Right atrial thrombosis in systemic lupus erythematosus

Sirs,
The finding of atrial thrombosis (AT) in the course of systemic lupus erythematosus (SLE) is a rare event. We report the case of a patient with SLE who developed deep venous thrombosis (DVT) and right AT concomitant with central vein catheter-related sepsis.

A 19-year-old female arrived in our department with fever, malar rash and arthritis. She underwent tests and laboratory values showed: microcytic non-hemolytic anemia (Hb 7.3 g/dl), leucopenia, thrombocytopenia, glomerular filtration rate (GFR) 53 ml/min, total proteinemia 5.1 g/dl, albuminemia 2.5 g/dl, gammaglobulins 23%, aPTT 44 s, total protidemia 5.1 g/dl, albumine 2.5 g/dl, gammaglobulins 23%, aPTT 25s [normal value (NV) < 25s], kaolin clotting time (KCT) ratio 0.97 (NV < 1.2), lupus anticoagulant (LAC) assessed by the dilute Russell viper venom test (DRVVT) 0.87 (NV < 1.2), hypocomplementemia, proteinuria 24 hr 6 g/l, and many red cells and hyaline casts in the urinary sediment. She was ANA positive 1:640 (homogeneous pattern), anti-Sm-RNP negative, and anti-Ro-La negative. Anti-dsDNA were > 120 U/ml (NV < 7); antカードiolipin antibodies (aPL) IgG 15.8, GPL U/ml (NV < 7), and IgM 6.6 MPL U/ml (NV < 4); and anti-2-GPI IgG 9 (<3.4) and IgM negative. Echocardiogram revealed mild pericardial effusion.

SLE was diagnosed and the following treatment was administered to the patient: i.v. methylprednisolone (750 mg/day for 3 days) followed by oral prednisone 1 mg/Kg/day and cyclophosphamide pulses (500 mg every 15 days), associated with albuprmin, ACE-inhibitors and diuretics. During hospitalization it was necessary to tunnel a central venous catheter down the subclavian artery.

Four months later the patient returned to the hospital complaining of pain, swelling and increased thermotactility of the left lower limb, and fever (39°C) preceded by chills. She was again admitted and underwent more tests. Laboratory tests were normal except for C-reactive protein (CRP) 6.37 mg/dl, PLT 62 x10^9/mm^3, and aPTT 44 s. Serial hemocultures were all negative. Echoc color doppler examination of the lower limbs revealed sural,iliac, femoral and popliteal deep venous thrombosis. Treatment was started with calciparine 7500 IU s.c. 3 times/day, piperacillin 2 g x 2 times/day i.v. and lavage of the venous catheter with vancomycin. A transthoracic echocardiogram revealed the presence of thrombus in the right atrium confirmed by a transesophageal echocardiogram which showed the presence of a large thrombus adherent to the entrance of the inferior vena cava and a thrombus close to the tricuspid valve (Fig. 1). The patient was given continuous infusion heparin along with acenocoumarol until an INR of 2.5 – 3.5 was obtained.

After 20 days of treatment echocardiography showed complete resolution of the atrial thrombosis, while echo color doppler revealed partial canalization of the common and external iliac veins. The catheter was removed and the tissue adherent culture showed Staphilococcus epidermidis. Eight months later the patient was in good general condition with just a mild increase of anti-DNA (13.7 U/ml). It was therefore possible to taper the prednisone and cyclophosphamide dosage while anticoagulant therapy remained unvaried.

This patient met some of the APS criteria, but not a sufficient number to classify her syndrome as APS (1). Although atrial thrombosis is a rare finding in SLE, even in patients with APS (2, 3) it is more frequently found in patients carrying a central venous catheter (4-6). Our patient developed simultaneously deep venous and atrial thrombosis; a similar event has been described in a previous case report but deep venous thrombosis occurred after the atrial thrombosis had completely resolved (7). The concomitant presence of the two thrombotic manifestations was linked to the coexistence of several risk factors: aPL, central venous catheter, systemic infection and high doses of steroids. Our data highlight the importance of echocardiography in the evaluation of possible sources of embolism in patients with thrombotic phenomena and aPL or other risk factors for thrombosis (2,3). Finally, it is important to emphasize the positive results obtained with heparin intravenous therapy followed by the oral administration of anticoagulants.

A. VACCA1, MD  A. MAMELI1, MD  P. GARAU2, MD  G. PASSI3, MD  L. MELONI3, MD  A. MATHIEU3, MD  R. MONTISC3, MD

1’II Chair of Rheumatology and 2Chair of Cardiovascular Diseases, Cagliari, Italy. Please address correspondence to:
Prof. Alessandro Mathieu, MD, Cattedra di Reumatologia II, Dipartimento di Scienze Mediche, via San Giorgio 12, I-09100 Cagliari, Italy.
E-mail: mathieu@pacs.unica.it

References