INTRODUCTION

Pulmonary thromboembolism (PTE) is the obstruction of the pulmonary arterial system by thrombi formed in the venous system and/or the right heart, or rarely, formed in situ. The incidence in the general population is difficult to assess, the venous system and/or the right heart, or rarely, formed in situ. The most frequent causes of PTE in children are: uncommon in the child, being most frequently detected on necropsy (2-4). The incidence in the general population is difficult to assess, the venous system and/or the right heart, or rarely, formed in situ.

CASE REPORT

Patient T.S., aged 17 years, coming from an urban area, was admitted to the Pediatric Clinic I in May 2005 for the confirmation of which cardiac IRM was indicated). On his admission to the clinic, the patient had a good general state, a posttraumatic wound at the level of the nasal pyramid. The evaluation performed in a territorial medical service initially excluded a neurological disorder responsible for the appearance of syncope, the evaluation being subsequently aimed at a cardiac cause. The following were performed: chest radiography, evidencing thickened bronchial walls by vascular mechanism (Fig. 1), confirming in this way pulmonary hypertension detected by echocardiography; electrocardiogram, which showed the presence of RBBB (Fig. 2), and cardiac IRM that allowed the exclusion of tumor masses in the heart cavities, at myocardial or extramyocardial level, refuting in this way the suspicion of right intratral tumor.

At this diagnostic stage, the cardiac causes of syncope (cardiac malformations, cardiac tumors, valvulopathies,
The poor clinical picture regarding pulmonary or cardiac manifestations made the diagnosis of pulmonary thromboembolism difficult; this was established across successive stages, starting from the most frequent causes of syncope at this age, the neurological, cardiac disorders (arrhythmias, congenital or acquired cardiac disease, intracardiac tumor) as well as metabolic disorders being excluded.

Given the positive family history for pulmonary thromboembolism and the presence of mild pulmonary hypertension in the absence of a congenital or acquired cardiac abnormality, paraclinical investigations were subsequently oriented in this direction; pulmonary perfusion scintigraphy showed the suppression of perfusion in the first apical segment of the upper lobe of the left lung. Pulmonary scintigraphy is a basic method in the evaluation of PTE, being used as screening in all cases of uncertainty, as well as for the assessment of posttherapeutic evolution. The combination of perfusion scintigraphy with ventilation scintigraphy is an extremely useful diagnostic modality in PTE, perfusion being affected while ventilation is normal.

Pulmonary arteriography (the gold standard), is a certain diagnostic method, with precise indications and a limited use (6). The new diagnostic methods are represented by MRA (magnetic resonance angiography) and CTPA (CT pulmonary angiography) – which allow the direct visualization of the thrombus. The methods are useful in the case of non-obstructive thrombi, with central location, where pulmonary perfusion can be normal (2,5,6,11). Other diagnostic methods used are: thoracic ultrasound, chest radiography (which provides non-specific data), electrocardiogram, the determination of serum D-dimer concentration (1,2,5,6,12,13).

Thrombophilia is genetically conditioned, its most frequent causes being represented by the Arg506Gln point mutation in the gene of coagulation factor V– Leiden mutation, and the (G20210A) point mutation in the prothrombin gene. In the first case, patients are resistant to the degradation of activated C protein, and in the second, there is an increased plasma prothrombin concentration which results in an increased risk for thrombotic events. Other causes are represented by the deficiency of proteins with an anticoagulant role (protein S, protein C, and antithrombin III), the high levels of factors VIII, IX, and X or of homocysteine. The association of these factors leads to an increased risk for thromboembolism (2,5,6,14-17). The investigations performed in the presented patient allowed to exclude the deficiency of antithrombin III, proteins S and C and the Leiden mutation. The presence of a family history (the mother deceased from PTE) supports a genetically conditioned cause, its determination requiring further investigations that were not available at the time of diagnosis.

The treatment of pulmonary thromboembolism in the child involves specific supportive measures, the administration of anticoagulants (fractioned heparin and warfarin) and thrombolitics (of these t-PA is the most frequently used) – (2, 18). Surgical measures can also be used (thrombectomy and the placement of filters at the level of the inferior vena cava in patients with recurrent PTE) – (1,2,5,6,19,20). The presented patient benefited from anticoagulant therapy with heparin and subsequently Sintrom, in order to prevent recurrences, his evolution being favorable.

**CONCLUSIONS**

Pulmonary thromboembolism is extremely rare in pediatric patients, but it should be suspected in the case of syncope or in the presence of pulmonary symptoms unexplained.
by routine paraclinical examinations.

REFERENCES


