Late Cardiac Perforation After Percutaneous Closure of an Atrial Septal Defect Using an Amplatzer Device

To the Editor:

Percutaneous closure of ostium secundum atrial septum defects is now accepted as a safe, effective alternative to surgical closure in selected cases. Cardiac perforation is rare, but usually observed only during the implant procedure. However, late cardiac perforations (days to months later) have been reported recently.

We describe a 21-year-old woman, diagnosed with an atrial septal defect consisting of ostium secundum, of diameter 22 mm, that led to considerable left-right shunt (L-R 3:1). The
defect was definitively closed with an Amplatzer device of diameter 26 mm, with an uneventful postoperative period. Follow-up echocardiography showed that the device was well positioned, with no evidence of residual shunt or interference with the valve or with the outlet of large vessels. There was no pericardial effusion. At 3 weeks the patient presented syncopal symptoms. She was diagnosed with cardiac tamponade by pericardiocentesis with drawing of 150 mL of hematic fluid. Subsequent echocardiography showed the absence of perfusion, as well as correct placement of the device. Since a self-contained perforation of the atrial wall was suspected, cardiac surgery was undertaken. An ulcerated lesion was found at the edge of the device, in the cranial area of the right atrial septum, where the roofs of both atria and the aortic root meet (Figures 1 and 2). The Amplatzer device was withdrawn and the atrial defect was closed with suturing of the atrial wall wound and an autologous pericardial patch. The postoperative course was satisfactory.

Cardiac perforation related to the implant of an Amplatzer device is a rare complication that usually occurs only during placement of the device.1 However, in a data review provided by the U.S. and Canadian drug agencies, 66% of the 29 cardiac perforations reported were late (post-discharge), 24% occurred 1-6 months later, and only 1 occurred more than a year later (3 years).2 The incidence is not assessable, as the total number of implants recorded is not provided. The patients with cardiac perforation presented chest pain, dyspnea, syncope, hemodynamic collapse, or even sudden death; where surgery or autopsy was performed, the cardiac perforation was found to be in the anterosuperior wall of the atria and/or in the adjacent aorta.

A deficient anterosuperior border or the insertion of a grossly oversized device have been mentioned as predisposing factors.3 Septal aneurysm has not been associated with further complications on follow-up.4 The actual incidence of this complication must be low, since the published series do not describe late perforations. In a series of 417 patients, only one late peripheral embolization and one late sudden death were described.1 Another study assessed 151 long-term (5-9 years) cases, without observing any deaths or major complications.5

In our setting, based on data provided by the Sección de Hemodinámica (Hemodynamics Section) of the Sociedad Española de Cardiología (Spanish Society of Cardiology),6 345 cases of atrial septal defect were closed percutaneously in 2005, with an incidence of 2.9% for complications and 1.2% for death.

Although rare, the potential fatal outcome of this condition requires strict selection criteria for patients and for choosing device sizes, as well as careful postoperative monitoring of these patients, with frequent echocardiographic follow-up during the first 6 months, particularly in the first month. If there is clear evidence of this complication, then the safest approach would probably be surgical withdrawal of the device and conventional closure of the defect once other potential causes for the patient’s symptoms are ruled out.

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To the Editor:

Santiago Ramón y Cajal complained bitterly that his publications would not have been delayed so often if his discoveries had been made in countries of greater scientific prestige, such as France or Germany, instead of Spain. Cajal knew what he was talking about. The 100th anniversary of his Nobel Prize was celebrated in 2006, and perhaps it is now fitting to remember that he was the first to show that Virchow’s cell theory applied to cardiac cells, as they are individual entities delimited by a sarcolemma.1 In this letter, we would also like to help raise awareness concerning certain unknown or forgotten aspects regarding the history of ischemic heart disease.

Horemkenesi (Figure A) was a member of the priestly caste of Ammon, as well as a foreman in charge of the construction of the Pharaohs’ pyramids of the Twentieth Dynasty at Thebes (around 1050 B.C.). According to the hieroglyphics found at his tomb, he had fallen suddenly onto the sand and his body was immediately infested with beetles before mummification. The forensic examination of the mummy 30 centuries later by researchers at the University of Minneapolis determined that he was approximately 60 years of age. Troponin concentrations in tissues taken from the abdominal cavity were found to be similar to the levels of individuals who had died from acute myocardial infarction (AMI) and been embalmed using a similar mummification process, and up to 15 times higher than controls who had not died from AMI.2,3 Thus, Horemkenesi is the first person in whom sudden death due to myocardial infarction has been documented.

In 1510, Leonardo da Vinci (1542-1520) (Figure B) performed an autopsy on an elderly man who had died with no apparent cause at the Hospital de Santa Maria Nuova, in Florence. In his writings, da Vinci describes that the arteries showed considerable tortuosity and states that death was “attributable to the weakness caused by a lack of blood in the artery that nourishes the heart and lower limbs.”4,5 This appears to be the first historical description of coronary death.

Alexander Borodin (1833-1887) (Figure C), composer of the unforgettable opera Prince Igor, was a renowned musician, outstanding chemist, and friend among others of Dimitri Mendelev, who discovered the periodic table of elements. He was also a famous physician who eventually became the director of the Russian Medical-Surgical Academy. After several angina attacks at age 53, Borodin died of sudden death due to ventricular rupture caused by AMI while attending a traditional Russian festival. While he was studying fatty degeneration of the myocardium, in 1871 he discovered with his colleague Krylov that this accumulation was the result of cholesterol, rather than triglycerides as expected.6 Although Borodin does not appear to have been aware of the pathological significance of his discovery, his findings came more than 40 years earlier than observations made by his fellow countryman Anichkov, who induced arteriosclerosis production in the arteries of rabbits fed a high-cholesterol diet.