Coronary Mycotic Aneurysm and Septic Pulmonary Embolisms

To the Editor,

Infectious endocarditis (IE) is a microbial infection of the endocardium. It can be classified in acute, subacute and chronic, according to duration and clinical severity. Its characteristic lesion, the vegetation, is an aggregate of platelets, fibrin, microorganisms and inflammatory cells. The extension of the infection surrounding the valve annulus is a high mortality factor and frequently causes heart failure and the need for surgical treatment. Mycotic aneurysms can be a complication of infectious endocarditis. It is more frequently seen in the aortic root or the Valsalva sinus, where erosion can cause pericarditis, haemopericardium, heart tamponage or fistulas in the left or right heart chambers.¹,²

We present the case of a 47-year-old male, with a personal history of hiatus hernia and obesity, who was admitted with a dry cough, fever, asthenia, anorexia, and loss of weight of several months evolution. Previously he had consulted a physician due to a cough and poor general state of health and had been given antibiotic treatment with levofloxacin. However, as the symptoms persisted he came in to the emergency service at
Letters to the editor

a heart-lung pump, the aortic valve was replaced by a mechanical St. Jude 23 mm prosthesis. It was found that the aortic valve had vegetation, a fistula and a mycotic aneurysm in the proximal part of the ostium of the RCA with the origin of the RCA within. Therefore, a pericardial patch was placed to occlude the aneurysm and a bypass of the RCA artery was performed with saphenous vein. No aortic periannular abscess was found. The immediate postoperative was favourable, and the patient is currently asymptomatic from the cardiovascular point of view.

Perivalvular extension is the most frequent cause of non-controlled infection in infectious endocarditis (IE) (including abscesses, aneurysms, pseudoaneurysms, and fistulae). Mycotic aneurysms are an infrequent but not disdainable complication of IE. These are usually located in the aortic root, Valsalva sinus and visceral or cerebral arteries; but rarely in the coronary arteries, and are usually an autopsy finding.3,4

Maria Facenda-Lorenzo, a Antonio Trugeda-Padilla, a Fernando Moujir-Nasser-Eddine, a and Rafael Llorens-León b

a Servicio de Cardiología, Hospital Universitario Nuestra Señora de Candelaria, Santa Cruz de Tenerife, Spain
b Servicio de Cirugía Cardiaca, Hospiten Rambla, Santa Cruz de Tenerife, Spain

REFERENCES

Acute Pulmonary Edema Secondary to Pregnancy in a Patient With the Mitochondrial Disease MELAS

To the Editor,

The MELAS syndrome (mitochondrial encephalopathy, lactic acidosis and stroke-like episodes) is a mitochondrial disease with heart manifestations in which conduction alterations and hypertrophic (more frequent) or dilated cardiomyopathy are the most outstanding.

A pregnant 23-year-old female undergoes, neurology and cardiology consultations for possible mitochondrial disease (short of height, deaf and with asymptomatic Wolff-Parkinson-White syndrome). On ECG concentric ventricular hypertrophy and normal ventricular function were found, with no other cardiovascular history. Her mother had died at 42 years of age diagnosed with MELAS syndrome, with hypertrophic cardiomyopathy, multiple episodes of acute lung oedema, deafness and repeat ictus. Her grandmother had died in the fourth decade of life due to non-specified heart causes.

From the 20th week of gestation on she began to have episodes in which she had difficulty breathing accompanied occasionally by typical chest pain. During week 23 she had a new episode of dyspnoea, with coughing and right hemithorax pain of pleural characteristics, which caused her to come in to maternity emergency and after which she remained hospitalized.

During physical exam paleness, jugular ingurgitation, tachycardial tones without murmurs and bilateral crepitant rale on auscultation were found. The ECG showed a sinus rhythm with 130 beats/min, a PR interval of 0.04 seconds and a delta wave. Analysis showed: creatinine, 1.4mg/dl; troponin I, 4.42 ng/mL; WBC, 14 370/µL (neutrophils, 13 520/µL, without band neutrophils). Basal blood gases showed pCO2, 24 mmHg; pO2, 45 mmHg, and pH, 7.44.

Twenty-four hours after admission the patient worsened and a chest x-ray showed a pattern of acute lung edema requiring orotracheal intubation and invasive mechanical ventilation. A heart US was performed (Figure 1) and concentric hypertrophy of the left ventricle was seen with a severely depressed systolic function (EF, 30%). Treatment with furosemide, nitroglycerine and dopamine at diuretic doses was initiated.

Four days after admission to the intensive care unit (ICU), fetal death was determined and expulsion took place, subsequently the placenta was extracted and curettage performed. On the tenth day the patient was extubated. The follow-up cardiac mycotic aneurysm of a coronary artery with a perforated mitral valvular aneurysm. J Am Soc Echocardiogr. 2009;22:542.