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Case report

Non-thrombotic vertebrobasilar stroke in Crohn's disease patient.

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Abstract

Crohn's disease is a chronic inflammatory bowel disease (IBD) that could affect any part of the gastrointestinal tract. The association between posterior circulation stroke and Crohn's disease is rarely reported and was controversial for long time. We report herein a case of a stroke in the right posterior inferior cerebellar artery (PICA) territory in a male patient with no cardiovascular risk factors who was monitored for ileocolic active Crohn's Disease.
The aim is to assess the pathophysiological correlation between this inflammatory condition and the predominance of vertebrobasilar strokes and to highlight appropriate management characteristics.

Keywords:
Crohn's Disease; stroke; thrombus; Hypercoagulability.

Introduction

Several concordant studies have ruled out significant increased thromboembolic accidents risk in Inflammatory bowel disease. However, the mechanisms are still unclear specially regarding the contribution of the systemic inflammation itself to the coagulability disorders [1].

Observation

We report a case of 53-year-old Caucasian male smoker with no other cardiovascular risk factors presenting to the emergency department for rotational vertigo associated with gait and postural instability that started four days ago. The patient had a 10-year history of ileocolic Crohn's disease irregularly followed up. The physical examination revealed discrete ataxia of the right lower and upper limb, loss of pain and temperature sensation on the left side of the body sparing the face, abolition of the gag reflex, oropharyngeal muscle paralysis with uvular deviation to the left, dysphonia, and a right-sided Bernard-Horner syndrome. The Diagnosis of a complete Wallenberg syndrome retained with NIH stroke scale of 5. Brain MRI revealed a right paramedian pontine stroke affecting the lower side of the right cerebellum, consistent with the territory of the right posterior inferior cerebellar artery (PICA) visible in the FLAIR sequences. No arterial occlusion or thrombus were detected on the T2-star and 3D-TOF sequences (figure1a, 1b).
The 24-hour Holter monitoring, transthoracic and transesophageal echocardiography, CT brain angiography and Doppler ultrasound of the neck vessels were normal. Biology revealed inflammatory syndrome with no hyperleukocytosis or thrombocytosis. CRP was 252 g/L and fibrinogen 5.58 g. The plasma protein electrophoresis had an inflammatory distribution. Lipid panel showed high levels of cholesterol. There was no diabetes. Serology for HIV, and hepatitis B, C were negative. Anti-cardiolipin antibodies and anti-beta2-glycoprotein antibodies (b2GP1) also revealed negative results. Antiphospholipid syndrome (APS) workup was negative along with complement levels and ANCA. Vitamin B9, B12 and homocysteine levels were within normal. The test for arterial thrombophilia turned out to be negative (no Factor V mutation, no prothrombin gene mutation and no protein S and C deficiency). However, a high level of Factor VIII (295.9%) and Factor V (210%) were noted. Abdominal CT scan and colonoscopy confirmed colonic active Crohn's disease. Antiplatelet aggregation and corticosteroid therapy were prescribed. The neurological evolution was satisfactory following functional and motor rehabilitation, reaching an NIHSS score of 1 and a Rankin score of 1. Only minimal residual ataxia persisted on the right side of the body. Treatment with ani-TNF alpha was prescribed for the Crohn's disease.

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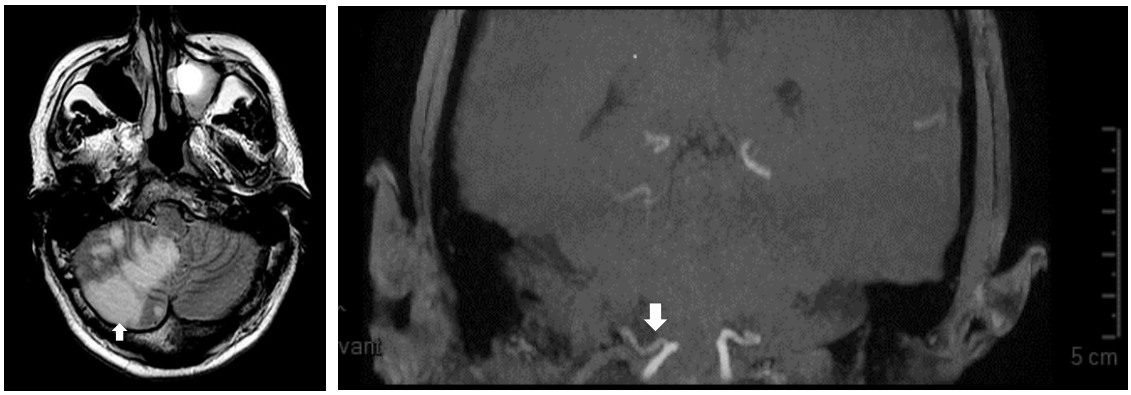

Figure 1: a-paramedian pontine stroke affecting the lower side of the right cerebellum
b- No evidence of arterial PICA thrombus on the T2-star and 3D-TOF sequences

Discussion

Several studies in the literature reported significant association between chronic inflammatory bowel disease (IBD) and thrombo-embolic accidents [2]. Posterior circulation strokes in Crohn disease patients are uncommon. Posterior circulation strokes could be explained by a slower blood flow creating a high hemodynamic turbulence (hyperviscosity) and the loss of autoregulation of the smooth muscles of arteries media [3].

Our patient presented a significant increase in Factor VIII, Factor V, CRP and fibrinogen which could generate a different coagulability status providing favorable field to the stroke development. Several studies noted higher level of coagulation factors in IBD cases. These patients may be susceptible to experience posterior stroke due to high Factor VIII level. This factor is an anti-inflammatory marker and prothrombotic protein. Other authors found that Von Willebrand factor drives the association between elevated factor VIII and poor outcomes in patients with ischemic stroke. However, the exact physiopathology is still unclear in these cases [4,5]. The contribution of some other blood disorders like thrombocytosis or hyperhomocysteinemia was reported. IBD integrating bigger inflammatory syndromes including vasculitis and vascular wall diseases may constitute a bias of the cause-to-effect association of the systemic inflammation and the thromboembolic disease [6]. Diagnosis of non-thrombotic stroke requires multiple investigations including anti-phospholipid antibodies (APA), anti-cardiolipin antibodies, anti-beta2-glycoprotein antibodies (b2GP1), ANCA, homocysteine, Antithrombin III, Protein C, Protein S, Factor V Leiden and Prothrombin II assessment. Small atheromatous plaques must be ruled out on the imaging as well. The angio-MRI 3D reconstruction of cerebral vessels is the standard diagnostic tool. The management of non-thrombotic strokes is still non-consensual. In our practice we believe that antiplatelet aggregation agents are efficient and safe. The control of the systemic inflammation and Crohn disease remission may decrease the risk of stroke recurrence.

Conflict of Interest: None

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