found to be non-inferior to the full-dose regimen (5 mg twice daily) in preventing venous thromboembolism in cancer patients who had already completed 6 months of anticoagulant therapy for VTE/PE (pulmonary embolism). Moreover, the reduced dose was associated with a lower rate of clinically relevant bleeding compared to the full-dose regimen.

Nevertheless, DOACs are contraindicated in patients with one of the following bleeding factors: unoperated gastrointestinal (GI) or genitourinary (GU) malignancies, history of recent bleeding or within 7 days of major surgery, significant thrombocytopaenia (platelet count < 50 000/μL), severe renal dysfunction (creatinine clearance (CrCl <15 mL/min), or GI comorbidities. The minimal duration of anticoagulation is 6 months [1,15]. However, patients with cancer are also at high risk of bleeding during anticoagulant treatment. For this reason, a periodic assessment of the risk/benefit ratio should be performed. The most famous and well validated score to calculate VTE in cancer

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patients is the Khorana score [16] while existing risk scores for bleeding perform poorly after cancer associated thromboses (CAT) [17].

It is mandatory to highlight that the patient described in the report came to our attention before the publication of the 2022 ESC cardio-oncology guidelines and the 2023 update of the ASCO guidelines. He developed episodes of both venous and arterial thrombotic non-infective (marantic) endocarditis with multiple embolic events involving the brain and spleen. Recurrent thrombosis with no other explanation, associated with marantic endocarditis, in cancer patient, is known as Trousseau's syndrome [4]. Although neither the ASCO guidelines nor the ESC cardio-oncology guidelines specifically mention it, Trousseau's syndrome is associated with reduced quality of life and worsened prognosis [5]. The overall incidence in hospitalized cancer patients was 8.0 cases per 1000 person-years (p-y), with the highest incidence in the first year after cancer diagnosis (15.0 cases per 1000 p-y), decreasing to 6.3 cases per 1000 p-y in the second year, and 4.2 cases per 1000 p-y thereafter [18]. A study by Sørensen and coworkers found that one-year survival rate for the cancer group with VTE was 12%, compared with 36% in the control group without VTE [19].

This paraneoplastic syndrome involves hypercoagulability related to patient, cancer and treatment (*Table 2*). Patient-related factors include age, inactivity, and cardiovascular comorbidities. Cancer-related factors include tissue factor (TF), plasminogen activator inhibitor (PAI-1), mucins, cytokines, and hypoxia [4]. TF directly induces the conversion of factor VII to factor VIIa, resulting in the constitutive activation of the coagulation cascade. It has been reported that, in addition to coagulation, TF is also associated with cancer metastasis and angiogenesis [20]. Moreover, the sialic acid moieties of mucin from adenocarcinomas cause a nonenzymatic activation of factor X. All in all, hypoxia (decreased oxygenation) could increase the expression of genes that facilitate coagulation, including TF and PAI-1 [4]. PAI-1 inhibits the activation of plasminogen, a key step in the dissolution of blood clots. On the other hand, about treatment related factors, chemotherapeutic agents such as platinum compounds, hormonal agents, tamoxifen, growth factors (granulocyte colony-stimulating factor and erythropoiesis-stimulating agents) and antiangiogenic agents increase the risk of thrombosis [20]. Chemotherapy itself, independently of the underlying pathophysiology of the neoplastic process, induces a hypercoagulable state by also acting on TF and PAI-1. Indeed, Wrzeszcz et al. demonstrated that adjuvant therapy in patients with invasive breast cancer significantly increased plasma concentrations of TF and PAI-1 [21]. However, when anticoagulant therapy is added, the hemostatic balance may shift toward bleeding risk. In fact, several studies have shown that the concomitant administration of DOACs and chemotherapeutic agents increases the risk of bleeding, primarily due to pharmacokinetic interactions involving CYP3A4 and P-glycoprotein (P-gp) inhibitors or inducers. The tyrosine kinase inhibitors (TKIs) represent the most clinically significant class of agents in terms of drug–drug interactions (DDIs) with DOACs, as they are potent inhibitors of P-gp [22].

Nowadays CVCs (Central Venous Catheter) are gaining a pivotal role for the long-term administration of anticancer drugs and blood sampling, but they are associated to an increased thrombotic risk [23], as illustrated by the case presented.

Several types of cancer are associated with the Trousseau's syndrome. Of these, lung cancer is most frequently associated with malignancy-related ischaemic stroke [24]. Although the diagnosis is generally associated with a worse prognosis in such patients, early recognition—particularly with regard to histological subtypes—may improve clinical outcomes. In a recent study, Yoshimine et al. [5] observed that patients with more favourable prognoses exhibited non-adenocarcinoma histotypes, a high frequency of EGFR mutations, and were more likely to receive immune checkpoint inhibitor (ICI) therapy. Based on these observations, the authors propose that when Trousseau's syndrome develops in patients with lung cancer, continuous heparin therapy should be initiated, and genetic testing along with PD-L1 immunostaining should be promptly performed to guide appropriate treatment. However, this study has several limitations, including its monocentric design and limited sample size. Therefore, further research is warranted to validate these findings.

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Additionally, since our patient did not meet the Duke's criteria and had an active tumor, the endocarditis seen on TOE is categorized as marantic endocarditis [7], t ypically linked to paraneoplastic hypercoagulability from solid tumors, especially lung, gastrointestinal, and pancreatic cancers. This form of endocarditis is often underestimated on TTE but should be suspected in the presence of Trousseau's syndrome [25]. The mitral valve is the most frequently affected. Its involvement is associated with a high rate of clinical complications and mortality [26]. The ESC guidelines recommend LMWH as the first-line treatment [7] in marantic endocarditis. Valid alternatives may include vitamin K antagonists (VKA) or UFH. There are no data to support the use of direct oral anticoagulants in non-bacterial thrombotic endocarditis (NBTE) [27]. The role of surgery is controversial, but it may be considered in select cases with significant persistent valvular dysfunction [28]. ntibiotics are not indicated.

Table 1. Anticoagulation Acute Strategies in Cancer Patients with Thromboembolism [1,13,15].

Therapy	Indications	Advantages	Limitations/Contraindications
Low Molecular Weight Heparin (LMWH)	First-line for treatment and prevention of VTE in cancer patients.	Proven efficacy, reduced VTE vs warfarin, easy dosing	Injection route, bleeding risk, impaired renal function.
Direct Oral Anticoagulants (DOACs) (edoxaban, rivaroxaban, apixaban)	Alternative to LMWH in selected patients.	Oral administration, non-inferior efficacy. Apixaban don not increased bleeding risk.	Avoid in unresected GI/GU cancers, CrCl <15, platelets <50k, recent surgery, bleeding risk
Vitamin K Antagonists (VKAs)	Alternative if DOACs/LMWH not suitable	Oral, long experience	Drug–food interactions, INR monitoring required, less preferred
Unfractionated Heparin (UFH)	Hospitalized patients, rapid reversal needed	Short half-life, reversible	Requires monitoring (aPTT), IV route
Anticoagulation Duration	≥6 months recommended (individualized)	Reduces recurrence	Reassess bleeding periodically, especially in advanced cancer

Table 2. Cancer-Related Thromboembolic Risk Factor [4,18].

Category	Risk Factors
Patient-related	Older age, immobility, comorbidities (e.g., hypertension), history of thrombosis
Tumor-related	Histological type (especially adenocarcinoma), tumor burden, metastasis
Biological mediators	Tissue factor (TF), mucins, PAI-1, cytokines, hypoxia
Treatment-related	Chemotherapy (e.g., platinum compounds), hormonal therapy, antiangiogenics
Drug interactions	DOAC metabolism affected by CYP3A4/P-gp inhibitors (e.g., tyrosine kinase inhibitors)
Procedural	Central venous catheters (CVCs), recent surgery

5. Conclusions

Our case highlights the severity of Trousseau's syndrome and marantic endocarditis, conditions that should be suspected in cancer patients. Early diagnosis and appropriate anticoagulation therapy can significantly reduce mortality and morbidity. However, the risk of bleeding must be weighed,

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especially in advanced cancer patients, necessitating a multidisciplinary approach and a stretched follow-up involving an expert in thrombosis and hemostasis.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Institutional Review Board (or Ethics Committee) of Mayo Clinic (IRB#22-004000 2024-04-20) for studies involving humans.

Informed Consent Statement: Not applicable as this study involves a retrospective review of previously collected clinical data and all the reviewed data were de-identified to protect patient privacy.

Data Availability Statement: The original contributions presented in this study are included in the article. Further inquiries can be directed to the corresponding author.

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Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

ANA:	antinuclear antibodies
ASCO:	American Society of Clinical Oncology
CAT:	Cancer associated thromboses
CRP:	C-reactive protein
CVC:	Central Venous Catheter
DDIs:	drug–drug interactions
DOAC:	Direct oral anticoagulant
ED:	Emergency department
ENA:	extractable nuclear antigens
ESC:	European Society of Cardiology
EEU:	Enzyme-Linked Immunosorbent Assay Equivalent Units
GI:	gastrointestinal
GU:	genitourinary
ICI:	immune checkpoint inhibitor
LMWH:	low-molecular-weight heparin
NBTE:	non-bacterial thrombotic endocarditis
NIHSS:	National Institutes of health Stroke Scale
PAI-1:	plasminogen activator inhibitor
PE:	Pulmonary embolism
PICC:	peripherally inserted central catheter
P-gp:	P-glycoprotein
TF:	tissue factor
TKIs:	Tyrosine kinase inhibitors
TOE:	transesophageal echocardiogram
TTE:	Transthoracic echocardiography
UFH:	unfractioned heparin
VKA:	vitamin K antagonists
VTE:	venous thromboembolism
WBC:	white blood cell

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