# **REVIEW ARTICLE**

# Iatrogenic Right Atrial Thrombus Complicated by Pulmonary Embolism: Management and Outcomes

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ARTICLE HISTORY

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DOI: 10.2174/1573403X16999201124201632 **Abstract:** Right atrial thrombus can originate from distal venous sources or can be iatrogenic, secondary to the placement of central venous catheters, atrial devices, or surgeries. One of the most common complications of Central Venous Catheters (CVCs) is thromboembolism, which can be either fixed to the right atrium or can be free-floating. Device-related Right Atrial Thrombosis (RAT) can result in catheter occlusion, vascular occlusion, infection, and pulmonary embolism. The true incidence of these complications is unknown because the diagnosis may not be considered in asymptomatic patients, and it might be missed by Transthoracic Echocardiography (TTE). In this literature review, we discuss iatrogenic etiologies of RAT that is complicated by pulmonary embolism. We highlight the importance of maintaining a high index of suspicion of iatrogenic RAT, possible complications, and its management.

**Keywords:** Central venous catheters, right atrial thrombus, thromboembolism, pulmonary embolism, iatrogenic, transthoracic echocardiography.

# **1. INTRODUCTION**

Acute Pulmonary Embolism (PE) leads to 50,000 deaths and contributes to 10% of the hospital-associated mortality every year in the United States [1, 2]. The source of PE can be the right atrium. The incidence of RAT is not wel-1-defined because many asymptomatic patients are likely to be undiagnosed [3]. RAT can be classified into type A and type B categories. Type A thrombi are free-floating, and type B thrombi are fixed to the right atrial wall. RAT can originate from a distal site within the body or can be iatrogenic due to pacemaker leads, closure devices, and central venous catheters [4, 5]. The incidence of asymptomatic CVC-related thrombosis ranges from 5-62%, while the incidence of symptomatic CVC-related thrombosis is 28% [6]. In addition, RAT can be complicated by the development of Pulmonary Embolism (PE), which can occur in 4-6% of CVC-related thrombosis cases [7]. Overall, PE occurs as a complication in 36% of the patients with RAT [8].

### 2. METHODS

We searched PubMed, Ovid Medline, and Google Scholar using the following search terms: iatrogenic, device-related, central venous catheter, right atrial thrombus, pulmonary embolism. We conducted a systematic review of the literature for iatrogenic RAT cases associated with PE and its management.

# **3. CASE PRESENTATION**

The patient is a 42-year-old female who was presented with mild abdominal pain and syncopal episodes. She had a past medical history of type 1 diabetes mellitus, end-stage renal disease on hemodialysis *i.e.*, on Monday, Wednesday, Friday via right femoral vein using permacath, morbid obesity, hypertension, hyperlipidemia, history of nephrolithiasis, peripheral vascular disease, and pseudotumor cerebri. She also had a recent placement of an elective Hemodialysis Reliable Outflow (HeRO) graft, which was complicated by steal syndrome, requiring graft ligation and revision to loop graft. The night prior to the presentation, the patient went to sleep with no prodromal signs or symptoms. She woke up the following morning and underwent dialysis. Towards the end of the dialysis session, she developed palpitations, lightheadedness, and tunnel vision that led to multiple syncopal episodes. She had one syncopal episode that lasted 2 minutes without sustaining head trauma. There was no witnessed seizure activity and no post-syncopal confusion.

In the ED, vital signs were BP 127/86, heart rate 92, afebrile, and saturating 97% on 2 L  $O_2$  via nasal cannula. Lab work was remarkable for an anion gap metabolic acidosis with bicarbonate level of 18, beta-hydroxybutyrate of 1.0, BUN 26, creatinine 4.6, ALP 115, ALT 6, total bilirubin 0.4, lactate level 1.5, and initial troponin 0.04. EKG showed normal sinus rhythm with new T wave inversions in the anterior leads and poor R wave progression (Fig. 1). Chest x-ray showed no cardiomegaly and no pulmonary infiltrations. The patient was found to be in DKA and was treated accordingly. On the following day, the patient was noted to be hy-

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potensive with systolic blood pressure ranging from 60 to 70 and the bradycardial heart rate ranging from 40 to 50, requiring a transfer to the ICU, three 250 cc boluses of NaCl, and initiation of a dopamine drip. She was also started on midodrine 10mg PO TID. Her home carvedilol was held. Lab work in the ICU revealed normal CBC, hyperkalemia with a potassium level of 6.2, anion gap metabolic acidosis with a bicarbonate of 14, and lactate level 4.8. Stat EKG showed new T-wave inversions in inferior leads with no electrocardiographic signs of hyperkalemia. The patient was noted to be persistently bradycardic in the 40s with systolic blood pressure in the 60s. She was given another bolus of normal saline and started on norepinephrine. ABG showed pH 7.29,  $pCO_2$  32,  $pO_2$  72, and bicarbonate level 15. The syndrome of bradycardia, renal failure, AV nodal blockade, cardiogenic shock, and hyperkalemia (BRASH) was diagnosed. She underwent echocardiography, which showed a markedly dilated right ventricle. Her recurrent syncopal events warranted CT angiography of the chest, which revealed an extensive clot burden in the distal Superior Vena Cava (SVC) surrounding the HeRO catheter, extending into the right atrium as well as bilateral pulmonary artery emboli with extension to the distal branches (Fig. 2). The clot burden was also visualized on Trans-Esophageal Echocardiography (TEE) (Fig. 3).

She was started on tPA. During tPA administration, she decompensated to Pulseless Electrical Activity (PEA). Cardiopulmonary resuscitation resulted in the return of spontaneous circulation; however, she continued to have 5-6 rounds of PEA arrest over 45 minutes. Bedside echocardiogram revealed severe Right Ventricular (RV) dysfunction and dilatation. Since the patient was a poor vascular candi-

date, she was started on a heparin drip and underwent insertion of VA-ECMO by cardiothoracic surgery. ECMO served as a temporizing measure for the recovery of RV function as heparin was given to reduce the clot burden. She was also Venovenous Hemodialysis started on Continuous (CVVHD). Due to the risk of persistent emboli to the lungs exacerbating RV strain, the decision was made to undergo sternotomy and right atrial thrombectomy with the removal of the HeRO catheter. However, intraoperative epiaortic ultrasound and TEE did not reveal a thrombus in the main Pulmonary Artery (PA): left PA, or right PA. Therefore, she did not undergo a pulmonary embolectomy. Upon completion of the surgery, her chest remained open because of coagulopathy. She had a wound vac connected to her chest and was given cefazolin 2 grams for 8 hours until the closure of the chest two days later. At the time of chest closure, RV function had improved, the ECMO was weaned, and she was decannulated. Two days later, she developed signs of right limb ischemia with rising CK and absent pulses. She was brought back to the operating room for right femoral artery patch angioplasty, and four-compartment fasciotomies after angiography showed a dissection flap and thrombus causing occlusion. Also, her platelet count decreased over the course of her ICU stay, prompting evaluation of Heparin-Induced Thrombocytopenia and Thrombosis (HITT) with laboratory testing for platelet-factor 4 and serotonin-release assay. Heparin was then switched to argatroban. The patient continued to have thrombi due to a suspected hypercoagulable state. She became progressively acidotic and hypothermic, with low cardiac indices and hypotension, requiring maximal inotrope and vasopressor support. After a discussion with the patient's family, comfort care measures were instituted. The patient died of multiple organ failure.

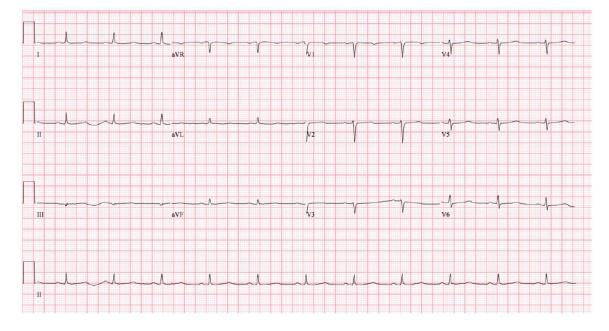


Fig. (1). EKG showing normal sinus rhythm with poor R wave progression. (A higher resolution / colour version of this figure is available in the electronic copy of the article).

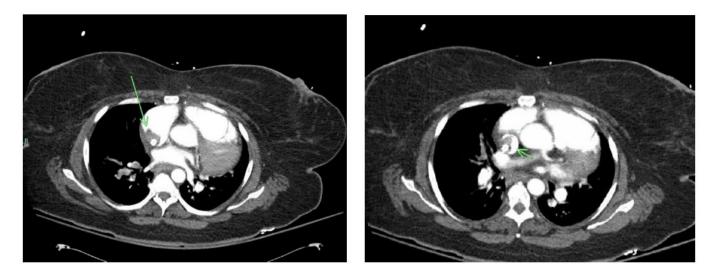


Fig. (2). CT angiography of the chest showing thrombus in the lower SVC, associated with the tip of the left IJ sheath and in the right atrium. (*A higher resolution / colour version of this figure is available in the electronic copy of the article*).



Fig. (3). TEE revealed an octopus-like clot in the SVC with highly mobile appendages, extending into the right atrium. (A higher resolution / colour version of this figure is available in the electronic copy of the article).

# 4. DISCUSSION

After the literature review, we found 23 cases of iatrogenic RAT complicated with PE, as shown in Table 1. 16/23(69.6%) were catheter-related, 1/23 (4.3%) was shunt-related, 5/23 (21.7%) were pacemaker-related, and 1/23 (4.3%) occurred in the setting of CABG. Factors that increased the risk of RAT development included large atrial size and insufficient emptying rate from the right atrium [8].

# Table 1. A literature review showing 23 cases of iatrogenic RAT complicated with PE.

Author	Iatrogenic Etiology	Treatment	Outcome
Burns et al. [5]	PICC line	Thrombolysis with rTPA	Platelet count recovered, and hydroxyurea initiat- ed for CML.
Stavroulopoulos <i>et al.</i> [7]	Internal jugular hemodialysis ca- theter	Subcutaneous tinzaparin 14000 IU/day, fol- lowed by acenocoumarol	Echocardiography was performed at weekly inter- vals and showed complete dissolution of the thrombus 3 months later. Oral anticoagulation was continued for 6 months, and the course of the patient was uneventful.
Karavidas et al. [16]	Transvenous pacemaker lead thrombosis	Heparin and warfarin	Complete resolution of thrombus seen on TEE 20 days later, no further episodes of PE in 6 months of follow up.
Amankwah et al. [24]	Transvenous pacing wires	Catheter-directed thrombolysis (CDT) with ul- trasound-assisted EKOS EndoWave infusion catheter	Was discharged home on warfarin, 1-year surveil- lance echocardiogram showed no recurrence of thrombus.
Habibi et al. [25]	Pacemaker lead in the RA	Surgical excision	Resolution of PE on warfarin
Ruiz-bailen et al. [26]	AV synchronous pacemaker im- plantation	Alteplase then performed surgery	Died during surgery
Senarslan et al. [27]	CABG for multivessel disease	Clot retrieval from the right atrium	Died due to persistent RV failure and pulmonary HTN, despite maximizing the inotropic support.
Abid <i>et al.</i> [28]	Dialysis catheter in the internal jugular vein	The internal jugular dialysis catheter was re- moved, and a femoral dialysis catheter inserted	No further episode of chest pain. Was stable as an outpatient on hemodialysis awaiting renal trans- plantation.
Coan <i>et al.</i> [29]	Indwelling hemodialysis ca- theter (HeRO graft) occlusion two weeks after cadaveric renal transplantation	Removal of HeRO catheter	Therapeutic anticoagulation with heparin and Coumadin was recommenced.
Gehling et al. [30]	Central venous catheter	Anticoagulation and immediate operation	Died of protracted shock
Ghani <i>et al.</i> [31]	Patient 1: subclavian hemodialy- sis catheter Patient 2: subclavian hemodialy- sis catheter	Patient 1: anticoagulation and antibiotics Patient 2: anticoagulation, antibiotics, then atri- otomy	Both patients died.
Gressianu et al. [32]	Indwelling CVC	Open-heart surgery: removal of both the per- manent catheter and the adherent friable irregu- lar mass	Complete resolution of clinical symptoms.
Kingdon et al. [33]	Patient 1: Central venous ca- theter Patient 2: tunneled right subcla- vian catheter Patient 3. two single-lumen dialy- sis catheters	Patient 1: Warfarin Patient 2: anticoagulation with goal INR 3.5 Patient 3: therapeutic anticoagulation	Patient 1: died Patient 2: received a successful cadaveric renal transplant in April 2000 Patient 3: died.
Kinney et al. [34]	Pacemaker lead thrombus	Multiple courses of heparin, surgical removal of pacing wire, and RAT	Unremarkable recovery.
Lanza <i>et al</i> . [35]	CVC-related right atrial throm- bosis	Initial anticoagulation, then mass was surgical- ly removed <i>via</i> a minimally invasive approach with right minithoracotomy access	Prompt recovery.
Mujanovic <i>et al.</i> [36]	A ventriculoatrial shunt that was inserted 20 years ago	Standard median sternotomy and use of car- diopulmonary bypass, right atriotomy with the extraction of the distal part of the shunt ca- theter; pulmonary arteriotomy with the extrac- tion of a 3x3 cm calcified mass	Development of a right pleural effusion requiring drainage and decortication; TTE showed no resid- ual masses.
Ram et al. [37]	The internal jugular venous ca- theter	Unfractionated heparin, followed by warfarin	Symptom-free at the end of 6 weeks, repeated echo after 2 months did not show RAT.
Vicol <i>et al.</i> [38]	Thrombus adherent to the tip of the Port-a- Cath	Surgical removal of the thrombus	Uneventful postoperative recovery.
Vyalhakar et al. [39]	CRAT and septic pulmonary em- bolism	Unfractionated heparin	Discharge against medical advice and loss to fol- low up.
Zhang et al. [40]	RIJ tunneled cuffed catheter	Transcatheter thrombolysis and fraxiparine	Uneventful after treatment, thrombus disap- peared, anticoagulation stopped.

Note: All cases in the table had right atrial thrombus with pulmonary embolism.

There are two types of thrombi discussed in the literature: type A and type B. Type A thrombi originate in the deep venous system and easily embolize due to their freefloating nature [9]. By contrast, Type B thrombi are stationary, fixed to the right atrial wall, less likely to embolize, and are less susceptible to thrombolysis [6].

#### 4.1. Diagnosis

The most commonly reported diagnostic modality for RAT and PE was echocardiography. Both transthoracic echo (TTE) and transesophageal echo (TEE) reported in the studies were included in our review. Compared to TTE, TEE has better sensitivity and specificity [6]. Since TTE might fail to detect intracardiac thrombi, TEE has been recommended to be used as an initial diagnostic modality [10].

Another imaging tool is multidetector Computed Tomography (CT), which was found to be useful when used emergently in hemodynamically unstable patients with suspected PE. CT has 97% sensitivity for the identification of pulmonary arterial emboli [11, 12].

However, Burns *et al.* found that spiral CT may be difficult to perform in hemodynamically unstable patients, who should undergo echocardiography instead. Compared to TTE, TEE demonstrated better detection and characterization of right heart thromboembolism (RHTE) [5].

#### 4.2. Management

Clot burden within the right atrium and pulmonary artery can be treated effectively with thrombolytics, with recombinant tissue plasminogen activator (rtPA) being preferred over other thrombolytics due to its specificity for fibrin. Also, rtPA has a short half-life, which permits surgical intervention if thrombolysis is ineffective [6].

Furthermore, there is a correlation between the thrombus subtype and the likelihood of successful thrombolysis. Patients with type A thrombi with concomitant PE benefit from systemic thrombolysis, as it leads to thrombus resolution with restored blood flow through the pulmonary circulation and reduced pulmonary hypertension [6]. The strategy of thrombolysis is effective but carries a risk of embolization with type B clots [5]. By contrast, Type A clots can be lysed without such an increased risk [5].

Other strategies that can be used in thromboembolism management include surgical embolectomy and anticoagulation. Surgical removal of an embolus may be effective but can be complicated by cardiogenic shock [8]. Nezami et al. found that simultaneous TTE-guided RA mechanical thrombectomy and fluoroscopy-guided pulmonary artery embolectomy corrected right atrial and PA pressures [13]. A device for mechanical retrieval of PE was suggested to have more benefit and less risk of bleeding compared to systemic thrombolysis with tPA. Multiple clot retrieval devices, such as Angiovac and FlowTriever, have been found to be effective interventions in the patients instead of open surgical embolectomy [13, 14]. Regarding anticoagulation, either heparin or low-molecular-weight heparin (LMWH) can be used. Compared to heparin, LMWH has a longer half-life, better bioavailability, and dose-dependent response [15]. The use of intravenous heparin should be followed by warfarin or thrombolysis in patients with PE, caused by a pacemaker lead [16]. When this anticoagulation regimen fails, the pacemaker lead should be removed [16]. Compared to surgical intervention and heparin use, thrombolysis has advantages, which include wide availability, simple administration, cost-effectiveness, and a comparable survival rate [17-19].

Even though there is evidence to support the use of various treatment modalities for RAT with PE, there is no consensus surrounding the most appropriate treatment strategy for patients with RHTE without PE.

#### 4.3. Morbidity and Mortality

The mortality rate associated with patients who have a mobile right heart thrombus is between 21% - 44%, even though the right heart is the source of pulmonary emboli less than 10% of the time [20]. Patients who do not undergo treatment have a mortality rate that approaches 80-100% [21]. When the source of RAT is a catheter, the risk of pulmonary thromboembolism is increased by 40%, with a mortality rate approaching 28% - 31% [18, 22].

Patients who undergo treatment also face risks. For example, systemic thrombolysis has a 22% risk of major bleeding, a 3% risk of intracranial bleeding, and a breakup of mobile thrombi with distal embolization, causing recurrent PE [23]. Compared to systemic tPA, localized tPA has a reduced risk of bleeding while effectively disintegrating the clot. Amankwah *et al.* showed a favorable outcome using localized tPA during catheter-directed thrombolysis [24].

# CONCLUSION

Given the number of cases of RAT complicated with PE, it is important to include RAT as a potential etiology of PE, especially in patients who have indwelling devices or central venous catheters. Thus, clinicians should suspect thromboembolic complications related to CVC or PICC lines in symptomatic patients. Earlier detection of catheter-related thrombosis with TEE can reduce the risk of complications, and guide the clinicians in selecting the appropriate, individualized treatment strategy, with the goal of obtaining favorable outcomes.

#### **CONSENT FOR PUBLICATION**

Not applicable.

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None.

#### **CONFLICT OF INTEREST**

The authors declare no conflict of interest, financial or otherwise.

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