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A Case of Cerebral Venous Thrombosis in the Patient with Dengue

Dr. Mandip Tilara[*], Dr. Asha N. Shah[**], Dr. Sharon Negi[*], Dr. Jay Dadhania[***]

[*] 3rd Year Resident, Department of Medicine, B.J. Medical College, Ahmedabad.
[**] Professor & Head, Department of Medicine, B.J. Medical College, Ahmedabad.
[***] 2nd year resident, Department of Medicine, B.J. Medical College, Ahmedabad.

Abstract- We hereby presenting a case of 30 year old male patient with Dengue fever found to have cerebral venous sinus thrombosis. Dengue infection can be either asymptomatic or progress to involve **hemorrhagic manifestations with shock.** Thrombotic events have not been extensively reported, despite the wide range of procoagulant state during illness.

Index Terms- Dengue fever, Cerebral venous sinus thrombosis, Procoagulant state.

I. Case Report

A 30 year old male patient, chronic alcoholic for 10 years presented to emergency department of civil hospital, Ahmedabad with generalized tonic clonic convulsions. He had 4 episodes of seizure in one day, each lasting for 2 min, associated with uprolling of eyeballs , frothing from mouth & post ictal confusion lasting for 5-10 min.

Patient also had high grade fever with chills and rigors for 4 days preceding seizures which was intermittent in nature and relieved by medication. Patient also had diffuse, severe and throbbing type of headache on admission not completely relieved by medication.

Patient did not have any bleeding manifestations, altered sensorium, focal neurological deficit, vomiting or decrease urine output.

On admission, patient had hypotension (BP 90/60 mm of Hg), Tachycardia (104/min) and elevated temperature (102 degree F).

On examination, patient was conscious oriented and irritable with no focal neurological deficit or neck rigidity.

II. Investigations

Hb was 14.2 gm%
Hematocrit was raised (55.1%)
Platelet count were decreased (80,000/ cumm)
TLC of 14,000/cumm with lymphocytosis.
Coagulation profile was normal.

Peripheral smear was negative for malarial parasite and blood culture was negative for any organisms.
Liver function and Renal function tests were within normal limits.

Patient found to have Dengue NS1 antigen positive and Ig M antibody positive.

MRI Brain was suggestive of thrombosis of superior saggital sinus, right transverse sinus and sigmoid sinus and cortical branches.

Patient was treated with iv fluids,, subcutaneous enoxaparin 1 mg/kg/day in 2 divided doses with daily monitoring of platelet counts, aPTT levels. After 3 days of subcutaneous heparin, he was started on oral warfarin keeping INR between 2-3.

III. Discussion

Many factors might increase thrombotic risk in dengue fever. Dehydration is the foremost which leads to increased hematocrit which predisposes to venous thrombosis.

Dengue virus may down regulate thrombomodulin thrombin protein C complex formation thus reducing activated protein C. Low concentration of plasma anticoagulant proteins C and S and antithrombin III have been detected in severe dengue but have not been associated with clinical thrombosis. No procoagulant risk factor was identified in this case.

Dengue virus activates endothelial cells and increases the expression of thrombomodulin . Lin, et al described host antibodies formed against dengue non structural protein that had cross reactivity with host endothelial cells which can lead to inflammatory responses. Increased PAI-1 plasma levels were also observed. Disseminated intravascular coagulation and consequent microthrombi formation may contribute but have not been associated with large vessel thrombosis. Antibodies against phospholipids, cardiolipin and increased lupus anticoagulant have been associated with thrombotic events in peripheral arteries and cerebral vasculature. Venous cerebral vasculature thrombosis and ischemic stroke not associated with any risk factor have been rarely reported in dengue fever . As the thrombosis was clinically detected at admission, loss of endothelium non thrombogenic protective factors mayhave been the cause.

IV. Conclusion

Clinicians should sought for rare thrombotic manifestations in case of dengue fever apart from more common hemorrhagic manifestations.

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AUTHORS

First Author- Dr.Mandip Tilara 3rd Year resident, Department Of Medicine, B.J. Medical College, Ahmedabad

Second Author- Dr. Asha N. Shah, Professor & Head, Department Of Medicine, B.J. Medical College, Ahmedabad.

Third Author- Dr. Sharon Negi, 3rd year resident, Department of Medicine, B. J. Medical College, Ahmedabad

Forth Author- Dr. Jay Dadhania, 2nd year resident, Department of Medicine, B.J. Medical College, Ahmedabad.

Correspondence Author- Dr. Mandip Tilara ,3rd year resident, department of Medicine, B.J. Medical College, Ahmedabad.
Email ID:- drmandiptilara@gmail.com