

Figure 2. A: polymorphic ventricular tachycardia refractory to cardiac defibrillation. B: yellow arrhythmogenic histiocytoid nodules on leaflets of tricuspid valve (black arrows). C: histological section of the left ventricle displaying large foamy granular cells (white star) and normal myocardial cells (black star).

appeared as sudden deaths in children with no particular history. These patients frequently presented a supraventricular or ventricular arrhythmia uncontrolled by conventional antiarrhythmic treatments. The Wolff-Parkinson-White syndrome, found in almost 10% of the cases, may suggest a diffuse heart disease and may be a marker of gravity. The echocardiographic phenotype is variable. Symmetric or asymmetric hypertrophic cardiomyopathy

with a trabeculated endocardium is very suggestive. Cardiac malformations (ventricular septal defect, atrial septal defect, Shone syndrome) are reported. A normal echocardiography may be found in a focal form of the disease. Histologically, HC is characterized by the presence of nodules with more or less scattering in the myocardium or the endocardium.² The excision of arrhythmogenic nodules is the only treatment that may produce a complete cure and disappearance of arrhythmia.³ Histiocytoid cells have an arrhythmogenic potential and conventional treatment of arrhythmia is not effective. In every case, medical treatment resulted in the death of the patient. For the rare patients who benefit from surgical excision, the long-term prognosis is unknown. Heart transplantation was reported once, in a 30-month-old child.⁴ The etiopathogenesis of the HC is not yet clearly established; HC should be considered as an X-linked mitochondrial disease affecting cardiac muscle. This hypothesis seems confirmed following an association with MLS syndrome, a rare neurodevelopmental disorder that associates linear cutaneous erythematous lesions of the face and the neck, eye abnormalities, and neurological lesions in females monosomic for Xp22.3.⁵

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Available online 25 May 2011

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doi:10.1016/j.rec.2010.11.015

Pulmonary Embolism Caused by Cysto-Atrial Shunt Fragment

Embolia pulmonar causada por un fragmento de una derivación cistoauricular

To the Editor,

A 40-year-old woman presented to the accident and emergency department complaining of acute onset of headache and dizziness, along with right-sided pleuritic chest pain and shortness of breath. She had a history of a symptomatic right temporal lobe arachnoid cyst treated with a cysto-atrial shunt at the age of 25. Her vital signs were stable and ECG was unremarkable.

Chest radiography depicted a sizable, serpentine-shaped, foreign body within the left hilum (Fig. 1A) and a lateral skull X-ray showed fragmentation of the cysto-atrial catheter at the level of the skull (Fig. 1B). A noncontrast chest computed tomography demonstrated a large catheter fragment embolizing the pulmonary trunk and the left main pulmonary artery (Fig. 2). Patient underwent urgent percutaneous fragment retrieval with a snare apparatus catheter followed by cystoperitoneal shunt implantation, and made an uneventful recovery.

Placement of the distal catheter of a cerebrospinal fluid diversion apparatus in the right atrium is a well-established alternative when peritoneal insertion is contraindicated. Cardiac thrombus formation and chronic thromboembolic pulmonary hypertension are the most common cardiovascular complica-

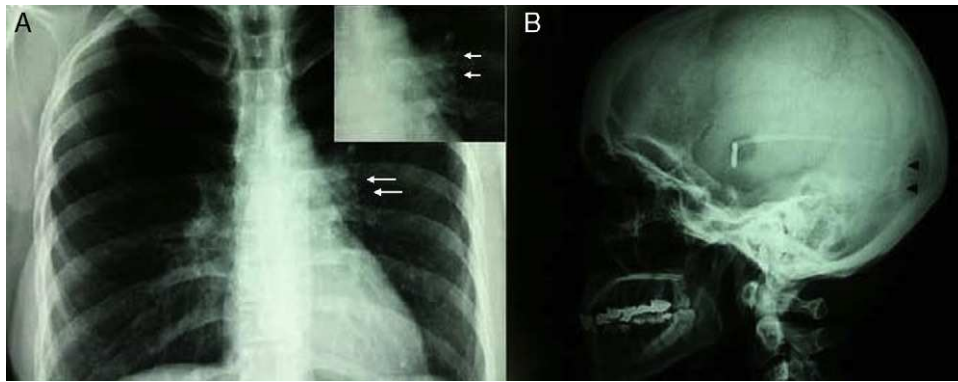


Figure 1. A, Anteroposterior chest radiograph showing serpentine-shaped foreign body within the left hilum (arrows). B, Lateral skull X-ray demonstrating a fragmented cysto-atrial catheter (arrowheads).

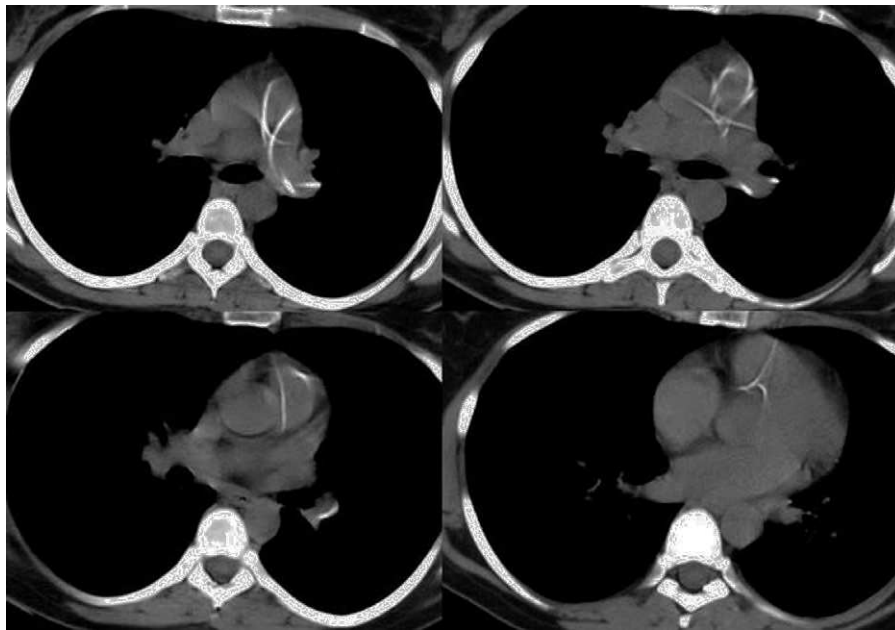


Figure 2. Chest computed tomography scan showing extensive embolization of the pulmonary trunk and main left pulmonary artery by the fragmented catheter.

tions.^{1,2} Migration of catheter fragments to the pulmonary vessels is an extremely rare and potentially fatal complication requiring urgent invasive treatment.

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Available online 12 July 2011

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doi:10.1016/j.rec.2010.11.026