Letters to the Editor

Impending paradoxical embolism and dynamic left ventricular outflow obstruction in a patient with recurrent pulmonary embolism and secondary pulmonary hypertension

In patients where a right atrial thrombus is associated with a patent foramen ovale, paradoxical arterial embolism has been observed. In rare cases, right atrial thrombi have been found trapped while crossing a patent foramen ovale\[1,2\]. We report a case of recurrent pulmonary embolism and impending paradoxical embolism diagnosed by conventional and transoesophageal echocardiography. This finding was associated with dynamic left ventricular outflow obstruction due to acute pulmonary hypertension.

A 64-year-old Caucasian male had been admitted to hospital for a week with dyspnoea. Precordial echocardiography revealed a right atrial mass suggestive of a mobile thrombus. Thrombolytic therapy was started with streptokinase but was complicated by severe pulmonary embolism requiring cardiopulmonary resuscitation. Percussion/ventilation scintigraphy of the lung showed absent perfusion of the right upper and lower lobes and perfusion deficits in the lobus lingualis and anterior upper left lobe segments. Phlebography demonstrated an extensive acute deep vein thrombosis in both legs, partial occlusion of the greater saphenous veins, superficial and deep femoral veins and thrombotic material protruding into both distal iliac veins.

Trans-thoracic echocardiography showed a dilated and hypokinetic right ventricle, a dilated right atrium, and flattening of the interventricular septum with paradoxical systolic motion. The left ventricle was small with modest concentric hypertrophy and hyperdynamic wall motion; the aortic valve appeared normal. Using continuous wave Doppler of tricuspid left ventricular outflow tract gradient to 25 mmHg. Left ventricular function was normal without hyperdynamic wall motion.

Figure 1 A-C Transoesophageal echocardiogram (TEE) showing a mobile thrombus (arrows and TH) in right and left atrium (RA and LA) trapped in a patent foramen ovale.

Therapy for most patients with RA thrombus mainly consists of surgical removal of the material with an excellent outcome\[1,3\]. In the patient described here, in contrast, systemic thrombolytic therapy with urokinase was chosen due to an unacceptable high surgical risk. In the past, successful thrombolysis had been described only for isolated right atrial thrombi\[4\] but not for a thrombus trapped in a patent foramen ovale with the risk of paradoxical embolism. However, we feel that lysis of mobile thrombus trapped in the foramen ovale should be reserved for patients who cannot be operated on\[5\] because of potential further embolism during thrombolysis. At present, surgery still appears to be the favourable therapy as it also allows thrombendarterectomy of the pulmonary artery.

This case is also remarkable because of the presence of a significant left ventricular outflow obstruction due to high right ventricular pressure resulting from pulmonary hypertension. Together with hypertrophy and presumably low preload of the left ventricle, the patient developed a dynamic left ventricular outflow obstruction\[3\]. The obstruction was reversible by thrombolytic removal of the obstructing material in the pulmonary artery and the right atrium. To our knowledge this is the first reported case of documented reversible LV obstruction in the setting of pulmonary artery thromboembolism and atrial thrombus in transit.

References

Acute myocarditis mimicking myocardial infarction in an HIV infected patient

Recent reports have described patients with acute myocarditis masquerading as acute myocardial infarction[1-2]. We observed a similar case in a patient infected with the Human Immunodeficiency Virus-1 (HIV-1).

A 47-year-old woman was admitted to hospital with severe chest pain of sudden onset suggestive of myocardial infarction. There was a 3-year history of chronic hepatitis C which had been managed with a 12-month course of interferon-alpha; this had been discontinued 8 months before admission. The patient had also been seropositive for HIV-1 for 8 years. Both viral infections were transmitted by blood transfusion in 1985.

The patient had previously remained free of all opportunistic infection and was asymptomatic for any HIV-associated illness. The CD4 cell count had been about 400 per mm³ for 3 years when therapy with zidovudine was started.

The patient was well until 2 days before admission when she developed a cough and a 38 °C fever. On admission, the chest-pain, that had lasted for about 20 min, was no longer present. At physical examination, the heart was normal. No cardiac murmur or pericardial friction rub were heard. Blood pressure was 120/70 mmHg. There were no clinical features suggestive of cardiac failure or shortness of breath, but there was slight hepatic enlargement that had been previously noted. Her temperature was 37.8 °C.

There were no clinical or biological signs of liver failure. Transaminase levels were 1-5 times normal, serum albumin and plasma prothrombin time were normal. Other laboratory findings showed a mild neutropenia with 1400 neutrophils per mm³. Serum creatine kinase was within normal values. Despite therapy with intravenous nitrate infusion, another episode of chest pain occurred 24 h after admission. The electrocardiogram which was unremarkable at admission now showed inversion of T waves in leads V1 to V6 associated with an elevation of ST segment in leads II, III and aVF. The chest X-ray was normal. No abnormalities were seen on two-dimensional echocardiography, and there was no pericardial effusion or left ventricular wall dysynchrony. Creatine kinase levels remained unchanged. A radionuclide perfusion lung scan showed no sign of pulmonary embolism.

Coronary angiography was performed, which showed no abnormality of the coronary arteries. Viral antibody titres were serially evaluated and compared with those obtained from several previous blood samples. IgG and IgM antibody levels for T. gondii, Epstein-Barr virus, parvovirus B19, Lyme disease, mycoplasma and rickettsia remained unchanged. Hepatitis B markers remained negative.

Blood and stool cultures for bacteria as well as blood cultures for T. gondii remained negative. There was no cytomegalovirus viremia. Viral cultures for enteroviridae (Echocoxsackie viruses) were not performed. Viral antibody titres for Echocoxsackie viruses were not evaluable, due to hypergammaglobulinemia and related serum anti-complement activity.

The mild transient diarrhoea, lasting for 2 days, occurred but there was no further chest pain. The electrocardiogram normalized within 3 days, and follow-up echocardiography remained normal. The diagnosis of myocarditis mimicking myocardial infarction was ultimately retained. Fifteen months after the onset of her illness, the patient was well and free of all symptoms suggestive of progression of HCV or HIV infection. Therapy with zidovudine 500 mg daily was continued during and after the illness.

Myocarditis due to either opportunistic infections, HIV-1 itself or antiviral therapy has been recognized with an increased frequency among patients infected with HIV-1[3-5]. Considering the initial symptoms of cough, fever and the subsequent diarrhoea, the self-limiting cardiac illness reported in our patient was very probably due to acute myocarditis. Despite multiple blood cultures and serial serum testing, no responsible pathogen was isolated.

As the patient totally recovered, no histological study was performed. Nevertheless, recognizing HIV-1 infected patients with myocarditis mimicking myocardial infarction may have clinical implications. Millaire et al. suggested that echocardiographic examination and subsequent cardiac catheterization might be recommended prior to thrombolytic therapy in young (HIV-seronegative) patients without cardiovascular risk factors, with viral symptoms[5]. Our report suggests that HIV-infected patients should be considered with a similar approach.

References


