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Case Report

Cerebral Thrombosis due to « Protein S » Deficiency: About a Case

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Abstract: Thrombosis of the dural venous sinus or cerebral veins is a complete or partial occlusion that can affect one or more main sinuses. Involvement of the feeding cortical veins can lead to heterogeneous manifestations (headaches, altered consciousness, behavioral abnormalities, convulsions, speech difficulties and focal or generalized neurological deficits). The incidence of CVT is estimated at 5 per 1million. Venous thromboembolism [TE] is a multifactorial disease, and protein S deficiency [PSD] constitutes a major risk factor. Treatment of CVT with Heparin followed by VKA is recommended by current guidelines and should be started as soon as the diagnosis of CVT is confirmed we will report the case of a 17-year-old male with a medical history of anemia that was admitted in our ICU ward for a cerebral veinous thrombosis complicated with thrombo-embolic pattern. The initial treatment of TE followed the treatment guidelines of the American society of stroke. However, there are no guidelines of long-term treatment in children with PS deficiency. The actual recommendations didn't state the best moment to introduce an anticoagulant.

Keywords: Cerebral Venous Thrombosis (CVT), Protein S Deficiency (PSD), Venous Thromboembolism (VTE), Anticoagulation Therapy, Dural Venous Sinus.

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INTRODUCTION

Thrombosis of the dural venous sinus or cerebral veins is a complete or partial occlusion that can affect one or more main sinuses. Involvement of the feeding cortical veins can lead to heterogeneous manifestations (headaches, altered consciousness, behavioral abnormalities, convulsions, speech difficulties and focal or generalized neurological deficits) [1]. The incidence of CVT is estimated at 5 per 1 million [2].

Venous thromboembolism [TE] is a multifactorial disease, and protein S deficiency [PSD] constitutes a major risk factor [3].

Treatment of CVT with Heparin followed by VKA is recommended by current guidelines and should be started as soon as the diagnosis of CVT is confirmed [4].

CASE REPORT

We will report the case of a 17-year-old male with a medical history of anemia that was admitted in our ICU ward for a cerebral veinous thrombosis complicated with thrombo-embolic pattern.

The clinical exam at the admission found a patient that is intubated with anisocoria The blood simples showed an anemia with HB = 4,5, a TCA ratio at 0,8. An MRI was performed (fig1) that showed a venous thrombosis in the transverse and sigmoid sinuses complicated by an infarction with cerebral oedema.

Our strategy was to introduce heparin using an electric pump and gradually increase the posology from 300ui/kg/d to 500ui /kg/d for a TCA ratio>1. optimizie hemoglobin and manage the oedma with mannitol.

During his early 48h of hospitalization he presented other thrombo-embolic complications such as pulmonary embolism, brachial and cephalo-brachial veins thrombosis.

During his hospitalization we performed a Serie of etiological exams:

The antiphospholipid, anti BETA2gp1, anti cardiolipins, and lupus checkup came back negative. The antigliadin blood checkup was also negative. Homocysteine and vitamin B12 screening came back normal.

A COVID-PCR was performed and came back negative.

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A thrombophilia checkup came back with a protein S rate at only 39% the rest of the checkup was normal.

Referring to our finding we decided to introduce the acenocoumarol for a target IRN (2-3)

The patient is weaned from mecanical ventilation with a Glasgow score 15/15 and no sequeals. He is discharged from our ward.

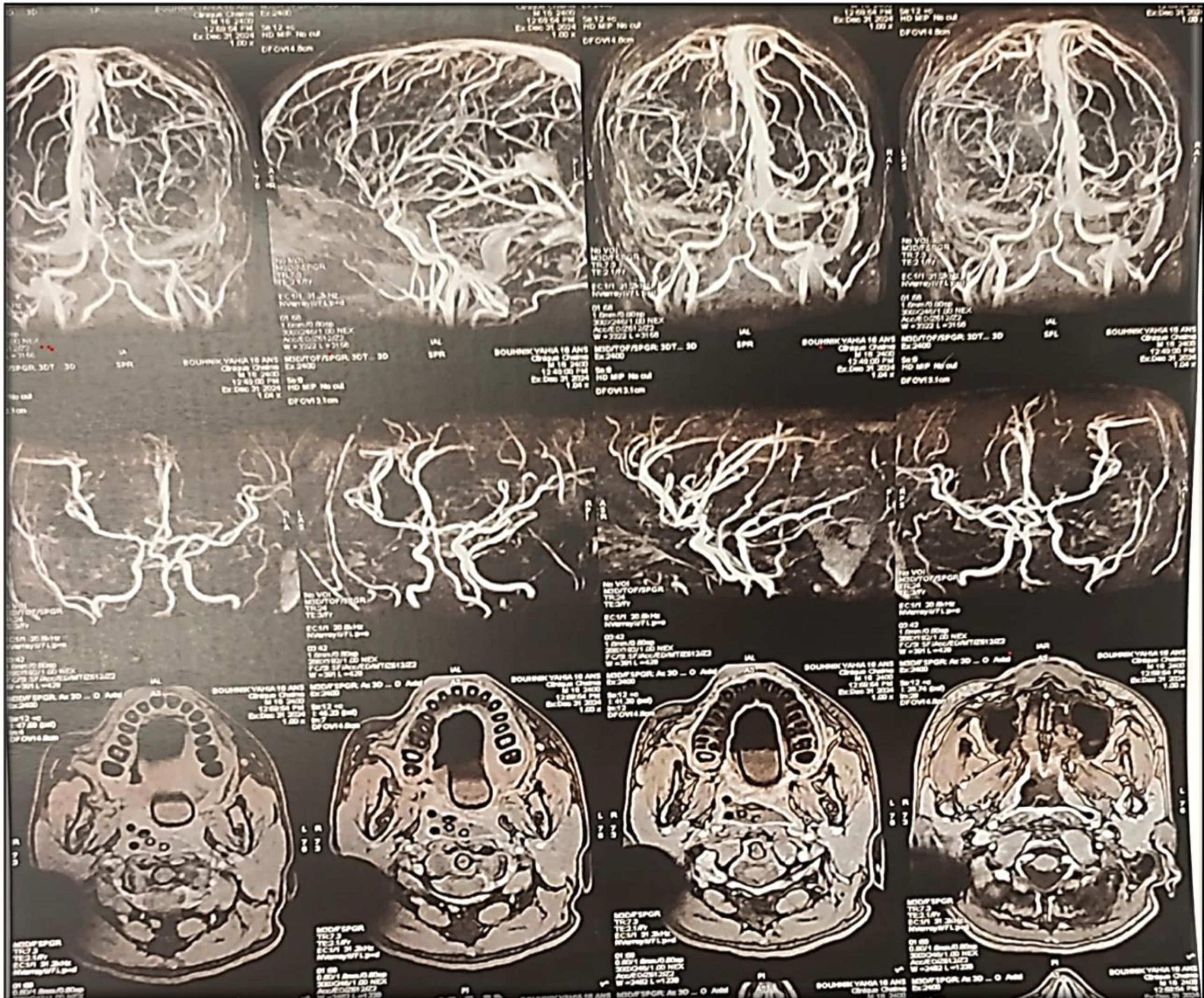

Fig. 1: MRI results chowing an extended transverse thrombosis to the sigmoid sinus

DISCUSSION

The currently most commonly tested hereditary thrombophilia include deficiencies of antithrombin, protein C, or protein S and the gain-of-function mutations FVL and PGM. Lupus anticoagulants, anticardiolipin antibodies, and anti–β2-glycoprotein 1 antibodies [5]. In our case it was due to protein S.

The initial treatment of TE followed the treatment guidelines of the American society of stroke [4]. However, there are no guidelines of long-term treatment in children with PS deficiency.

Therefore, long-term anticoagulants should be adjusted according to each patient's needs. We could relate this to the multiple genotypes of Protein S deficiency.

The existing treatment guidelines provided by the American Heart Association-American Stroke Association (AHA-ASA) and European Stroke Organization (ESO) recommended initial systemic heparinization using either unfractionated or LMWH, followed by long-term oral anticoagulation with warfarin. However, the actual recommendations didn't state the best moment to introduce an anticoagulant [6]. In our case, we introduced in the early 24 h of the diagnosis.

CONCLUSION

Protein S deficiency is a major risk factor for CVT. Early introduction of anticoagulants is recommanded. However, the guidelines neither state lenght of treatment in children with PS deficiency nor state the best moment to introduce an anticoagulant. Further more studies are required in order to defines those points.

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