The Hemodialysis Catheter-Related Right Atrial Thrombus: Case Report

Hemodialyiz Kateteri ile İlişkili Sağ Atriyal Trombüs

ABSTRACT Central venous catheter (CVC) is widely used at clinics for dialysis, administration of chemotherapy, blood products, total parenteral nutrition, large volumes of fluids, or multiple and concurrent intravenous drugs. Right atrial thrombus (RAT) has often been reported both for the subclavian and internal jugular haemodialysis catheters as a life-threatening complication of what has become a routine procedure. Transthoracic echocardiography (TTE) was performed in the patient as work-up for hemodialysis catheter-induced thrombus. Then transesophageal echocardiography (TEE) was performed to evaluate the association between thrombus and hemodialysis catheter. Thrombolytic therapy was initiated but the patient died the following day. The present case suggests that echocardiography should be considered and performed at certain time intervals even though patients are free of symptoms in such cases.

Key Words: Thrombosis; catheterization


Anahtar Kelimeler: Trombus; kateter


Thrombosis is a relatively common complication of central venous catheter (CVC) with an incidence between 1.9% and 42%, depending on the site of catheter insertion for comparable duration of catheterization and patient’s characteristics. 1, 2 Although right atrial thrombus (RAT) is 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 The common mechanisms of clot formation appear to be related to intraluminal clot elongation 4 or endothelial right atrium (RA) damage secondary to the continuous friction of the catheter on the RA free wall. 5 Although CVC-related thrombosis is often asymptomatic, the occurrence of right atrial thrombosis warrants adequate treatment because it can
result in potentially life-threatening complications. We report a patient with end-stage renal failure on hemodialysis 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 the superior vena cava (SVC) emptying site.

CASE REPORT

A 51-year-old man with end-stage renal disease was started on hemodialysis via dialysis catheter which was inserted into the right subclavian vein 2 months ago, and then he was admitted to our hospital with a 5-day history of dyspnea on exertion and coughing. Clinical examination revealed slightly increased intensity of the pulmonary component of the second heart sound. The pulse rate was within normal limits. Systemic blood pressure was 110/70 mmHg. Chest X-ray was normal. The cardio-thoracic ratio was found to be 0.5. The patient was in sinus rhythm. ECG showed slight right axis deviation but there was no voltage criterion for right ventricular hypertrophy.

TTE revealed an abnormal mass of 2.6 x 3.3 cm in dimension, which was a non-mobile thrombus in the RA but the catheter was not seen (Figure 1). The left heart chamber sizes were within normal limits. The right atrium and ventricle were slightly enlarged. There was no dysfunction of the left ventricle (L.V). Both systolic and early diastolic filling were within normal limits. Other important echocardiographic data included the pulmonary flow pattern studied by continuous wave Doppler Echocardiography, which revealed increased peak systolic pulmonary artery pressure (SPAP) 35 mmHg (normal up to 30 mmHg). Diastolic (DPAP) and mean pulmonary artery pressure (MPAP) were 7 mmHg and 18 mmHg, respectively (normal DPAP= 6 mmHg, normal MPAP= 12 ± 2 mmHg). The mean pulmonary pressure/mean systemic pressure (Pp/Sp) ratio increased to 0.2 (normal value around 0.1205 ± 0.0045). TEE was performed and confirmed our previous findings determined by TTE (Figure 2). The catheter tip was clearly delineated within the right atrium with a large mass attached to the atrial free wall.

Because of the potential high risk of pulmonary embolism, such a measure as CVC was not removed. In this situation we planned thrombolytic treatment firstly. A thrombolytic and anticoagulant treatment with rh-tPA, 0.1 mg/kg per h for 12 h and heparin, 10 IU/kg per h for 24 h was consequently started. Blood counts and coagulation tests were as follows: WBC 5x10^9/L, Hb 12.7 g/L, platelets 62x10^9/L; prothrombin time 81% (normal range 75-112), thromboplastin time 24 s (normal range 23-32), fibrinogen 3.5 g/L (normal range 1.6-4.5) fibrin degradation products (FDP) 345 μg/L (normal value < 350). The patient developed pulmonary embolism at the second days of thrombolytic treatment. Unfortunately, he was not responded to cardio pulmonary resuscitation.

DISCUSSION

A number of case reports have been published regarding CVC-associated RA thrombi. Postulated mechanisms include intraluminal clot elongation, fluid dynamics of the RA with the catheter tip being located in the region of relative separation or stagnation of blood flow, and mechanical irritation of the RA free wall. Most thrombi are likely symptomatic; however, some evidence suggests that almost half (46%) of all catheter tips located in the right atrium will develop clinically significant thrombus.

In a recent prospective study 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. The thrombi were located on the RA wall opposite 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. Mechanical irritation of the catheter on the mobile atrial wall also provides clot stimulus as in our case.

TTE is often used first as in our case; however, TTE can be limited in visualizing the SVC, catheter and small thrombi. TEE is the most effective imaging technique in identifying catheter tip placement and cardiac thrombi in most cases. In our case, there was need to use TEE to establish and confirm the diagnosis.

The benefit of systemic prophylaxis with low-molecular-weight heparin (LMWH) or warfarin has not been well established. The sixth guidelines from the American College of Chest Physicians (ACCP) state that 1 mg/day warfarin or LMWH administered once a day are valid prophylactic options for CVC thrombosis. Although many previous studies support the use of low-dose warfarin, most recent studies do not. In a study, 1 mg/day warfarin resulted in 0 of 52 (0%) patients developing CVC thrombosis, versus 4 of 65% (6%) patients not receiving warfarin developing CVC thrombosis (p= 0.06). Low-dose warfarin or LMWH prophylaxis remains controversial. On the other hand, during hemostatic defects (protein C or protein S deficiency, antiphospholipid antibodies, 2021 prothrombin mutation or Factor V Leiden mutation, malignancy e.g.) low-dose warfarin or LMWH prophylaxis are warranted.

As the mortality risk associated with atrial thrombus is quoted at 28% to 31%, an appreciation of this possible complication and its management is imperative to the medical and surgical community. Management of RAT has limited options. Simply the catheter does not reduce the risk of serious complications. The choice of surgery or thrombolytic therapy has always been a therapeutic dilemma. Presence of syncope or an infected atrial thrombus surgical removal of the thrombus is required. Because of the thrombus can organize and infiltrate the RA musculature and secondarily, an infected atrial thrombus can be potentially lethal if not treated. Surgical removal of the thrombus is nonetheless only a second-line option for where medical therapy fails, because of its invasiveness. The current literature definitely favors thrombolytic therapy. An interesting alternative method in high risk surgical candidates or those with contraindications to anticoagulation or thrombolysis may include endovascular stripping or thrombectomy via femoral access, but evidence suggests this is not a permanent measure as the clot recurs. The optimal treatment for catheter-induced RAT remains controversial, although most physicians advocate drug therapy without major surgery.

In conclusion, we believe that CVC should be advised to be positioned in the superior vena cava or at its junction with the RA. Protrusion of catheter tip into the right atrium should be prevented not to contribute to a clot formation.
HEMODİYALİZ KATETERİ İLE İLİŞKİLİ SAĞ ATRİAL TROMBÜS

REFERENCES


