



## Pathology Biology Section – 2011

### G14 A Case of Lethal Peripartum Eosinophilic Myocarditis

*Maria Carolina Romanelli, MD\*, Eloisa Maselli, MD, Biagio Solarino, PhD, Section of Legal Medicine - University of Bari, Piazza Giulio Cesare, 11, Bari, 70124, ITALY; and Giancarlo Di Vella, PhD, Sezione di Medicina Legale, DIMIMP, University of Bari, Policlinico, Piazza G. Cesare, Bari, 70121, ITALY*

The goal of this presentation is to report an uncommon case of lethal peripartum cardiomyopathy in a young woman. A complete forensic approach was performed through autopsy, histological, and microbiological examinations and final results showed that the cause of death was due to an Eosinophilic Myocarditis (EM).

This presentation will impact the forensic science community showing that eosinophilic myocarditis is a rare, potentially fatal disease if left untreated.

Eosinophilic myocarditis is a histological diagnosis characterized by a mixed inflammatory cell infiltrate containing a variable amount of

eosinophils within the myocardium. This phenomenon may be associated with a variety of disease such as idiopathic hypereosinophilic syndrome (IHES), hypersensitivity myocarditis, giant cell myocarditis, toxic myocarditis, Churg-Strauss syndrome, or parasitic infection.

Clinical presentation includes a wide spectrum of nonspecific signs and symptoms: chest pain, fever, shortness of breath, chills, cough, but they are not always present at the same time and sometimes unusual symptoms, such as epigastric pain, can be the only indication of a pathological state. They can be also associated with peripheral eosinophilia and transient or persistent left ventricular dysfunction.

EM is considered, together with coronary heart dissection, one of the clinical presentations of peripartum cardiomyopathy that usually occurs one month before to six months following delivery. EM etiology and pathogenesis are unknown: eosinophils may be present and activated because of the systemic hormonal perturbation occurring during the period of uterine involution.

A major problem is that EM is rarely recognized clinically and is often first discovered only at postmortem examination.

A correct diagnostic approach in these patients should include an echocardiogram study (with evidence of low ejection fraction and decreased left ventricular systolic function) and an endomyocardial biopsy (confirming eosinophils as a major inflammatory cell component).

If successfully diagnosed, EM can be treated with beta-blockers and ACE inhibitors to support heart failure and corticosteroids to reduce the inflammatory process that is involving the myocardium. Prognosis is strictly linked to ventricular function recovery because those patients with severe myocarditis-induced heart failure have less survival chances if normal cardiac function is not restored.

Few EM cases are reported in literature and most of them are based only on autopsy diagnosis.

A case is reported of a 29-year-old woman who was admitted to critical care unit in respiratory and cardiac failure, three weeks after giving birth. Patient clinical history was non-existent for allergy or autoimmune diseases. The third day after birth, she complained of thoracic pain but echocardiogram was negative. During hospitalization physicians treated her with antacids and gastric inhibitors and then she was discharged with prescription of proton pump inhibitors with the suggestion of gastroenterology visit. The following three weeks were characterized by growing anterior and back thoracic pain associated with general discomfort, but neither specific symptoms nor peripheral eosinophils increase were present; only inflammatory indexes (velocity of erythrocyte sedimentation, VES, and creatine kinase, CK) were slightly increased. With progressive and worsening clinical symptoms, she was finally sent to emergency room in critical condition: dyspnea, confusion, fever, and tachycardia. Echocardiogram showed severe left ventricular systolic dysfunction and 25% of ejection fraction; chest radiograph and TC displayed pleural effusion with general edema. The young woman died after seven hours of cardio-respiratory failure and no medical approach was effective. External examination of the body was completely negative. Autopsy revealed bilateral pleural effusions, increased lung weights, and hepatomegaly. Heart was normal in size and shape, but myocardium and papillary muscles showed malacic areas. Histological examination pointed out massive eosinophilic infiltrates, more evident in cardiac samples. The cause of death was indeed attributed to peripartum eosinophilic myocarditis.

The role of "peripartum" in the etiopathogenesis of such cardiomyopathy as well as possible medical liability in lacking diagnosis and treatment of myocarditis will be discussed.

#### **Eosinophilic Myocarditis, Peripartum, Heart Failure**