Abstract

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Management of venous thrombosis therapy in atypical site: a complex case of lumbar puncture complicated by cerebral venous sinus thrombosis

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Background

Cerebral vein thrombosis (CVT) is a manifestation of unusual site venous thromboembolism (VTE). CVT encompasses thrombosis of the cerebral veins (cortical or deep veins) and dural venous sinuses, has an estimated annual incidence of 3 to 4 cases per million adults, and is the cause of 0.5–1% of strokes. CVT usually shows a clear prevalence of females (approximately 75% of cases). Among the main conditions favouring CVT are thrombophilia (about 38% of cases), oestrogen-progestin therapy (about 33% of cases), pregnancy/puerperium, cancer, and myeloproliferative syndromes. Local risk factors for CVT have also been reported, such as mechanical causes (e.g., head injury, central venous catheter placed into the jugular vein, or neurosurgical procedures, reported in 3–5%) or infections (e.g., otitis, mastoiditis, meningitis, reported in about 10% of cases). Early diagnosis and adequate anticoagulant therapy represent the main challenges for the clinician, to avoid long-term neurological sequelae.

Case report

a 28-year-old woman, admitted for severe headache, not responsive to NSAIDs, and left weakness, underwent MR of the brain with evidence of thrombosis of the superior longitudinal sinus, the bilateral sigmoid sinus, associated with infarction in the right fronto-parietal white matter, with evidence of abnormal impregnation of the walls of the sagittal sinus (characteristic sign of the "delta"), straight sinus and part of the transverse.

Approximately three months before she reported fleeting episodes of diplopia and concomitant finding of bilateral papilledema at an ophthalmological evaluation. At that time, the neuroradiological findings, including MR angiography, were negative and a diagnosis of pseudotumor cerebri was therefore made. Interestingly, she had performed lumbar puncture (LP) with cerebrospinal fluid pressure measurement about two weeks before admission, which showed a value of 340 mmH2O. After the LP she had presented exacerbation of the headache. Pulsating headaches were also reported in previous years. She had never taken oestrogen-progestin therapy. Laboratory tests revealed thrombocytosis (643,000/mL). The search for the V617F mutation on the pseudokinase domain (JH2) of the JAK-2 gene was positive and, following specialist haematological evaluation, the diagnosis of JAK-2 positive essential thrombocythemia was done. The laboratory investigation also revealed the presence of the G20210A mutation of the prothrombin gene in heterozygosity. Treatment with warfarin (target INR 2-3), oncocarbide, and acetazolamide, was started. The patients stopped warfarin after some months, due to warfarin resistance, and rivaroxaban 20 mg daily was started and continued for long term, without major/minor bleedings and without recurrences.

Conclusions

CVT after LP is a well-known phenomenon, and a CVT should be suspected in any patient who undergoes LP and develops refractory and transformed headache. The guidelines for the treatment of CVT suggest a therapeutic conduct like that established for VTE in typical sites: oral anticoagulants for at least 3 months, to be maintained for long term in patients with recurrent thrombosis or with conditions at high risk of recurrences such as in the case of the patient described. There is growing evidence on the use of DOACs in CVT. In this case, the patient was treated with rivaroxaban 20 mg for about 3 years, without bleeding complications or thrombotic recurrences.