

# Coincidence of peripartum cardiomyopathy and systemic lupus erythematosus

Współwystępowanie kardiomiopatii połogowej i toczenia rumieniowego układowego

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Peripartum cardiomyopathy (PPCM) is rare disease of the last month of pregnancy and up to five months postpartum. We present the case of a 26-year-old female who developed PPCM in the course of systemic lupus erythematosus (SLE). The first symptoms of autoimmune disease (polyarthritis, hair loss, skin rash) appeared in the 16<sup>th</sup> week of gestation (WG). Laboratory tests revealed leucopaenia and thrombocytopaenia. In serological tests the presence of antinuclear antibodies, anti-dsDNA antibodies, and anticardiolipin antibodies was observed. In echocardiography the left ventricular (LV) size was 42 mm and ejection fraction (EF) was 70%. Final diagnosis of SLE was made in the 22<sup>nd</sup> WG. Treatment with immunoglobulin (30 g) and methylprednisolone pulse (3 × 500 mg) was started. Subsequent oral methylprednisolone therapy (32 mg daily) was applied. In the 26<sup>th</sup> WG the patient was admitted to hospital for continuation of the treatment — immunoglobulin (30 g), methylprednisolone pulse (3 × 250 mg), and subsequent oral methylprednisolone therapy (36 mg daily). During hospitalisation symptoms of pulmonary embolism (dyspnoea, dry cough, tachycardia) and increased level of D-dimer (5030 ng/mL) were observed. The treatment with dalteparin was started and patient was sent to the Department of Cardiology where finally pulmonary embolism was ruled out (angio-CT). In the 31<sup>st</sup> WG placental abruption occurred. Due to the status of the foetus caesarean section was performed. During operation the status of the patient worsened (saturation fluctuations, tachycardia, vaginal bleeding requiring blood transfusion). Chest radiogram revealed generalised enlargement of the heart and lung changes, which may correspond to pulmonary embolism. Two days after operation sudden cardiac arrest (asystole) occurred twice. In echocardiography (performed the day after) LV size was 51 mm and EF was 10%. The subsequent echocardiography additionally revealed moderate mitral and mild tricuspid insufficiency. Tachycardia was still observed. Cardiological treatment included: dalteparin, perindopril, carvedilol, digoxin, and spironolactone. In control echocardiography after four weeks LV size was 46 mm and EF was 10–15%. Moderate mitral and severe tricuspid insufficiency was present. In the latest echocardiography (after another six weeks) LV size was 33 mm (Fig. 1) and EF was 25–30% (Figs. 2, 3). Trace mitral and tricuspid insufficiency was observed. The patient was initially considered for cardioverter-defibrillator implantation and is now under our observation. The present treatment is as follows: methylprednisolone, enoxaparin, ramipril, carvedilol, spironolactone, and furosemide. One of the suggested mechanisms of PPCM is peri-/postpartum oxidative stress, which may trigger the proteolytic cleavage of prolactin into 16 kDa fragment with potent antiangiogenic, proapoptotic, and proinflammatory properties. This theory is supported by the observation that in animal models of PPCM inhibition of prolactin secretion by bromocriptine prevented the onset of the disease. Prolactin also plays an important role in modulating the immune response and may promote autoimmunity. The role of hyperprolactinaemia in autoimmune diseases, including SLE, is discussed. In our patient hyperprolactinaemia occurring during pregnancy may involve both PPCM and SLE.

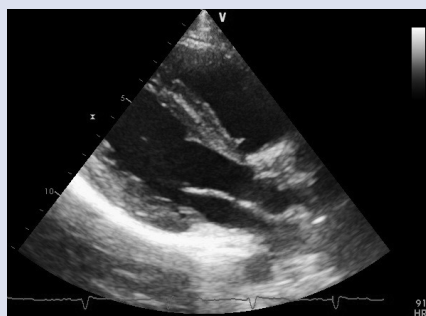


Figure 1. Parasternal long-axis view

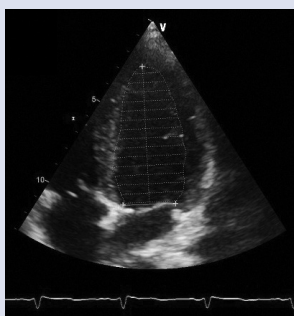


Figure 2. Assessment of ejection fraction with the biplane Simpson's method

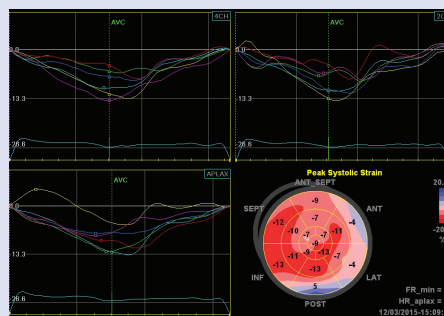


Figure 3. Strain curves and bull's-eye plot of peak systolic longitudinal strain by speckle tracking echocardiography

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**Conflict of interest:** none declared

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