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Case Report

Cerebral Venous thrombosis in SS homozygous Sickle Cell Disease Associated with Hypothyroidism: a Case Report

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Abstract

Cerebral venous thrombosis (CVT) is a rare form of stroke that affects the venous system. It's a multifactorial condition, which may be triggered by hemoglobinopathies such as sickle cell disease (SCD). We report the case of a SCD patient, admitted in the Internal Medicine department for progressively worsening headaches associated with generalized seizures. The diagnosis of superior sagittal sinus thrombosis associated with hypothyroidism was made. The clinical outcome was favorable with anticoagulation. This case highlights the importance for clinician to not overlook SCD, and hypothyroidism as relevant etiological factors of CVT.

Kew Words: cerebral venous thrombosis; homozygous; sickle cell disease; hypothyroidism

Introduction

Cerebral venous thrombosis (CVT) is a form of stroke affecting the venous system, particularly dural venous sinuses. It represents 0.5 to 1% of all stroke cases and mainly affects young adults, especially women with prothrombotic conditions [1]. Many conditions may contribute to the occurrence of CVT, such as clotting and endocrine disorders, and hemoglobinopathies like sickle cell disease (SCD)[2,3]. The clinical presentation of CVT is polymorphic, depending on the location and extension of the thrombosis. The most frequent symptoms are headaches (80 to 90% of cases), associated with visual disturbances, seizures, motor or sensitive deficits, altered mental status and/or meningeal syndrome [4]. Some recent studies suggest the implication of hypothyroidism and SCD, in the development of clotting disorders as well as thromboembolic diseases [5,6]. We report a case of CVT in a SCD patient who also had hypothyroidism, illustrating complex etiopathological mechanisms.

Case report

A 50 years old woman with SS homozygous SCD diagnosed in her childhood, but lost to follow-up, was admitted in the internal medicine department of the Garoua general hospital. She presented with worsening headaches, associated with a generalized tonic-clonic seizure which occurred one day before admission. The history revealed that the patient had received a blood transfusion for anemia, five days earlier. Physical examination showed left facial palsy and a left pyramidal syndrome with 4/5 muscle power. A head CT-scan was done showing diffuse cortical atrophy. We pushed the investigations further by performing a magnetic resonance angiography which showed a thrombosis of the superior sagittal sinus **(figure 1)**. Laboratory workups showed normocytic normochromic anemia with 8.5 g/dl of hemoglobin. Basic hemostasis tests were normal. HIV and hepatitis B and C serologies were negative. Serum creatinine was 169mg/dl (glomerular filtration rate = 41.31ml/mn/1.73m^2) and the natremia was 159mmol/L. Thyroid function tests revealed a hypothyroidism with a TSHus of 1.008 UI/mL, FT4 of 1.61pmol/L and FT3 of 9.881pmol/L. Thrombophilia and immunological investigations were not done because of timing and logistical constraints. The patient was treated with low molecular weight heparin switched with acenocoumarol (target INR 2-3), carbamazepine 200mg bid, nefopam 10mg tid, levothyroxine 25µg od, and hydration with crystalloids (1500mL/day). The outcome was favorable with amendment of headaches, seizures, and normalization of kidney function and natremia.

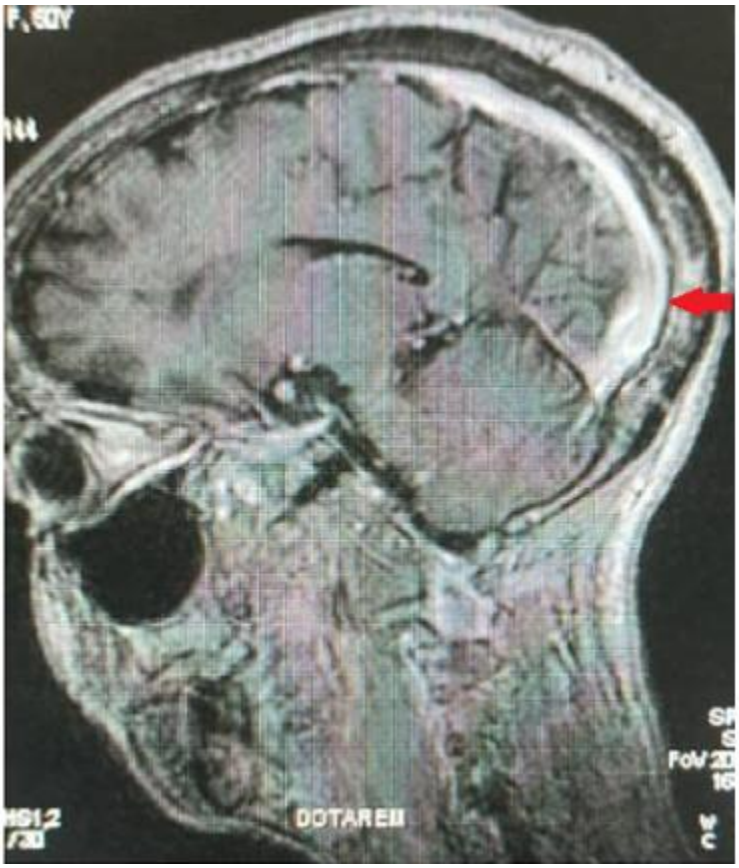

Figure 1: Cerebral magnetic resonance angiography showing the thrombosis of the superior sagittal sinus (red arrow)

Discussion

The clinical spectrum of CVT is large. The classic presentation includes signs of raised intracranial pressure, focal neurologic deficits and/or seizures. All these signs were present in the case of our patient. CVT is a multifactorial disease, which can result from as many risk factors as hereditary thrombophilia, oral contraceptives, pregnancy and post-partum period [4,7]. Some studies have also showed the involvement of SCD in clotting activation. In fact, SCD patient exhibit low plasmatic level of C and S proteins, while there is an increase of tissue factor expression and thrombin formation [6]. These patients also present moderate thrombocytosis due to functional asplenia, and increased platelet aggregation [6]. Other evidence reports increased adhesion of sickle red blood cells to the endothelium [8]. Recent studies have reported cases of hypothyroidism in patients affected by CVT [9,10]. Hypothyroidism may promote a hypercoagulable state by decreasing fibrinolysis through high level of alpha 2-antiplasmin and plasminogen activator inhibitor-1. The rare association of SCD and hypothyroidism inexorably leaded to the pathogenesis of CVT in this patient.

Conclusion

Cerebral venous thrombosis is a rare disease, with multiple causes, among which sickle cell disease and hypothyroidism should not be overlooked. They play an important role in alteration of the clotting cascade. Consequently, these etiologies should be investigated in all cases of CVT.

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