

Mohammad K. Ghani  
Fernando Boccalandro  
Ali E. Denktas  
Eddy Barasch

## Right atrial thrombus formation associated with central venous catheters utilization in hemodialysis patients

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M. K. Ghani · F. Boccalandro  
A. E. Denktas  
Medical School,  
University of Texas-Houston,  
Houston, Tex., USA

E. Barasch (✉)  
St. Francis Hospital,  
Department of Research and Education,  
Stony Brook University SUNY,  
100 Port Washington Blvd.,  
Roslyn, N.Y., 11576, USA  
e-mail: Eddy.Barasch@chsli.org  
Tel.: +1-516-6224534  
Fax: +1-516-6224568

**Abstract** *Objective:* To report five patients on hemodialysis via an indwelling central venous catheter who developed a thrombus located exclusively on the right atrial wall opposing the emptying site of the superior vena cava and to determine the possible cause of this location. *Design:* Transthoracic echocardiography was performed in four of the patients as work-up for suspected endocarditis or pulmonary embolism and in one patient for syncope evaluation. The right atrial clots were confirmed in all the patients by transesophageal echocardiography. *Design and setting:* General intensive care unit of a university hospital, tertiary referral center. *Patients:* Five patients with end-stage chronic renal failure on hemodialysis via subclavian or internal jugular vein catheter. *Interventions:* Three of these patients underwent surgical throm-

bectomy, and two others were medically treated. *Measurements and results:* The clots were 2–4 cm in length and three of them were infected. Two of the three surgically treated patients and one of the two medically treated patients died. All the patients had the catheter tip in the right atrium, in two of them the bent catheter rubbed the atrial endocardium, and in all the cases the clot was located on the atrial free wall facing the superior vena cava emptying. *Conclusions:* We postulate that the mechanism of thrombus formation at this location is related to friction of the catheter on the atrial endocardium, and therefore positioning the distal segment of the central venous catheters in the right atrium should be avoided.

**Keywords** Thrombus · Central venous catheter · Right atrium

### Introduction

Thrombosis is a relatively common complication of central venous indwelling catheters (CVC), with an incidence between 1.9% and 42% depending on the site of catheter insertion for comparable duration of catheterization and patients characteristics [1, 2]. Although right atrial (RA) thrombi are thought to be uncommon, a recent report showed that within 6–8 weeks of catheter insertion 12.5% of patients had thrombus at the tip of the catheter [3]. In that study all the thrombi were visualized in those patients in whom the catheter tip was in the RA

and none in those with catheter tip at junction of the superior vena cava (SVC) and RA or within the SVC itself. The common mechanisms of clot formation appear to be related to intraluminal clot elongation [4] or endothelial RA damage secondary to the continuous friction of the catheter on the RA free wall [5].

We report five patients with end-stage renal failure on hemodialysis (Table 1) with indwelling RA catheters and in whom a clot was found by transthoracic echocardiography (TTE) and confirmed by transesophageal echocardiography (TEE) to be in the RA attached to its free wall opposite to the SVC emptying site.

**Table 1** The main clinical, echocardiographic, and surgical characteristics of the five studied patients (*DM* diabetes mellitus, *HT* arterial hypertension)

Patient no	Age (years)	Sex	Reason for admission	Medical history	Time to diagnosis <sup>a</sup>	Clot size (cm)	Infected clot	Blood cultures	Treatment	Outcome
1	23	F	Fever, chills	Type I DM, HT	5 weeks	3.5/3	Yes	<i>S. aureus</i>	Surgical	Poor <sup>b</sup>
2	57	M	Syncope	Type II DM, HT	6 months	3.1/2.5	No	—	Surgical	Died <sup>c</sup>
3	55	F	Fever, chest pain	Type I DM, HT	4 months	3.7/3	Yes	<i>S. aureus</i> , <i>Enterococcus</i>	Medical	Died <sup>d</sup>
4	55	F	Fever, diarrhea	Type II DM,	8 months	4/2.1	Yes	<i>S. aureus</i> , yeast	Surgical	Died <sup>e</sup>
5	23	F	Dyspnea	Type I DM	4 months	2.5/2.1	No	—	Medical	Good

<sup>a</sup>Time between catheter insertion and echo diagnosis of a RA mass

<sup>b</sup>Transferred to hospice

<sup>c</sup>Intractable heart failure

<sup>d</sup>Pulmonary embolism

<sup>e</sup>Postoperative pulmonary embolism

## Case reports

### Patient 1

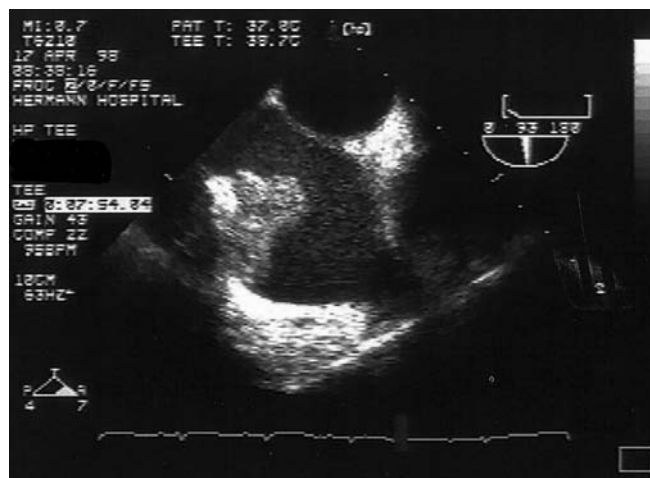
A 23-year-old woman with a history of type 1 diabetes, hypertension, and end-stage renal disease (ESRD) was admitted with a 4-day history of high fever and chills 6 weeks after placement of a right internal jugular catheter for initiation of dialysis. Examination showed her to have a temperature of 40.5°C and a 3/6 holosystolic murmur at the left lower sternal border. Blood and catheter tip cultures grew methicillin-sensitive *Staphylococcus aureus*. TTE revealed moderate tricuspid regurgitation and a possible a RA mass. Confirmed by TEE, the mass was attached to the RA free wall opposite site to SVC emptying. This mass was surgically excised, and on pathology an infected 3.5×3.0×0.8 cm clot was found. Following surgery the patient was transferred to a long-term care facility.

### Patient 2

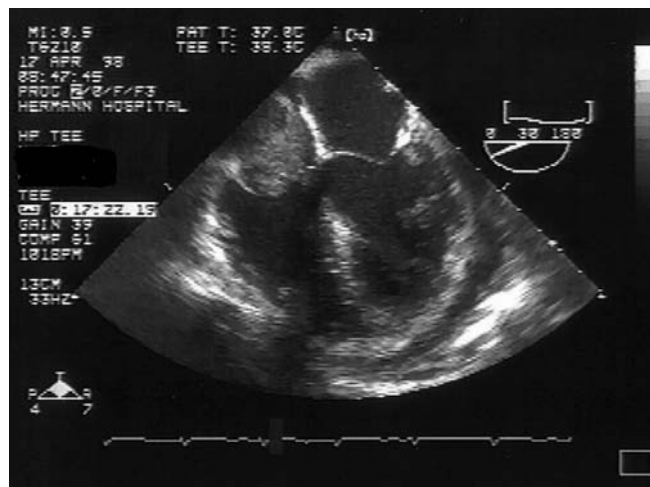
A 57-year-old man with history of hypertension, diabetes mellitus, and ESRD on hemodialysis via a subclavian vein inserted Quinton catheter for the past 6 months was admitted for recurrent syncope. Examination revealed increased jugular venous pressure, S3, and mild peripheral edema. TTE showed an enlarged, hypocontractile left ventricle and a RA mass of 3.1×2.5 cm prolapsing in diastole into the right ventricle (Figs. 1, 2) confirmed by TEE to be located on the atrial wall opposite to SVC emptying. At surgery an organized thrombus was found, but the patient developed postoperative worsening of congestive heart failure and died 8 days later.

### Patient 3

A 55-year-old woman with 23-year history of diabetes mellitus, hypertension, and ESRD on hemodialysis via subclavian catheter for the past 4 months was admitted with 2 days history of fever and chest pain. On examination she had a temperature of 38.3°C and a 2/6 systolic murmur at the apex. Blood cultures grew *Enterococcus* and methicillin-resistant *S. aureus*. TTE showed moderate mitral regurgitation and a RA mass attached to the dialysis catheter which was confirmed by TEE. The 3.7×3.0 cm mass had the same location as those in the previous two patients. Anticoagulation and antibiotics were initiated, but the patient developed pulmonary embolism, myocardial infarction, and pulseless electrical activity and died.



**Fig. 1** Multiplane TEE at 93° interrogation angle in patient no. 2, showing a large clot attached to the endocardium of the right atrial free wall on the opposite site of SVC emptying. The hemodialysis catheter was removed



**Fig. 2** Systolic frame of the TEE stirred at 30° in patient no. 2, showing the clot touching the tricuspid valve leaflets and a small pericardial effusion. In diastole this clot was prolapsing into the right ventricular cavity

#### Patient 4

A 55-year-old woman with history of diabetes mellitus, below-knee amputation, and 6-year history of ESRD on hemodialysis for the past 8 months via subclavian CVC was admitted with fever and diarrhea. On examination we found a 3/6 systolic murmur at left lower sternal border. Blood cultures grew *S. aureus* and yeast (non-*Candida albicans*). TEE revealed a 3.9×1.4 cm RA mass attached to its free wall and severe tricuspid regurgitation. In spite of anticoagulation therapy and antibiotics the patient continued to be febrile and developed septic pulmonary embolism and endophthalmitis. Atriotomy was performed, and a 4×2.1 cm infected thrombus was found attached to the RA endocardium facing the SVC. The postoperative course was complicated by pulmonary embolism and cardiogenic shock, and the patient died the following day.

#### Patient 5

A 23-year old woman with diabetes mellitus and ESRD on dialysis via subclavian Quinton catheter underwent TTE for evaluation of pericardial effusion. This revealed a moderately sized pericardial effusion, moderate tricuspid regurgitation, and a RA mass. On TEE a 2.5×2.1×1.3 cm RA mass attached to its free wall just was identified. The tip of the Quinton catheter was in the RA moving back and forth being embedded into the thrombus. The catheter was removed, and anticoagulation therapy was initiated. After 6 months of coumadin treatment the thrombus resolved completely, and the clinical course remained uneventful.

## Discussion

The incidence of thrombus formation in SVC or RA associated with an indwelling CVC varies from 2% to 17%, with a systemic infection rate of 6.7% [1, 2, 3, 4, 5]. A number of case reports have been published regarding CVC-associated RA thrombi, and the postulated mechanisms include intraluminal clot elongation [3], fluid dynamics of the RA with the catheter tip being located in the region of relative separation or stagnation of blood flow [2], and mechanical irritation of the RA free wall [4].

The five patients presented here had thrombosis associated with CVC placed for hemodialysis; three of them had signs of systemic infection, and all had the catheter tip extending into the RA. The thrombi were located on the RA wall opposite to the entrance of the SVC. Trauma to the free wall by the catheter tip or, less likely, by the jet of infused medication might be the underlying mechanism of endothelial damage and clot formation, as previously proposed [4]. In our series only one patient had an uneventful course. In this case the clot was an incidental finding on TTE, and there were no symptoms related to the presence of the blood clot. However, three patients presented with systemic infection and one with syncope possibly due to transient obstruction of right ventricle inflow. It is clear from the surgical specimen obtained from these patients that with long-term indwelling catheters the thrombus can organize and infiltrate the RA musculature and, secondarily, become infected, with dire consequences.

In a recent prospective study [2] in which 55 patients were evaluated by TEE 46% of the catheters placed in the RA developed thrombus within 1 week, in contrast to patients in whom the catheter was at the RA-SVC junction or within the SVC in whom no thrombus was found at 1 or 6–8 weeks. All the patients who developed thrombus in that study were asymptomatic. There have been cases reported in the literature with infected thrombus in the RA in the presence of a CVC [3, 5, 6, 7, 8, 9, 10, 11]. Although not very common, an infected atrial thrombus can be potentially lethal if not treated, and in many cases surgical removal is required [10].

In cases of a RA thrombus associated with CVC, if the thrombus is not adherent to the atrial wall or SVC, it has been proposed that removal of the CVC might result in massive pulmonary emboli and possible death [8]. Therefore the use of TEE even before the CVC is removed can be advocated. Apart from removing the CVC antibiotic use is of utmost importance. Most common organism in these cases appears to be *Staphylococcus* species [11]. Anticoagulation to prevent further propagation of the thrombus and emboli has been suggested, but occasionally this is not effective [12]. Thrombolytic therapy or IIb/IIIa receptor antagonist has also been used in some cases [13, 14]. In the case of a freely moving thrombus or prolapsing thrombus across the tricuspid valve the risk of massive pulmonary embolism is great, and thrombolytics should probably not be used [15]. Although a meta-analysis of such cases showed no difference in outcome after treatment with heparin, thrombolytics, or surgery, the reviewed patients were not infected. Therefore it has been suggested that when there is an infected RA thrombus, surgery should be considered [10].

Based on the results of this report and on the previous studies we believe that CVC should be advised to be positioned in such a manner to avoid its tip protruding into the RA and potentially contributing to a clot formation. Although routine echocardiographic surveillance of such patients is not considered indicated by the present guidelines, further studies are warranted to determine the cost-effectiveness of such a strategy for identifying the presence of an intra-atrial thrombus at an early stage of development when anticoagulation therapy might be more effective and the potential for infection is less threatening.

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