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HEPETATIS C VIRUS A RARE CAUSE OF CEREBERAL VENOUS SINUS THROMBOSIS

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ABSTRACT:

Cerebral venous sinus thrombosis is challenging condition because of its variability of underlying pathological causes. Numerous conditions can cause cerebral venous sinus thrombosis (CVST), and often more than one cause can be found in an individual patient. The causes leading to CVST can be classified into two broad categories; infective and non-infective. Among infective causes, chronic hepatitis C infection is considered as one of the extremely rare causes of cerebral venous sinus thrombosis.

Keywords: Hepatitis C Virus, cerebral venous sinus thrombosis

INTRODUCTION

Cerebral venous sinus thrombosis is one of the rare condition which nowadays is being diagnosed more frequently due to greater awareness and availability of better diagnostic modalities. Predisposing factors can be identified in up to 80% of the patients. [2] The main predisposing conditions causing cerebral venous sinus thrombosis are either infectious (causing 8% of cases in a series) or noninfectious. [2] Among non-infectious causes, the connective tissue disorders, granulomatous or inflammatory disorders and malignancies are the most frequent causes. [3] In addition 20% to 35% of the cases have no identifiable cause. [4] We report case of a 32-year-old male with chronic hepatitis C virus infection as an infrequent cause of cerebral venous sinus thrombosis. [5-7]

CASE REPORT

A 32 years old male patient presented with history of headache for less than 2 weeks, history of one episode of transient blurring of vision 1 week ago. Afterward he had an episode of generalized tonic-clonic seizure followed by development of left hemiparesis. There was no history of hypertension, diabetes mellitus, or drug intoxication. He is a known case of Hepatitis C virus for 3 months and is on anti-viral therapy Sofosbuvir and Velpatasvir for the past 3 months. On presentation his GCS was 8/15 (E3, M4, V1) with left sided weakness and a power of 3/5 in both upper and lower limbs. Cranial nerves and fundoscopy was normal. There were no signs of chronic liver disease on examination. His cardiovascular and respiratory examination was unremarkable. The initial laboratory tests including hematology, biochemistry and coagulation profile was normal. CT- brain was unremarkable. The initial laboratory tests including hematology, biochemistry and coagulation profile was normal. CT- brain was unremarkable. Magnetic resonance imaging (MRI) brain with Magnetic resonance venography (MRV) was done. MRI showed an infarct in right high parietal area (Figure. 1). MRV revealed superior sagittal sinus thrombosis (Figure 2). Antinuclear antibody (ANA) test was sent which came out negative.

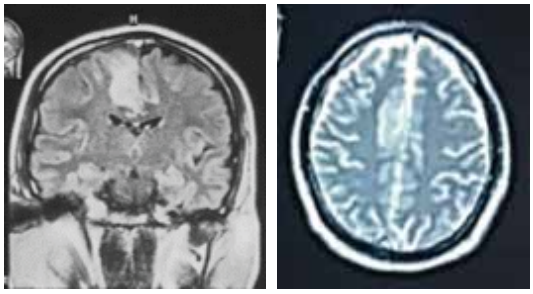

Figure 1. MRI brain plain.
Coronal FLAIR (A) and T2 weighted Axial (B) images showing hyper intense signal in right high Parietal parafalcine region.

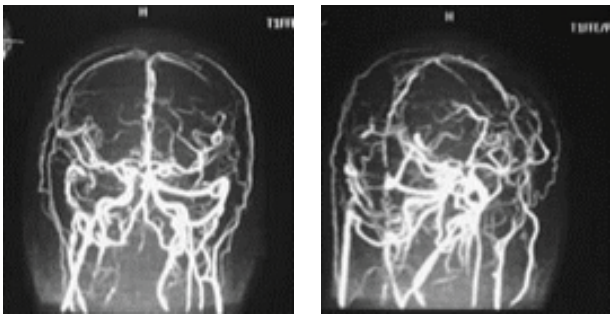

Figure 2. MRV brain.

MRV shows signal void in middle part of superior saggital sinus.

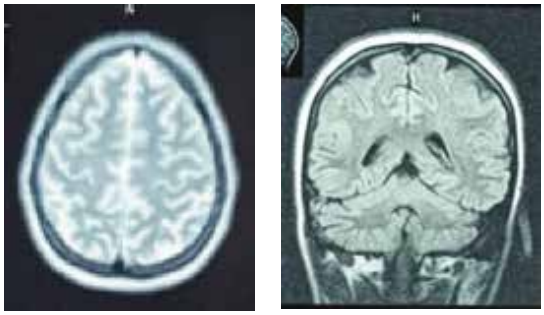

Figure 3. MRI brain plain.

T2 weighted Axial (A) and Coronal FLAIR (B) images showing resolution of lesion post therapy.

He was started treatment with anti-epileptics, anticoagulation and hydration. HCV PCR was repeated which was negative and antiviral drugs were stopped. Rivaroxaban was started in a dose of 15mg twice a day, which was later on switched to 20mg once a day after 3 weeks. With initiation of treatment dramatic improvement was seen, GCS improved to 15/15 and power of left side of body improved to 4/5 in both upper and lower limbs. After completion of six months of treatment with Rivaroxaban his neuroimaging was repeated. MRI brain with MRV scan which showed optimal visualization of superior sagittal sinus and no evidence of dural sinus thrombosis (Figure 3 and 4).

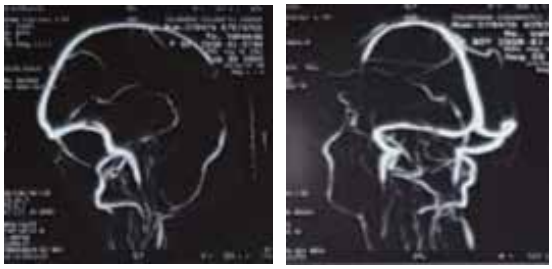

Figure 4. MRV brain.

MRV shows normal flow in all sinuses post therapy.

Thrombophilia workup turned out to be negative including Lupus anticoagulant, Factor V Leiden, Anti thrombin-III, Protein C and S and PCR for thrombin Gene mutation (G20210A) (Table 1). His general and neurological examination after six months of treatment was unremarkable with full recovery of left sided body weakness.

TESTS	PATIENT'S RESULTS	NORMAL VALUE
Anti Cardiolipin Antibodies IgG	2	<10.0 GPL-U/ml
Anti Cardiolipin Antibodies IgM	1	< 7 PL-IgM-U/ml
Anti beta2 glycoprotein 1 Antibody IgM	1	<10 U/ml
Anti beta2 glycoprotein 1 Antibody IgG	2	<10 U/ml
Lupus Anticoagulant (LA1)	46	35-53 seconds
Antithrombin-III	105	75-125% activity
Factor V Leiden	1.2	>0.80
Pro C Normalized ratio	1.1	>0.80
PCAT	85.3	85-200 seconds
PCAT/O	35.8	35-55 seconds
PCR for Prothrombin gene mutation	Negative	Negative

Table 1. THROMBOPHILIA SCREENING TESTS

DISCUSSION

Cerebral venous sinus thrombosis results usually as a consequence of procoagulant conditions. [8] Cerebral venous sinus thrombosis has been described as a process of continued imbalance between prothrombotic and thrombolytic states, leading to progression of venous thrombosis with time. [2][The mechanism by which cerebral venous sinus thrombosis occurs in patient with hepatitis B and C is not fully established, but it is thought that hepatitis is associated with formation of anti-cardiolipin antibodies. [9,10] Hence, the virus exclusively or in the combination with others such as anti-phospholipid syndrome results from formation of anti-cardiolipin antibodies during chronic hepatitis infection and can shift the delicate procoagulant/ thrombotic balance towards thrombosis.[7] However in our case anti-cardiolipin and other autoantibodies were negative. There are some data that suggest that HCV, itself might be responsible for thrombotic events.[6] It is hypothesized that protein that envelopes HCV has procoagulant activity and also virus genome that encodes for protein serine protease may have procoagulant activity. [11] In addition to that treatment used for HCV is capable of inducing variety of antibodies that also act as procoagulant. [12] Mostly this association is seen with INF-alpha drug. Since our patient was on sofosbusavir and velpatasvir, there is no documented data available for these two drugs that could possibly causing procoagulant state.

CONCLUSION

In conclusion, we suggest that Hepatitis C testing should be included in the investigation of unknown etiology of cerebral venous sinus thrombosis. Since our patient was known case of Hepatitis C and took oral antiviral therapy (sofosbusavir and velpatasvir) it is hypothesized that Hepatitis C virus uniquely is cause of cerebral venous sinus thrombosis.

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Author's contribution:
Haris Majid Rajput; data collection, data analysis, manuscript writing & review
Iqra Athar; manuscript writing, manuscript review
Neelma Naz Khattak; data analysis, manuscript writing, manuscript review
Anam Anis; data analysis, manuscript writing, manuscript review
Mazhar Badshah; data analysis, manuscript writing, manuscript review