

Deep Cerebral Venous Sinus Thrombosis (Cvt) with Excellent Recovery in HIV Patient on Antiretroviral Therapy

Ishraga Elamin*

Department of Internal Medicine, King Abdullah Medical City, Kingdom of Saudi Arabia

*Corresponding author: Ishraga Elamin, Department of Internal Medicine, King Abdullah Medical City, Kingdom of Saudi Arabia, Tel: 00966534020944; E-mail: ishragaelwasila@yahoo.co.uk

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Abstract

Deep cerebral venous sinus thrombosis occurs in HIV patient with normal CD4 count. We report a patient who developed CVT while on reverse transcriptase inhibitors for the last four years, with low protein S level.

Keywords: Deep cerebral venous sinus thrombosis; HIV patient; Reverse transcriptase inhibitors

Introduction

Cerebral venous sinus thrombosis (CVST) is the presence of blood clot also called as acute thrombosis in the dural venous sinuses. Here the case of 40-year-old male has been described who was diagnosed with HIV and deep cerebral venous sinus thrombosis which was treated with antiretroviral therapy.

Case Presentation

This is a 40-year-old male, diagnosed with HIV 4 years ago, on antiretroviral therapy (Efavirenz 600 mg daily and Emtricitabine/tenofovir 200/245 mg daily). He had 5-months history of headache and mild unsteadiness, not associated with nausea or vomiting. He presented on 15th of September 2016 to a peripheral hospital with recurrent prolonged seizures. The seizures were aborted with Benzodiazepine. The patient was intubated, sedated and transferred to Neuro critical care unit at King Abdullah Medical City. Upon arrival, his neurological examination was limited due to full sedation but noted to have bilateral papilloedema and no obvious localizing signs or meningeal irritation (Figure 1). Plain CT brain showed a fairly symmetrical bilateral basal ganglionic and thalamic hypodensity.

MRI brain on admission showed bilateral thalamic and basal ganglion hyperintensity on Flair, T2 and DWI, T1 showed signal hypointensity of deep cerebral veins, vein of Galen, and straight sinus, T1 post gadolinium showed filling defect of straight sinus and internal cerebral veins. Feature of obstructive hydrocephalus with dilatation of both lateral ventricles with periventricular hyperintensity secondary to the mass effect of the oedematous thalami (Figures 2 and 3).

Based on these findings leviteracetam was initiated, external ventricular drainage (EVD) was inserted, heparin IV infusion was started, and patient was taken to angio-suit for emergency thrombectomy. Thrombectomy of straight sinus thrombus was carried out with recanalization of about 80% of the straight sinus up to the vein of Galen, Heparin was eventually replaced by Warfarin.

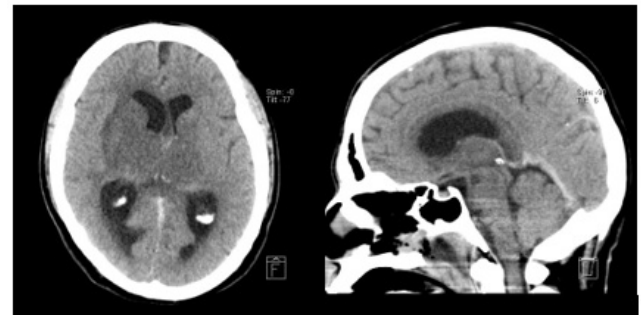


Figure 1: Plain CT brain on admission.

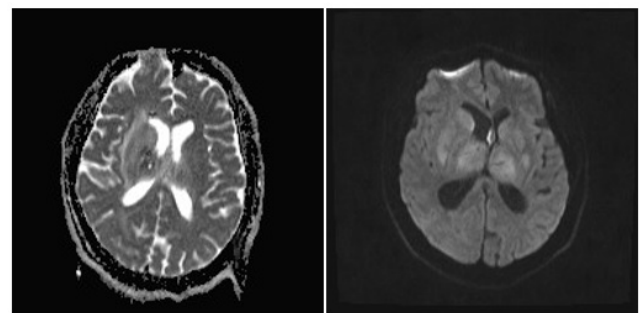


Figure 2: MRI/V brain with and without gadolinium on admission.

Laboratory investigations revealed: Hb 7.9, WBC, platelets, ANCA, ANA, syphilis, anticardiolipin, anti Ro, La, B12 were all normal or negatives. Low vitamin D level of 12.9. Factor V: 121.4 (70-120), Antithrombin 3: 121 (80 -120), Protein S was low at 62 (70-140), Prothrombin 2: 148 (70-140), factor 2 Prothrombin G20210A and factor V Leiden mutation were all negative. HIV viral load was 371 copies/ml and CD4 count was 559.

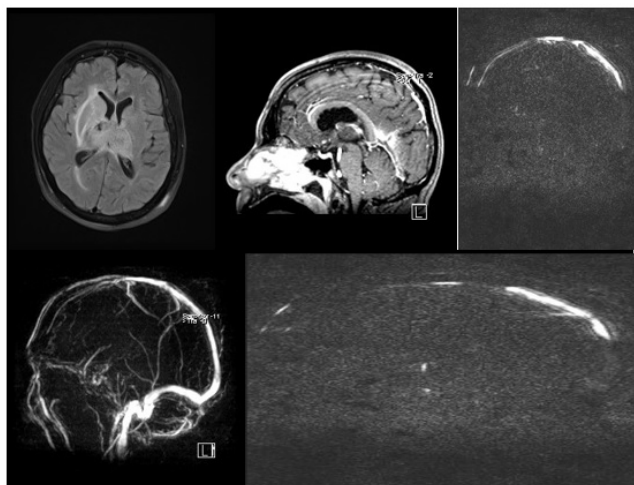


Figure 3: Findings leviteracetam.

The patient gradually regained his consciousness but with left side weakness. EVD was removed after a week and his muscle power on the left side improved with physiotherapy. He was discharged home on leveteracetam 1500 mg BID and Warfarin in addition to his antiretroviral therapy (Figure 4).

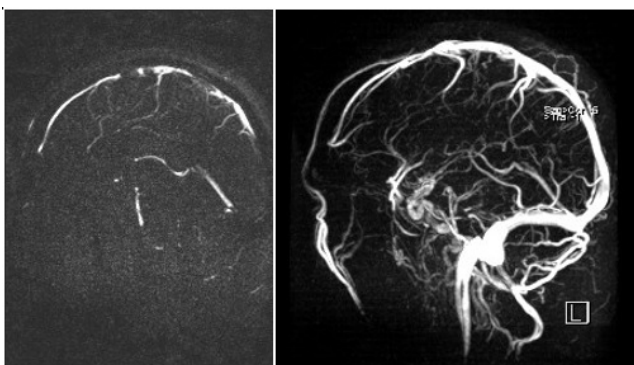


Figure 4: Follow up MRI brain with contrast and MRV 7 weeks following initial presentation.

Discussion

Cerebral venous sinus thrombosis (CVST) is a potentially life-threatening condition representing 0.5% of all strokes [1,2]. Though CVST were not frequently reported in HIV patients in the literature, HIV increase the risk for cerebrovascular disease. 85% of cerebral venous sinus thrombosis has prothrombotic risk factor [3]. By enlarge; CVST could be due to hypercoagulable diseases, inflammatory and infectious diseases including dental abscesses, otitis media, mastoiditis, facial infections, and endocarditis [4,5].

In HIV patients, CVST has been associated with aberrancies in factors essential in the coagulation pathway. In a study of 109 HIV-infected patients, venous and/or arterial thrombosis etiology was: protein C deficiency, elevated factor VIII concentrations, high

fibrinogen concentrations, and free protein S deficiency [6]. HIV infection is also associated with an elevated level of D-dimer, along with other markers of dysregulated coagulation and fibrinolysis, which indicates that HIV infection could be associated with a pro-thrombotic state [6].

Furthermore, mechanism for the noted hypercoagulability in patients with HIV infection includes the presence of, heparin cofactor II, antithrombin. And antiphospholipid antibodies. A few studies highlighted a high prevalence of antibodies against protein S in HIV infected patients, leading to significantly low protein S activity in about 31%-76% of patients [7]. It has been noticed that protein S deficiency is not correlated with severity of HIV disease. Moreover, it seems that thrombosis is highly related to low CD4 counts [7,8], which is not the case in our patient. However, protein S level in our case is mildly low at 62 (70-140) but unfortunately protein S activity was not checked.

Konin et al. reported two cases of venous thrombosis associated with protein S deficiency in HIV patients. In the first case they reported superior mesenteric vein thrombosis in 53-year-old HIV patient with protein S deficiency. The second one was a case of cerebral venous sinus thrombosis in HIV patient who was in his third decade. Both of them were not yet on antiretroviral therapy [9].

In spite of the fact that the absolute risk of thrombosis in patients who are not on combined antiretroviral therapy is almost 6 folds in comparison to healthy population of the same age, there is an additional risk of thrombosis in patients on HAART [10,11]. The prothrombotic effect of HAART is reported to be more common in combinations including protease inhibitors (PIs) [12,13]. Protease inhibitors based therapy promotes thrombosis by inducing platelets and endothelial dysfunction [13]. Nonetheless it is still unclear whether non-protease inhibitors are prothrombotic or not [11,14]. Our patient is on reverse transcriptase inhibitors.

Conclusion

In this HIV patient with normal CD4 count and deep cerebral venous sinus thrombosis, the underlying cause can be possibly in part related to his relatively low protein S and to his treatment with reverse transcriptase inhibitors? However, the possibility of other factors leading to CVT is still open and further case reports might help address this issue.

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