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Cerebral Venous Thrombosis Following Varicella Zooster Infection: Case Report

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Case Report

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Abstract

Background

Cerebral venous thrombosis (CVT) is a rare yet life-threatening neurological condition[1]. While primary causes are well-documented, secondary post-infectious triggers, particularly Varicella Zoster Virus (VZV) infection, are frequently overlooked. This report presents a case of CVT following a recent VZV infection, emphasizing the importance of considering vascular complications in young patients presenting with acute neurological deficits.

Case Presentation

A 30-year-old male presented with a 6-day history of high-grade fever, severe headache, confusion, and a generalized tonic-clonic seizure. Two weeks prior, he had been treated for a widespread vesicular rash diagnosed as Varicella Zoster. Neurological examination revealed confusion (GCS 14/15) without focal neurological deficits. Brain CT and CT cerebral angiography confirmed a hemorrhagic venous infarct in the left temporal lobe secondary to thrombosis of the left transverse and sigmoid sinuses and the internal jugular vein.

Management and Outcome

The patient was managed with anticoagulation (Enoxaparin transitioned to Rivaroxaban) and supportive therapy. He demonstrated significant clinical improvement, with symptoms resolving within 10 days of admission.

Conclusion

This case highlights the potential for VZV to induce severe intracranial venous thrombosis through vasculopathic mechanisms. Clinicians should maintain a high index of suspicion for CVT in patients presenting with unexplained neurological symptoms following a recent VZV infection.

INTRODUCTION

Cerebral Venous Thrombosis (CVT) represents a challenging clinical entity, accounting for approximately 0.5% to 3% of all strokes[1]. Its clinical presentation is highly variable—ranging from isolated headache to encephalopathy and seizures—making early diagnosis difficult[2].

The etiology of CVT is broad, often involving a combination of prothrombotic states, pregnancy, oral contraceptive use, or malignancy. However, post-infectious triggers represent a critical, albeit less common, cause[1]. Varicella Zoster Virus (VZV), while classically known for dermatological and benign

neurological manifestations, possesses significant vasculotropic potential. VZV-induced vasculopathy occurs when the virus invades the vessel wall, promoting endothelial inflammation, intimal proliferation, and a subsequent pro-thrombotic milieu[3, 4].

This case report details the clinical course of a 30-year-old male who developed a hemorrhagic venous infarct secondary to CVT two weeks after a VZV infection. By documenting this association, this report aims to heighten awareness among clinicians, emphasizing that neurological deterioration post-VZV should prompt urgent neurovascular imaging to rule out venous thrombosis, even in the absence of traditional stroke risk factors.

CASE PRESENTATION

Mr T.P, a 30 year old male who presented 6days history of Fever, headache, 1day history of altered sensorium and an episode of seizure. Fever was said to be high grade and intermittent, headache was said to be sudden in onset,severe, generalized, with associated phonophobia, photophobia and projectile vomiting. A day before presentation patient was noticed to be talking irrationally, confused and has both short & long term memory impairment. Few hours before presentation he had a generalized tonic clonic seizure said to have lasted for about 5mins and resolved spontaneously with post-ictal drowsiness. No neck pain, visual impairment or differential limb weakness. No similar episode in the past

Two weeks prior to presentation he was diagnosed of varicella zoster infection as he had widespread breakout of pruritic vesicular rash on account of which he was placed on oral acyclovir. There was no history of trauma, URTI or illicit drug use. No history of recent travels. No other associated systemic symptoms

He is not previously known to have hypertension or diabetes mellitus.Not on any routine medications, no history of recreational drug usage however takes alcohol occasionally. His genotype is AA

Examination revealed a young man afebrile, not pale, anicteric, acyanosed, not dehydrated and no pedal edema.

He had multiple hyperpigmented maculopapular rash over his trunk, arms, and lower limbs as shown in Fig. 1.

Nervous system examination revealed a conscious but confused patient with GCS of 14/15, not oriented in time, place and persons, no signs of meningeal irritation,pupils equal and bilaterally reactive to light, no obvious cranial nerve palsy, no focal neurological deficit, tone, power and reflexes were all intact in all limbs.

Pulse rate was 68bpm, blood pressure 102/69mmHg, apex beat was located at 5th LICS mid-clavicular line, S1 and S2 heart sounds were heard, no added sounds. Chest was clinically clear.

An initial assessment of acute encephalitis was made to r/o intracerebral hemorrhage

He was started on IV Dexamethasone 8mg 8hrly, IV 20% Mannitol 250ml 8hrly for 24 hrs with IV frusemide 20mg after each dose of Mannitol, IVF 0.9% normal saline alternating with 5%dextrose water 500ml 4hrly, IV phenytoin 1g to run over 1hr then 250mg over 30min every 12hr, IV ceftriaxone 1g 12hrly and IV PCM 600mg 8hrly.

Brain CT scan done as shown in Fig. 2. revealed acute hemorrhage in the left temporal lobe with perilesional edema and midline shift noted.

CT cerebral angiography as shown in Fig. 3. revealed features suggestive of left temporal lobe hemorrhagic venous infarct likely secondary to cerebral venous thrombosis.

Full blood count which showed Pack Cell Volume 46.0%, White Cell count 7,100, Neutrophilia of 80.9, Platelet count 224,000/mm^3

EUCr,LFT,RBS,Urinalysis and clotting profile were essentially normal. Hiv, antiHCV,HbsAg were all non-reactive

A definitive diagnosis of cerebral venous thrombosis was made and patient was subsequently started on Tabs Nimodipine 30mg 12hrly, S/c enoxaparin 60mg daily.

Patient made clinical improvement as symptoms resolved and was discharged after 10days on admission. He was discharged on tab rivaroxaban 20mg daily, Tabs Dexamethasone 4mg tds, tab levetiracetam 250mg bd, tab citicoline 1g bd and tab cefixime 400mg daily

He is to present on outpatient bases for subsequent follow-up and to repeat Brain imaging after 4weeks

DISCUSSION

Cerebral Venous Thrombosis (CVT) occurs when a blood clot forms in the veins or dural sinuses that drain blood from the brain. While it accounts for only 0.5% to 3% of all strokes worldwide, it primarily affects people under 55years, with two-thirds of cases occurring in women[1]. Neurological complications from chickenpox occur in less than 1% of cases, and CVT is among the rarest of these[3]. While historically seen in children, adult cases of VZV-related CVT have been reported more frequently in recent years and tend to be more severe[5].

Cerebral Venous Thrombosis (CVT) remains a diagnostic challenge due to its protean clinical manifestations, which often mimic more common neurological conditions such as encephalitis, stroke, or migraine[1]. While the etiology of CVT is multifactorial as shown in Fig. 4—including inherited thrombophilias, oral contraceptive use, pregnancy, and malignancies—post-infectious etiology, particularly related to the Varicella Zoster Virus (VZV), is a clinically significant yet under-recognized trigger[6].

Pathophysiological Mechanisms

The temporal association in this case—the onset of neurological symptoms two weeks post-VZV infection—suggests an immune-mediated or direct vasculopathic mechanism. VZV is known for its neurotropism and vasculotropism. Beyond the classic dermatomal rash, VZV can induce a large-vessel or small-vessel vasculopathy[3, 4]. The virus replicates within the arterial and venous walls, causing direct endothelial damage, inflammation, and subsequent intimal proliferation. This inflammatory state creates a pro-thrombotic environment(trigger antiphospholipid antibodies and reduce protein S levels), which, when coupled with the systemic inflammatory response to the viral infection, likely precipitated the thrombosis in the transverse and sigmoid sinuses observed in this patient[1]. Other sinuses that could be involved are as shown in Fig. 5.

In this case, the patient's initial presentation with altered sensorium, seizures, and headache led to a high suspicion of encephalitis, which is a common diagnostic pitfall. The sudden onset of "thunderclap" headache, however, should always prompt neuroimaging to rule out vascular pathology. Our case highlights the critical importance of neuroimaging early in the workup of acute neurological decline, even when a recent viral illness provides a seemingly simple explanation like "encephalitis."

A significant clinical decision in this case was the initiation of full-dose anticoagulation despite the presence of a left temporal lobe hemorrhagic infarct. There is often clinical hesitancy to anticoagulate patients with intracerebral hemorrhage due to the fear of exacerbating bleeding. However, established guidelines emphasize that CVT-associated hemorrhage is often a direct consequence of venous congestion and ischemia due to the clot itself. Therefore, anticoagulation is the primary treatment to restore venous outflow[1, 7]. The patient's clinical improvement on Enoxaparin, followed by the successful transition to a Direct Oral Anticoagulant (DOAC) like Rivaroxaban, reinforces the safety and efficacy of anticoagulation in this cohort of patients, provided the neurological status is closely monitored. Endovascular therapy is usually considered in patients whose symptoms deteriorate despite being on therapeutic anticoagulation[1, 8].

CONCLUSION

This case serves as a valuable reminder that CVT should be considered in the differential diagnosis of young patients presenting with neurological deficits following a recent varicella-zoster infection. While VZV is typically viewed as a dermatological or benign neurological condition, its potential to induce severe intracranial venous thrombosis is a critical clinical lesson. Early recognition, combined with a low threshold for initiation of anticoagulation, is pivotal for a favorable neurological outcome.

Declarations

- Ethics approval and consent to participate- Not applicable
- Consent to publish- Written informed consent for publication of this case and the accompanying images were obtained from the patient. I also confirm that a copy of the consent form would be available on request for review by the Editor-in-Chief of this journal.

- Data availability- Not applicable
- Competing interests- None
- Funding- None

Authors' contributions

- Sotubo Sotomiwa- Prepared the abstract, discussion and proof read the whole manuscript
- can also select other corresponding authors
- Ogunyemi Ayokunle,Oluwaseun Oduwole ,Precious Ali provided history and examination
- Daniel George,Titilayo Olayanju,Oluwamayomikun Aderibigbe supplied the images/figures

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Authors' information (optional)

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Figures



Figure 1

Hyperpigmented maculopapular rash on the trunk, upper and lowerlimbs

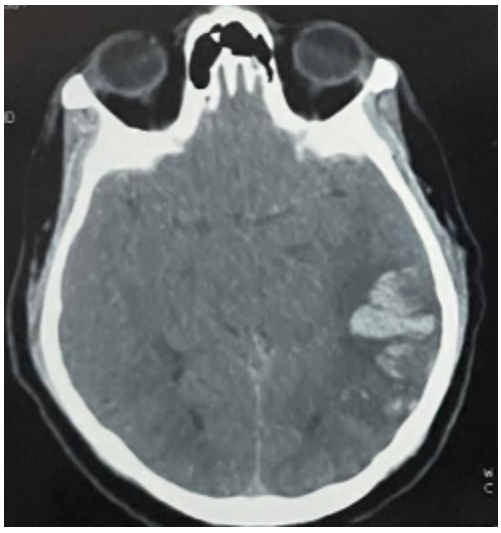


Figure 2

Hemorrhage in the left temporal lobe with perilesional oedema

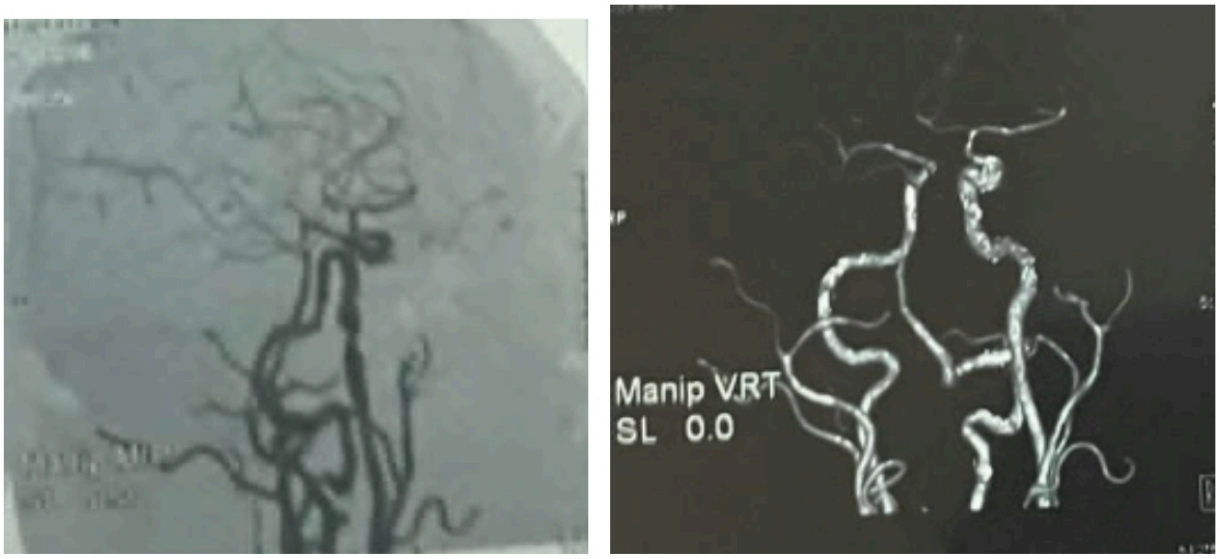


Figure 3

the left transverse and sigmoid sinus as well as the left internal jugular vein are not opacified due to thrombosis

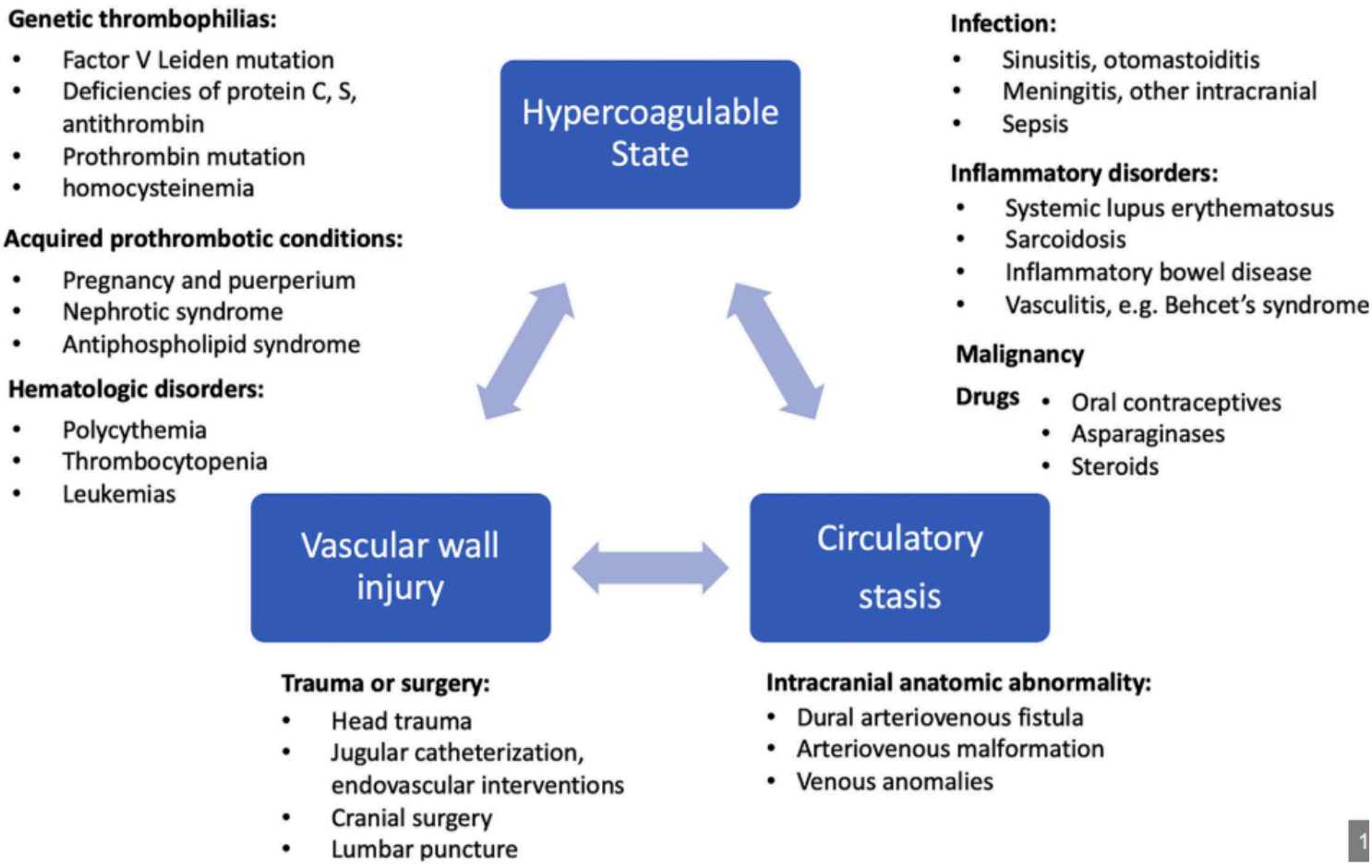


Figure 4

Aetiology of Cerebral venous thrombosis

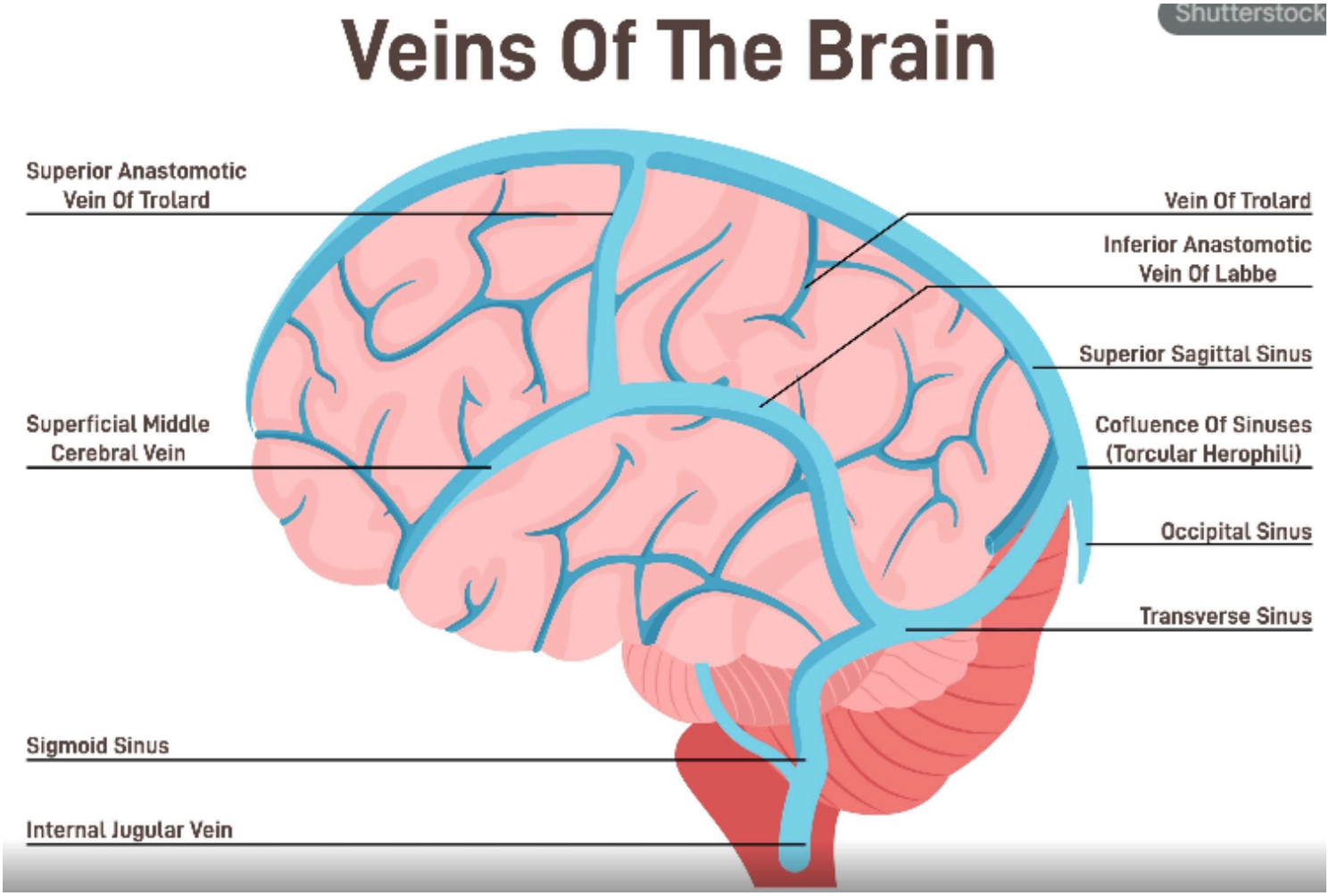


Figure 5

Venous sinuses of the brain