A CASE OF CEREBRAL VENOUS THROMBOSIS IN A PATIENT ON ESTROGEN-PROGESTIN THERAPY WITH A MILD THROMBOPHILIC PATTERN: THERAPEUTIC MANAGEMENT AND FOLLOW-UP

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AIM

We aim to describe a case of cerebral venous sinus thrombosis (CVST) in a patient with a mild thrombophilic condition.

PATIENT AND METHODS

We report the case of a 22-year-old woman with a history of migraine and polycystic ovary syndrome, treated with an oral estrogen-progestin contraceptive for the previous 11 months, who presented to the emergency department with a severe headache unresponsive to analgesic therapy. A brain CT scan, MRI, and laboratory tests were performed.

RESULTS

Neurological examination and routine blood test, including coagulation parameters, were normal. Brain CT revealed a hyperdense appearance of the cerebral straight sinus. (Figure 1). MRI (1.5T) showed the thrombosis in the straight sinus. (Figure 2).

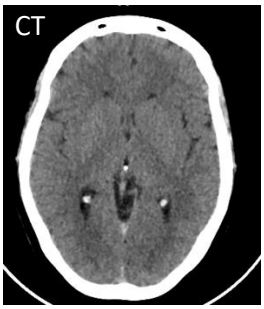

Figure 1

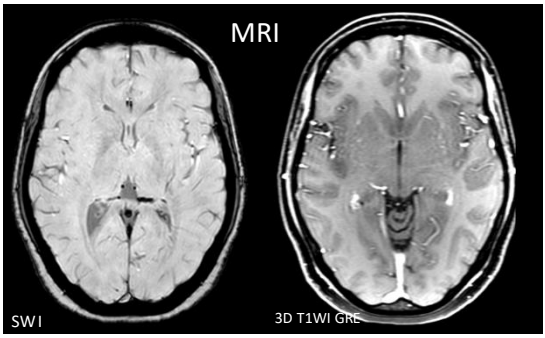

Figure 2

The patient was treated acutely with low-molecular-weight heparin (LMWH) at a therapeutic dose and was discharged on warfarin, targeting an INR of 2-3. A follow-up MRI performed one month later showed the presence of flow in the straight sinus. Thrombophilia screening, performed during the hospitalization, revealed a heterozygous mutation of factor II, a reduced level of protein S (40, reference range 54-123), and a heterozygous MTHFR gene mutation. Further follow-up MRIs at 5 months and 1 year confirmed patency of the cerebral venous sinuses. After 1 year, anticoagulant therapy was transitioned, in agreement with the hematologist, to prophylactic acetylsalicylic acid (100 mg/day), with an indication to use LMWH prophylaxis during future periods of increased thrombotic risk. At one year of follow-up, the patient has had no relapse.

DISCUSSION AND CONCLUSIONS

The risk of CVST is higher in women who use oral contraceptives [1], and this risk is further increased in patients with a thrombophilic pattern. Current guidelines recommend continuing anticoagulant therapy indefinitely in patients with severe thrombophilia (e.g., homozygous prothrombin gene G20210A variant, homozygous factor V Leiden genetic variant, deficiencies of protein C, protein S, or antithrombin, combined thrombophilia defects, or antiphospholipid syndrome) [2,3]. However, no clear recommendations exist for patients with a non-severe risk profile. This case highlights the importance of identifying triggering factors and adopting a personalized approach to the duration and type of antithrombotic therapy in low- to moderate-risk patients.

REFERENCES

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