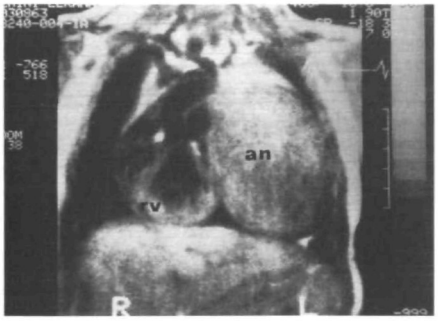


Figure 1 **A large mass demonstrated by MRI to be compressing the left heart. Axial images (not shown) demonstrated this mass to be connected to the left atrium. RV=right ventricle; AN=aneurysm.**

dilated the left atrium[1]. In adults LAA without other associated abnormalities is a rare condition with less than 40 cases reported[2]. As far as we know, only two cases presenting in the first year of life have been reported[2,3]. Congenital aetiology is supported by young age, lack of associated valvular abnormalities, and absence of histological demonstration of a specific degenerative disease in the atrial wall. The clinical presentation of congenital LAA is supraventricular tachyarrhythmias, systemic embolization or congestive heart failure[3]. Congestive heart failure may be related to the space-occupying aneurysmal sac impending pulmonary venous drainage[3]. This pathological process was the cause of dyspnoea in our patient. A chest X-ray may occasionally show an asymptomatic patient to have a deformed left cardiac silhouette, as the result of a prominent left atrial appendage[3]. The differential diagnosis includes valvular disease, pericardial cyst, mediastinal mass and LAA[3]. Cardiac cathetarization and angiography are the diagnostic study of choice for LAA[3]. Systemic embolization and atrial tachyarrhythmias might be induced by such an invasive technique. This caused us and others[4,5] to look for more sensitive and specific non-invasive diagnostic systems, such as colour Doppler and MRI.

Our case shows that in infants, as in adults, MRI, together with colour Doppler seems to supply all the information needed to carry out a surgical resection of congenital LAA[1,3]. Due to the risk of life threatening complications, aneurysmal resection is indicated even for asymptomatic patients[5]. The prognosis of congenital LAA after surgical treatment is excellent[1–5]; no systemic embolization has been reported subsequent to the operation. We conclude that LAA should be considered in the differential diagnoses in infants with mediastinal mass. MRI combined with colour Doppler are sufficient diagnostic tools for pre-surgical elaboration, even in infants with large congenital LAA. Cardiac catheterization in such cases should be omitted as it might lead to life-threatening complications.

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A case of infective endocarditis complicated with anterior mitral valve leaflet abscess

Infective endocarditis is a microbial infection of intact or degenerated cardiac values, the endothelium surrounding congenital or acquired cardiac defects and the endothelium of vascular malformations[1,2]. Although there are many complications of the disease, to our knowledge there are few reported cases complicated by an abscess of the cardiac valve leaflet[3].

We report a case of infective endocarditis very probably complicated by an abscess of the anterior mitral valve leaflet in addition to embolic complications.

A 19-year-old white male presented to the emergency room semi-conscious with fever, abdominal pain, weight loss, motor aphasia and a possible diagnosis of infective endocarditis. His physical examination revealed a body temperature of 38·8 °C, an apical third heart sound, a grade 2/6 pansystolic murmur on the apex, a right hemiparesis, cutaneous embolic phenomenae and a sacral grade 2 decubital ulcer.

Circulating blood cells showed a haemoglobin of $9\ g\ .\ dl^{-1}$, and a haematocrit of 28%. His white blood cells were $12\,200\ mm^{-3}$ with a left shift on the differential count. Erythrocyte sedimentation rate was $75\ mm\ .\ h^{-1}$. His ECG and chest X-ray were normal. A head computed tomography scan showed multiple infarctions. Blood cultures were drawn. His echocardiogram revealed a 1·3 × 0·7 cm mobile vegetation and a prolapse of the anterior mitral valve leaflet into the left atrium. Abdominal ultrasound showed a 12 × 7 cm splenic abscess which was drained and cultured. Three of four blood cultures grew methicillin resistant *Staphylococcus aureus* (MRSA) as did the splenic drainage specimen. On the 12th day of admission, there was no cessation of fever. A Doppler echocardiogram revealed a lesion compatible with an abscess on the anterior mitral valve leaflet (Fig. 1): grade 2–3/4 mitral insufficiency, and grade 1–2/4 tricuspid insufficiency. Surgery was ruled out because of the patient's poor clinical condition. Three weeks after admission his fever ceased and on the 42nd day of admission the patient left hospital, against medical advice. He was put on oral ciprofloxacine 500 mg four times a day and rifampicin 600 mg daily and was requested to

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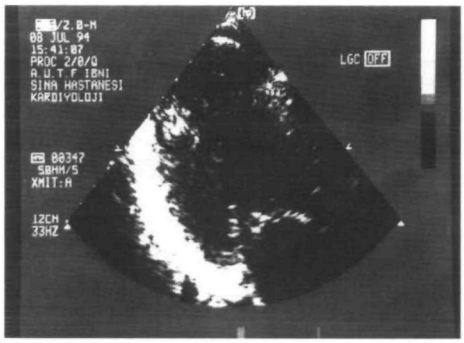


Figure 1. **Circular lesion, compatible with an abscess, on the anterior mitral valve leaflet.**

return to the clinic one month later. The patient failed to return and died at home 3 months later; no autopsy could be performed.

There are many complications of infective endocarditis but an abscess of the cardiac valves is rare[3]. The diagnosis of infective endocarditis is based on the criteria described by many authors, but only the recently described Duke criteria includes intravenous drug abuse and echocardiographic findings[4,5]. In the reported case, three major and two minor Duke criteria were fulfilled, although two major criteria were sufficient for the diagnosis of infective endocarditis. The sensitivity of the Duke criteria is higher than the other proposed criteria[4,5], but the specificity may be lower[5].

In the present case, MRSA was isolated but the disease was clinically resistant to treatment, possibly because of the abscess on the vessel-free anterior mitral valve leaflet. Although no abscess was seen on the first echocardiogram, it is possible to overlook a small one. The prognosis of infective endocarditis complicated with a mitral valve leaflet abscess is poor[3] and because of this early surgical treatment should be advocated.

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Pulmonary hypertension associated with thrombotic thrombocytopaenic purpura in AIDS patients

The association between the Acquired Immunodeficiency Syndrome (AIDS) with either pulmonary hypertension (PH) or thrombotic thrombocytopaenic purpura (TTP) is well known. It occurs in about 0·5%[1] and 0·6%[2,3] of AIDS patients, respectively. However, these three conditions combined have not been reported before, to our knowledge.

Patient 1

A 32-year-old homosexual man had been seropositive for HIV-1 for several years. He was treated for *Pneumocystis carinii* pneumonia (PCP) in 1994. He received zidovudine therapy and secondary prophylaxis with aerosolized pentacarinat and remained free of further opportunistic infection despite a CD4 cell count below 10/mm^3. Routine chest X-ray, electrocardiogram and a 2- dimensional echocardiographic examination (2-DE) coupled with Doppler assessment were normal.

Fever (39 °C) and progressive shortness of breath occurred in April 1995. Laboratory examinations showed microangiopathic anaemia with haemoglobin 6·8 g . dl^{-1}, LDH 2200 UI . l^{-1}, haptoglobin 0·09 g . l^{-1}, schistocytes (10%), thrombocytopaenia with a platelet count of 44 000/mm^3, associated with a mild increase in serum creatinin (140 μmol . l^{-1}) and proteinuria (2·5 g . 24 h^{-1}). These features met the clinical criteria for TTP associated with AIDS[4].

The heart was slightly enlarged on chest X-ray. Sinus tachycardia was noted on the electrocardiogram. A blood gas sample showed hypoxaemia (PaO_2: 65 mmHg) and hypocapnia (PCO_2: 32 mmHg). A radionuclide lung scan disclosed pulmonary embolism but showed diffuse perfusion inhomogeneities.

Echocardiography showed normal left ventricular (LV) volume and performance (51/35 mm, shortening fraction: 32%), and a new onset tricuspid regurgitation (TR) with the TR-velocity derived pulmonary artery systolic pressure 55 mmHg. No opportunistic infection was found, including in the lungs. Broncho-alveolar lavage was normal.

Plasma exchange (two series of four exchanges) associated with prednisone, dipyridamole and acetylsalicyclic acid resulted in a significant improvement within 3 months. Fever disappeared, haemoglobin increased to 9 g . dl^{-1}, platelet counts reached 93 000/mm^3, LDH levels decreased to 900 UI . l^{-1}. Schistocytes counts dropped only slightly from 10% to 3%. A repeat 2-DE showed improved pulmonary artery systolic pressure (30 mmHg) and normal LV function parameters.

The patient remained relatively well with maintenance therapy consisting of aspirin and dipyridamole until he was readmitted in September 1995 for pulmonary oedema. Laboratory examination showed acute relapse of TTP (anaemia 6·5 g . dl^{-1}, thrombocytopaenia 20 000/mm^3). Creatinine levels were 148 μmol . l^{-1}. Chest X-ray showed heart-size enlargement and pulmonary oedema. Two-DE showed mild LV dilatation (60/49 mm) with severe global hypokinesis (shortening fraction 18%). Pulmonary artery systolic pressure was 50 mmHg.

Despite supportive care with dobutamine, diuretics, and plasma exchanges, the patient worsened with

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