Abstract: P603

Acute inferior ST-elevation myocardial infarction, cerebral ischemic stroke and pulmonary embolism in a patient with thrombophilia

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Introduction: The simultaneous development of acute myocardial infarction, stroke and pulmonary thromboembolism is extremely rare.

Clinical case. A 44-year-old man was admitted with angina-like symptoms, shortness of breath and loss of consciousness. He been suffering from epilepsy since the age of 10, and was under constant medication - taking Oxcarbazepine, Phenobarbital. When he was admitted his condition was extremely acute - sopor, cyanosis, bilateral diffuse wheezing, blood pressure 85/45 mm Hg. ST-elevation in II, III, aVF and an a partial right bundle branch block was recorded on ECG. Hemoglobin level was at 146 g/l, thrombocytes - 132*10^9/l, D-dimer - 46500 ng/ml, LDH - 8244 IU/l, AST - 4273 IU/l, ALT - 2749 IU/l, creatinine - 218 µmol/l, procalcitonin - 3.76 ng/ml, and troponin – 7.1 µg/l. ECHO indicated right ventricular dilatation, pulmonary hypertension 70 mm Hg. Bilateral occlusive thrombosis of the veins of the lower extremities was shown by Doppler ultrasound. An occlusion of the posterior lateral branch of the right coronary artery was shown by coronary angiography, and a stent was performed. A massive pulmonary thromboembolism was shown by computer angiopulmonography. CT brain scan revealed an Ischemic stroke in the terminal branches of the left middle cerebral artery system.

Antithrombin III - 3.4% (normal range is 80%-120%), protein C - 36.8% (70-140%), folic acid - 1.8 ng/ml (3.1-20.5 ng/ml) were found. According to molecular genetic analysis, the patient is a carrier of polymorphisms of thrombophilia genes: MTHFR (C677T), MTHFR (A1298C), SERPINE (5G(-675)4G, ITGA2 (C807T). Mutations of factors V (Leiden), VII, VIII, II, integrin beta-3, fibrinogen; schizocytes and antiphospholipid antibodies were absent. Prothrombin was 78.2% (70%-130%), fibrinogen - 2.9 g/l (1.8-3.5 g/l). The patient received mechanical lung ventilation for 2 days, Dopamine for 18 days, antithrombin III, folic acid, anticoagulant therapy, and dual antiplatelet therapy. The patient improved and was discharged after 45 days.

Discussion. The simultaneous presence of an acute myocardial infarction, cerebral ischemic stroke, and pulmonary embolism was discovered. The most likely cause was the initial congenital deficiency of anticoagulants antithrombin III and protein C, together with the combined effect of genetic polymorphisms, which resulted in the reduced activity of methylenetetrahydrofolate reductase (MTHFR), and also in a reduction of the fibrinolytic activity of the blood (mutation SERPINE1(5G(-675) 4G), and in an increased platelet adhesion rate (mutation ITGA2 (C807T)). Probably, also there was an increased consumption of natural anticoagulants during the thrombosis cascade and a deficiency due to the anticonvulsant drugs, which increased the deficit of active folic acid.

Conclusion. In patients with multifocal thrombosis at a young and middle age, it is necessary to exclude the presence of congenital and acquired thrombophilia.