

Case Report





Case Report: Thrombosis in children

Abstract

Venous thromboembolism in pediatrics is constantly increasing phenomenon that causes significant complications and death in children. We present a case that was admitted to our institution, with multiple thrombi in the cardiac apex, massive pleural effusion and thrombosis in both pelvic extremities predominantly on the left, with no family or personal history of diseases, where the presence of laboratory results can be evidenced of the mutation for methyltetrafolate and elevation of hormocystinemia.

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Introduction

Venous thromboembolism in pediatrics is onstantly increasing phenomenon that causes significant complications and death. In children, most thrombocyte events in children are caused by central venous catheters (CVC), and by an acquired cause, many times cardiac.¹

However, cases have been reported where there are congenital deficiencies that contribute to the presentation of thrombosis such as 5,10-methylene-tetrahydrofolate reductase deficiency (MTHFR), which is a genetic disorder that can occur at any age and can be easily detected by an increase in homocysteinemia. In the forms of onset in adolescence or adulthood, the clinical picture is usually complex with the association of various neurological characteristics and thrombosis.²

We present a clinical case of a patient with cardiac thrombi and

extremities who presented altered mettetrafolate.

Case presentation

Admission to the intensive care service of our institution, a 10-year-old female patient who was transferred, due to a 1-month evolution with facial edema and lower limbs, without a family or personal history of diseases, this was exacerbated A picture with thermal rises and respiratory distress, burning chest pain, functional impotence in the lower limbs, an echocardiography was performed that reported multiple thrombi in the apex, massive pleural effusion, an ultrasound that reported hydronephrosis, and also an electrocardiogram that reported supraventricular tachycardia. Physical examination showed intercostal and suprasternal tracing, decreased vesicular murmur, cold edematous lower limbs with cyanosis, weak popliteal pulses, his initial hemogram reported: Hb: 11 mg / dl, gb: 7500, sec: 65%, lif; 28%, mon: 6%, plaq: 135,000 mm³, creat: 1.1 mg / dl, urea: 44 mg / dl, cholesterol: 149 mg / dl.^{3,4}



Figure I Homozygous for methyltetrafolate allelo.





Figure 2 Occlusion of the right iliac artery, with partial recanalization of the femoral bones.

She required vasoactive amines and mechanical ventilation due to the neurological deterioration, the thrombosis in the left ventricle was confirmed, due to data on a systemic inflammatory response, antibiotic coverage with cefortaxime and cloxacillin was started, treatment with sodium heparin was started for the thrombi.

During hospitalization, a new echocardiography shows an increase in the size of the thrombi, some stuck to the left ventricle, thinking about the risk of causing bacterial endocarditis, Amikacin is started, an angiotac was also performed that reported totally decreased arterial circulation in both pelvic extremities a left predominance compatible with a thrombosis. It was evaluated by multiple specialties Rheumatology, Immunology in search of multiple causes and laboratories are requested which reported: c3: 204 mg / dl, c4: 76 mg / dl, Hormocysteine: 14.2 umol / L, Anti DNa Ds: 10 IU / ml ,, ENA complex (SSA, SSB, SM, SM / RNP, SCL-70, JO-1) Non-reactive, Ancap: 0.33U / ml, Anca C; 0.31 U / ml,, TP: 12 sec, TPTA: 23 sec, Fibrinogen 371 mg / dl,, Ana: 0.32 U / ml. Negative for Protein C, S and antithrombin, Methyltetrafolate Polymorphism: present. 5-7

Conclusion

When these findings were evidenced, it was concluded that he had an acquired cardiac pathology factor and thus also has a genetic factor such as the alteration of methyltetrafolate, which is why it was decided to maintain anticoagulation indefinitely and to manage with folates.

The patient remained in intensive care for 20 days, later she was referred to the Pediatric service, currently her controls are carried out by the hematology service, the patient is stable on indefinite treatment with warfarin and folates.

Acknowledgments

None.

Conflicts of interest

The authors declare no conflicts of interest.

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