Successful Treatment of Cerebral Venous Thrombosis with Rivaroxaban: A Case Report with Brief Review

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Abstract

Cerebral venous thrombosis (CVT) is one of the rarest causes of stroke in general population, where thrombosis occurs in cerebral venous system. Treatment of CVT is mainly done by heparin followed by vitamin K antagonist. Direct oral anticoagulants are introduced which are safe and effective against traditional therapy, however recent protocol do not recommend. A 22 year-male came to emergency department with chief complaints of severe headache, multiple episodes of vomiting and 2- 3 episodes of loose stool for 3 days. He had two episodes of abnormal body movement in emergency department. Computed tomography (CT) scan of head and then CT venography of head also were done after admission that reported venous thrombosis in superior sagittal sinus, bilateral transverse sinus and internal cerebral vein. Patient was admitted and treated withdirect oral anticoagulant (rivaroxaban). He was discharged on 8th day of admission without any neurological deficits.

Keywords: Cerebral venous thrombosis, Direct oral anticoagulant, Rivaroxaban, Recanalization

Introduction

Cerebral venous thrombosis (CVT) is comparatively uncommon kind of stroke inclusive of cerebral vein, and/or cerebral venous sinus which accounts 0.5% to 1.0% of all cerebrovascular accident leading to death in less than 5% or morbidity i.e. lifelong dependency in 15 % of cases .[1,2] Patho-physiologically, CVT externalizes clinical manifestation by decreasing venous drainage and cerebral spinal fluids absorption leading to elevation in intracranial pressure or cerebral parenchyma dysfunction or bleeding.[3] Clinical manifestations include headache, vomiting, seizure, papilloedema, focal deficits and/or altered mental status.[4] Suggested treatment of CVT is anticoagulant unless contraindicated; intravenous unfractionated heparin or subcutaneous low molecular weight heparin along with long term oral vitamin K antagonist, warfarin for 3-6 month.[5] New direct oral anticoagulants were proposed to treat symptomatic venous thromboembolism over the past 10 years and have benefits over warfarin: no monitoring of international normalized ratio (INR) or adjustment of daily doses, and fewer chances of intracranial hemorrhages.[6] Here we report our successful experience in cerebral venous thrombosis treated with direct oral anticoagulant (i.e., rivaroxaban).

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Case presentation

This is a case of a 22-year-old man who presented in our hospital with complaints of dizziness, severe headache, nausea, and vomiting. Furthermore, he had 2-3 episodes of generalized tonic-clonic seizure in the emergency department, which were managed with IV Midazolam and Levetiracetam. He had a history of diarrhea few 2-3 days. The patient had an urgent CT scan that reported superior sagittal sinus thrombosis (Figure 1A). Further, a CT venogram was done, which showed thrombosis in the superior sagittal sinus, B/L transverse and internal cerebral vein (Figure 1B). He was admitted to the neurosurgery intensive care unit(ICU). An initial blood investigation with coagulation profiles was done, which were normal except that D-dimer was at a higher level (3.18mg/L). The patient was started on subcutaneous low molecular heparin,40mg once daily for 7 days, along with rivaroxaban (Rixab) at a dose of 10mg orally twice daily. The patient was stable neurologically during the hospital stay and was discharged well. He was discharged on the 8th day of admission. He was advised to take rivaroxaban (Rixab) at a dose of 10mg orally twice daily for 15 days at first, then 10mg orally once daily for 3months. Brain magnetic resonance imaging (MRI) and magnetic resonance venography (MRV) were done at the 3rd month to see recanalization of thrombosed venous sinuses (Figure 1C). Clinically, he was completely symptomless, and

radiologically, there was no sign of thrombosis in the cerebral venous system.

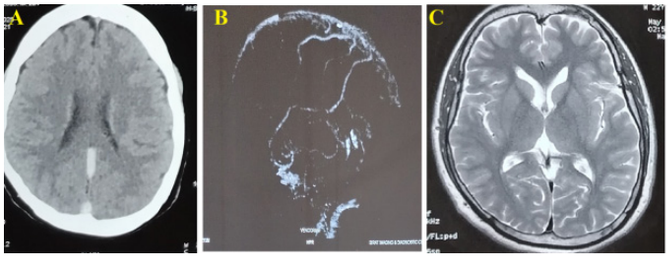

Figure 1. 1A shows an axial section of CT head with cerebral dural venous sinus thrombosis, 1B shows cerebral CT venogram with sagittal sinus, B/L transverse sinus, internal jugular vein, and cerebral vein thrombosis, and 1C shows an axial section of T2-weighted MRI brain with complete recanalization of dural venous thrombosis at 3 months during follow-up.

Discussion

Partial or complete blockage of one or more veins and/or cerebral dural venous sinus may be the cause of cerebral venous thrombosis (CVT), leading to cerebrovascular accident. CVT are described in mainly young adults (<50 years) as well as women and children, leading 0.5% to 1.0% of incidence of all cerebrovascular accident[7] and prevalence ranges 1.3 to 1.6 patients per 100,000 persons in high income countries, and higher in low to medium income countries have been explained.[8,9]CVT counterfeits multiple dural venous sinus, mostly affecting transverse and sagittal dural venous sinus.[10]Risk factors for CVT are similar to venous thromboembolic accident, which are multiple episodes of diarrhea and vomiting, dehydration, pregnancy, use of oral contraceptives, head trauma or drug abuser, inflammatory bowel diseases, malignancy, infectious diseases and thrombophilia..[3, 11, 12]Similar to these previous literatures, we noted that dehydration was major factor for the development of CVT in our patient. Increased intracranial pressure(ICP) and /or cerebral parenchyma dysfunction can be observed due to decreased venous blood drainage and cerebrospinal fluid absorption, which produce various clinical manifestations.[3]

The various clinical manifestations have been described because of various underlying situations, which are headache, vomiting, seizure, papilloedema, altered mental status, and/or focal neurological status, headache, seizure, papilloedema, focal neurological deficits, and/or altered state of consciousness, stupor, or coma.[13,14] Similar to previous studies, our patient had symptoms of severe headache, multiple episodes of vomiting, and multiple episodes of generalized seizure. The most accepted treatment for CVT includes injection of heparin followed by oral anticoagulant, i.e., warfarin,[11,15]however it has some convincing limitations like the need for laboratory monitoring tests to maintain an international normalized ratio (INR) in the range of 2-3, more possibility of internal hemorrhage, and drug interactions. [16]Therefore, scientists investigated new direct oral anticoagulants to overcome these problems. Recently, direct oral anticoagulant (i.e, rivaroxaban) has been introduced, which directly inhibits Xa factor as well as has similar benefits to injecting anticoagulant plus oral anti-vitamin K antagonist.[17] Furthermore, less intracranial bleeding or fatal bleeding in rivaroxaban-treated patients compared to warfarin-treated patients has been described.[18]

The knowledge with respect to use of newer oral anticoagulant (rivaroxaban) in cerebral venous thrombosis is not enough as well as studies on the small number of cases.[19]There was a study on seven CVT cases with use of oral anticoagulant i.e. rivaroxaban after initial injection heparin and was noted safe and effective.[19]Similarly, another study done by Mutgi et al, where two patients with CVT treatment with rivaroxaban for three months showed partial to complete re-canalization, and no bleeding or recurrence were described.[20] Furthermore, a study done by Rusin et al comparing three oral anticoagulants, i.e., dabigatran, rivaroxaban, and apixaban in eighteen, nineteen, and eight patients with CVT, respectively, where bleeding complications in three patients have been described: two from the rivaroxaban and one from the dabigatran group.[21] Similarly, Anticoli S et al studied 6 CVT patients with three different regimes of rivaroxaban treatment, where two patients were managed with heparin bridging therapy with warfarin for 15days, after that only warfarin for another three months, and daily 20mg rivaroxaban for another three months. The next two CVT patients were managed with seven days of heparin therapy followed by daily 20mg of rivaroxaban at the time of heparin discontinuance. The last two CVT patients were managed with 15mg of rivaroxaban twice daily for 21 days, then 20mg once daily, as a regimen for DVT. No recurrence of CVT, no major bleeding, and better neurological outcome were noted during the follow-up period(12 months).[22] Similar to this study, our CVT patient was treated with initial subcutaneous low molecular heparin for 7days followed by 10mg of rivaroxaban twice daily for 15days, then after 10mg of rivaroxaban once daily for three months, where no major bleeding, no recurrence and no adverse effects as well as excellent canalization excellent neurological outcome were reported during follow up period (3months).In contrast, another study done by Mustafa et al explained that the presence of new CVT was acknowledged as a treatment failure; however, complete anticoagulation with rivaroxaban, then later patient was treated with another anticoagulant i.e.150mg of dabigatran twice daily for six months.[23]Therefore, in response to to this report, further more studies about oral anticoagulant i.e. rivaroxaban for cerebral venous thrombosis are solidly required

Conclusions

In our case report, it shows that treatment of CVT with a direct oral anticoagulant is safe and effective. No recurrence, no bleeding, and no adverse effects were noted during the follow-up period of 3 months. However, larger, randomized, multicentre studies with a longer follow-up period are needed to validate this study.

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Ethical approval: This is a retrospective study, so informed

consent was taken from the institute, and a involved participant in this study.

Conflict of interest: Author certifies that they have no affiliations with or involvement in any organizations or entity with any financial interest, or non-financial interest.

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