Bangladesh Medical Res Counc Bull 2026;52(2): 115-118

DOI: 10.3329/bmrcb.v52i2.89099

CASE REPORT

A Rare Case of Cerebral Venous Sinus Thrombosis with Idiopathic Thrombocytopenic Purpura: Case Report

Maftahul Jannat, Kanuj Kumar Barman, SK. Mahbub Alam

Department of Neurology, Bangladesh Medical University (BMU), Shahbag, Dhaka

Abstract

Cerebral venous sinus thrombosis (CVST) in itself is a rarely encountered clinical entity, and its association with immune thrombocytopenic purpura (ITP) makes it a more unusual presentation. Here we reported a young patient of known ITP presented with sudden severe headache, convulsion and weakness of left side of the body. The case was thoroughly evaluated clinically, and then the diagnosis was made by clinical presentation along with raised D-dimer and magnetic resonance venography (MRV) of brain findings supportive of CVST. Though rare, a suspicion of cerebral venous sinus thrombosis should be considered in a patients with known ITP with features of raised intracranial pressure and focal neurological deficit. Concomitant occurrence of bleeding disorder and thrombosis is challenging to treat. But appropriate diagnosis and early intervention can save the patient.

Key words: Cerebral venous sinus thrombosis, Immune Thrombocytopenic Purpura, D-dimer, Magnetic resonance venograhy.

Introduction

Immune thrombocytopenic purpura is an autoimmune hematological disorder characterized by low platelet level due to destruction by autoimmune antibodies. Cerebral venous thrombosis is a serious condition caused by a thrombosis in the cerebral venous sinuses that occurs mostly in the presence of a hypercoagulable state.

Immune thrombocytopenic purpura (ITP) is an acquired autoimmune hematological disease, characterized by antibody-mediated platelet destruction causing thrombocytopenia.[1,2] Its clinical manifestations are widely variable that some patients can be completely asymptomatic or express some minor bleeding symptoms including purpura, petechiae, epistaxis, and hemorrhagic tendency. On the other hand, it may present with serious bleeding such as intracranial hemorrhage, especially during severe thrombocytopenia.[1,2]

***Correspondence:** Maftahul Jannat, Department of Neurology, Bangladesh Medical University (BMU), Shahbag, Dhaka.*
Email: dr.mj.swarna.26@gmail.com
ORCID ID: 0000-0002-7470-7936

Among the cerebrovascular diseases cerebral venous sinus thrombosis (CVST) is considered one of the rare case of stroke which can present in any age group and accounts for only 0.5% of all stroke cases.[3] Majority of risk factors that are associated with CVST mostly have a common pathophysiological ground of prothrombotic state either transient or permanent and immune thrombocytopenic purpura (ITP) also falls under that category.[4]

The occurrence of a significant thrombotic phenomenon on the background of immune thrombocytopenia can be considered a clinical paradox. Here we present a young male with known ITP was diagnosed as a case of cerebral venous sinus thrombosis on the basis of clinical features along with raised D- dimer and supportive findings of cerebral venous sinus thrombosis on Magnetic resonance venography (MRV) of brain.

The case presentation

A 15-year-old boy was admitted to the department of neurology of a tertiary care hospital in Bangladesh with a 2-week history of sudden severe headache with several episodes of vomiting. Two days before

admission, he also developed sudden-onset weakness of the left side of the body. He had no history of fever but had been suffering from acute gastroenteritis 3 days prior to the development of the headache. He was a diagnosed case of chronic idiopathic thrombocytopenic purpura (ITP) since 2019. Initially he was on treatment under a hematologist with Tab. Eltrombopag 25 mg for 3 years. But for the last 1 year, he was without treatment and lacked follow-up. He had no history of major bleeding manifestations. He had no family history of a similar type of illness.

General physical examination was normal including vital signs. On neurological examination, His higher psychic function including GCS was normal, there was bilateral papilloedema. Motor system examination revealed muscle power on the left side both upper and lower limb was MRC grading 3/5, deep tendon reflexes on the left side were exaggerated with extensor plantar response. Sensory and cerebellar signs were normal. Initial CT scan showed small area of hyper density on the left temporal region (dense clot sign over left transverse sinus) that could not explained the left sided hemiparesis. His initial platelet count was 90000/μL. Then we do MRI of brain which revealed haemorrhagic infact in the right thalamocapsulo-ganglionic area, part of midbrain. Then we do D-dimer and MRV of brain. D dimer was raised (1.49) and extensive venous sinus thrombosis with distended draining vein was seen on MRV. Then final diagnosis was made as a case of cerebral venous sinus thrombosis. Other thrombophilic screening test was normal except protein S level was found low 54.10% (reference value 70.00-125.00%). Patient was put on Inj. Enoxaparin 60 mg subcutaneously twice daily. Considering the initial diagnosis of ITP, Haematological consultation was taken and tab prednisolone was started. A week later he was improved clinically and d-dimer level was normal and platelet count was 120000/μL. Then patient was discharged with advice to take oral anticoagulant (rivaroxaben) for lifelong.

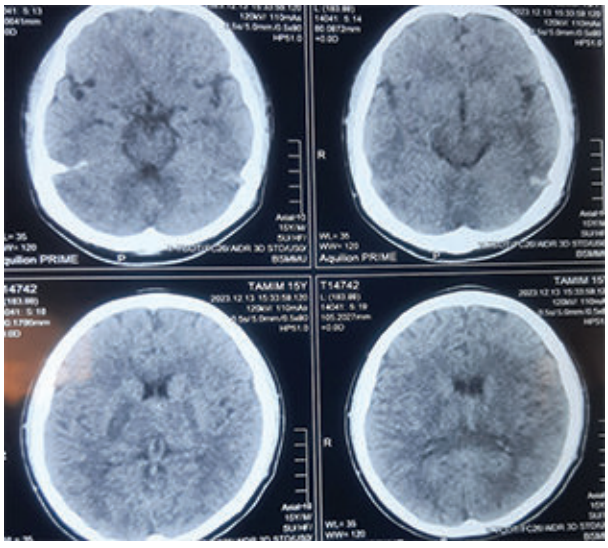

Figure 1: Plain multiaxial CT scan of head showing linear hyper dense lesion in left temporal region resembling dense clot sign over the left trenverse sinus.

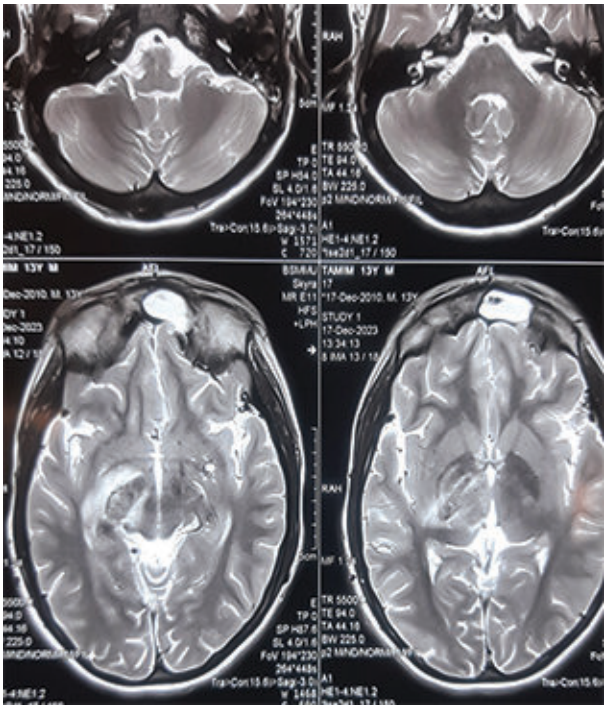

Figure 2: Multiaxial T2 sequence MRI of brain showing mixed intensity lesion in right thalamus, midbrain and part of capsuloganglionic area.

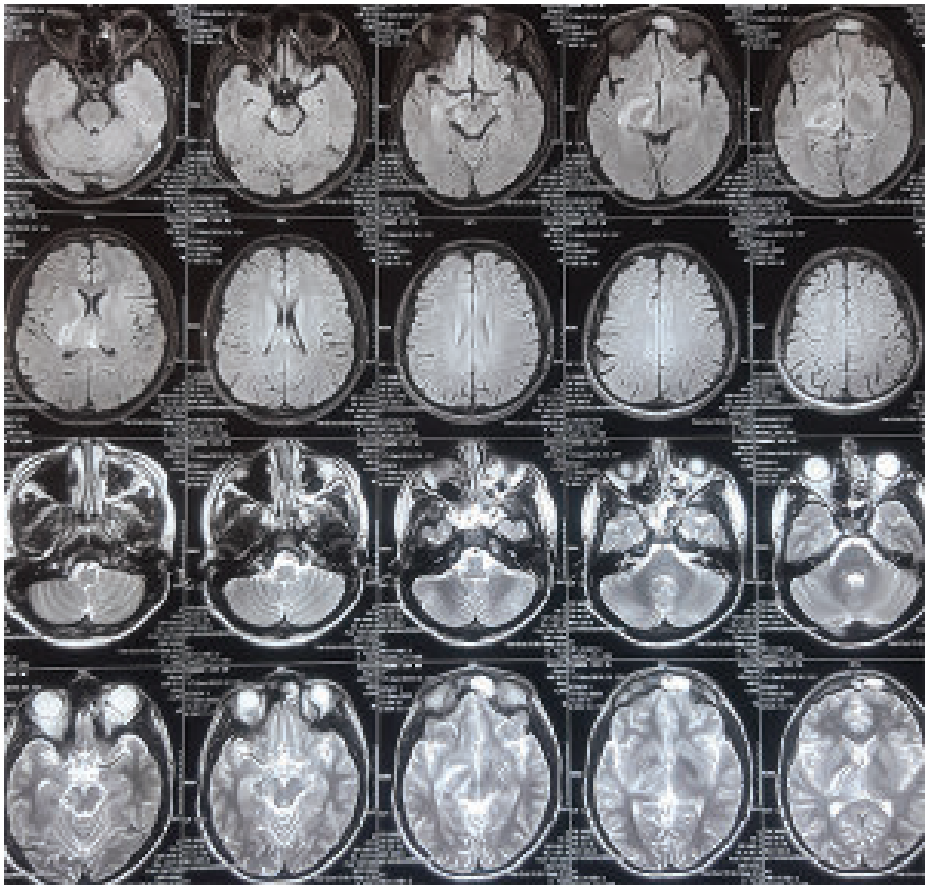

Figure 3: Multiaxial T2 and FLAIR sequence MRI of brain showing mixed intensity lesion in right thalamus, midbrain and part of capsuloganglionic area which showed blooming on GRE suggestive of haemorrhagic infarct.

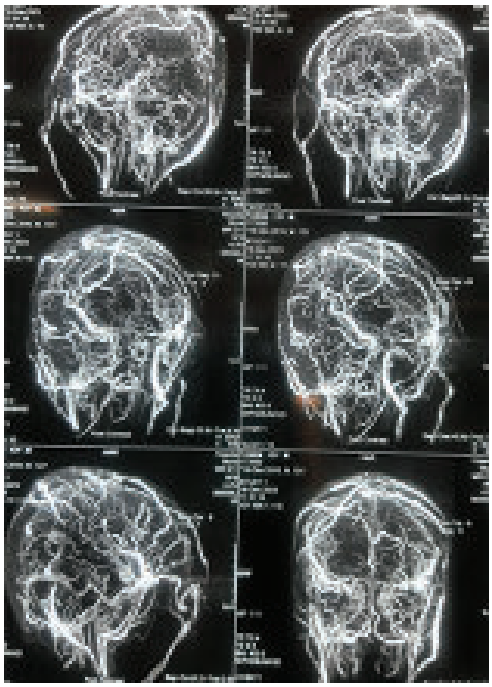

Figure 4: Post contrast MRV of brain showing extensive cerebral venous sinus thrombosis.

Discussion

The clinical presentation of CVST is often variable, and patients may present with a range of symptoms and signs depending on the site and extent of thrombosis and the underlying cause. The most common presentation includes signs of raised intracranial pressure, such as headache, vomiting, reduced visual acuity, papilledema, focal neurological deficits and seizures. Our patient presented with headache, vomiting, papilloedema and left sided hemiparesis.

ITP is an autoimmune disorder characterized by a thrombocytopania due to its destruction mediated by autoimmune antibodies[2,5]. Its spectrum of clinical manifestations is variable including completely asymptomatic cases as well as lifethreatening bleeding menifestations in severe cases. However, in other settings, patients may express minor bleeding manifestations such as purpura, epistaxis, petechiae, and bruising, as our patient.

Although bleeding is the main manifestation of ITP, the paradoxical process, that is, thrombosis, can also occur. Which may related to disease itself or due to its treatment or other factors related to risk of thrombosis.

Some previous studies suggested that patients with ITP have an increased risk for thrombosis [1,2,5,6,] . Many hypotheses can explain the thrombotic events in patients with ITP, and some of them are associated with the disease etiology and other are associated with its treatment (thrombopoietin receptor agonist, corticosteroids). Exposure to IVIG or thrombopoietin receptor agonist (Eltrombopag) increases the risk of thrombosis. However, the corticosteroids may also contribute to the thrombotic process by causing a hypercoagulable state, thus leading to thrombosis [2-7]. Whatever the hypothesis of development thrombosis in a patient with ITP is difficult to treat. A case of 19 year old Asian woman with idiopathic thrombocytopenic purpura complicated by CVST treated with Eltrombopag 75mg which may be the contributing factor as reported by Xin Zhang et al from a Chinese Medical University.[8] Our patient after initial diagnosis of ITP had received Eltrombopeg 25microgram daily for initial 3 years. He had no major bleeding manifestations or thrombotic events during treatment. But he stopped Eltrombopeg and lack of follow up for last one year before the development of cerebral venous sinus thrombosis. He had history of gastroenteritis three days before the onset of severe headache and was not treated properly. So dehydration may contribute to the development of CVST. But as concomitant occurrence of ITP and CVST is extremely rare, we were searching for other causes of thrombosis. All the thrombophilic screening tests are negative except Protein S, which was found low. That may contribute to the development of CVST in our patient. The disturbed equilibrium of prothrombic and thrombolytic processes is considered the main underlying Pathology of CVST. Hereditary prothrombotic conditions such as Factor V Leiden, deficiency of proteins C and S and antithrombin III as well as prothrombin gene mutations account for 10–15% of cases.[9] After diagnosis of CVST it is challenging to treat the patient with ITP because of chance of bleeding. We started Low molecular heparin and continued for 7days and patient's condition was improved without any bleeding manifestations and d-dimer level back to normal, then rivaroxaben was started and maintained. Platetet count was 90,000/iL before starting of treatment. Then we consulted haematologist and oral corticosteroid was started and platelet count increased (120000/iL). Following improvement we discharged the patient with advice for follow up.

Conclusion

Though rare, a suspicion of cerebral venous sinus thrombosis should be considered in a patients with known ITP with features of raised intracranial pressure and focal neurological deficit. Dehydration and low protein S level may be the contributing factor in our case. Concomitant occurrence of bleeding disorder and thrombosis is challenging to treat. But appropriate diagnosis and early intervention can save the patient.

Conflict of Interest: There are no conflicts of interest.

Funding Source: Not applicable

Ethical Clearance: Not applicable

Submit Date: 30 March 2026

Accepted: 08 July 2026

Final Revision Received: 16 August 2026

Publication: 20 August 2026

References:

1. Ntantos D, Karantali E, Prevezianou A, Angelopoulos P, Bostantjopoulou S. Spontaneous epidural hematomas due to cerebral venous thrombosis in a patient with immune thrombocytopenic purpura. *J Stroke Cerebrovasc Dis.* 2020;29(11):105244. DOI:10.1016/j.jstrokecerebrovasdis.2020.105244.
2. Park KM, Yang EJ, Lim YT. Dural venous sinus thrombosis and pulmonary embolism following immunoglobulin treatment in pediatric patient with immune thrombocytopenic purpura. *J Pediatr Hematol Oncol.* 2017;39(8)–e511. DOI:10.1097/MPH.0000000000000841.
3. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol.* 2007;6:162–170. DOI:10.1016/S1474-4422(07)70029-7.
4. Boulware R, Refaai MA. Why do patients with immune thrombocytopenia (ITP) experience lower bleeding events despite thrombocytopenia? *Thromb Res.* 2020;187:154–58. DOI:10.1016/j.thromres.2020.01.020.
5. Ruggeri M, Tosetto A, Palandri F, Polverelli N, Mazzucconi MG, Santoro C, et al. Thrombotic risk in patients with primary immune thrombocytopenia is only mildly increased and explained by personal and treatment-related risk factors. *J Thromb Haemost.* 2014;12:1266–73. DOI:10.1111/jth.12636.
6. Arboix A, Besses C. Cerebrovascular disease as the initial clinical presentation of haematological disorders. *Eur Neurol.* 1997;37:207–11. DOI:10.1159/000117444.
7. Girolami A, De Marinis GB, Bonamigo E, Treleani M, Vettore S. Arterial and venous thromboses in patients with idiopathic (immunological) thrombocytopenia: a possible contributing role of cortisone-induced hypercoagulable state. *Clin Appl Thromb Hemost.* 2013;19:613–18. DOI:10.1177/1076029612452114.
8. Zhang X, Zhang K, Li F. Cerebral venous sinus thrombosis in a young patient with idiopathic thrombocytopenic purpura: a case report. *Indian J Ophthalmol Case Rep.* 2023;3:1076–79. DOI:10.4103/IJO.IJO_85_23.
9. Deschiens MA, Conard J, Horellou MH, et al. Coagulation studies, factor V Leiden, and anticardiolipin antibodies in 40 cases of cerebral venous thrombosis. *Stroke.* 1996;27:1724–30. DOI:10.1161/01.STR.27.10.1724.