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EXTENSIVE CEREBRAL VENOUS SINUS THROMBOSIS WITH HEMORRHAGIC VENOUS INFARCTION FOLLOWING ADULT CHICKENPOX: A CASE REPORT

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ABSTRACT Neurological complications of chickenpox are uncommon in adults, and cerebral venous sinus thrombosis (CVST) is among the rarest. We report a 30-year-old man who presented with convulsions and altered sensorium 15 days after untreated chickenpox and was managed in the medical intensive care unit of a tertiary care hospital in Gujarat, India. Serum varicella-zoster virus (VZV) IgM by ELISA was positive. Contrast magnetic resonance imaging (MRI) of the brain showed loss of normal flow voids in the superior sagittal, right transverse and right sigmoid sinuses, both veins of Trolard and the superficial cortical veins, with early subacute hemorrhagic venous infarcts in both frontoparietal lobes and the right posterior temporo-occipital region, probable sulcal subarachnoid hemorrhage, edema and a 4 mm leftward midline shift. Laboratory tests showed neutrophilic leukocytosis, thrombocytosis, hypokalemia and raised transaminases. He received intravenous acyclovir, unfractionated heparin infusion, mannitol, levetiracetam and empirical antibiotics, and was discharged fully recovered (Glasgow Coma Scale 15/15). Recent chickenpox should prompt early venous imaging in adults with new neurological symptoms, and hemorrhagic venous infarction should not delay anticoagulation. The case also highlights the pool of varicella-susceptible adolescents and adults in India.

KEYWORDS : Chickenpox, Varicella-Zoster Virus, Cerebral Venous Sinus Thrombosis, Hemorrhagic Venous Infarction

INTRODUCTION

Varicella is usually a mild childhood illness, but disease acquired in adolescence or adulthood is more severe and more often complicated, including by central nervous system involvement.[1] In India, a significant proportion of adolescents and adults remain susceptible to varicella.[2] Varicella-zoster virus (VZV) can involve the cerebral vessels and cause ischemic and hemorrhagic stroke, aneurysm and subarachnoid hemorrhage.[3] Cerebral venous sinus thrombosis (CVST) accounts for 0.5–3% of all strokes and mainly affects young people and women of reproductive age.[4] CVST after primary varicella is described far less often; among 34 adults with cerebrovascular complications of VZV encephalitis reviewed by Wu et al.[5], only three had venous sinus thrombosis. We report an adult with extensive post-varicella dural sinus and cortical vein thrombosis and bilateral hemorrhagic venous infarction.

Case Report

A 30-year-old man was admitted under the Department of General Medicine of a tertiary care teaching hospital in Gujarat, India, with convulsions and altered sensorium 15 days after an episode of chickenpox (Figure 1) for which he had received no treatment, and was managed in the medical intensive care unit (MICU). On bedside assessment in the MICU he was hemodynamically stable, with a pulse rate of 68/min, blood pressure of 126/80 mmHg and oxygen saturation of 99% on room air.



Figure 1: Clinical Photograph of the Back Showing Scattered Papulovesicular Varicella Lesions.

Source: Patient photograph.

Serum VZV IgM by enzyme-linked immunosorbent assay (ELISA) was positive (Figure 2B), supporting recent VZV infection. Biochemistry showed preserved renal function and normal sodium, with hypokalemia, mild hypocalcemia, raised transaminases, low total protein and albumin, and raised lactate dehydrogenase. A coagulation profile three days after imaging showed a prolonged prothrombin time (INR 1.5) with a normal activated partial thromboplastin time, and a hemogram eight days after imaging showed neutrophilic leukocytosis with thrombocytosis (Table 1).

Table – 1 Laboratory Investigations

Parameter	Result	Interpretation
Hemoglobin	13.6 g/dL	Normal
Total leukocyte count	12,600/mm^3	Raised
Absolute neutrophil count	9,828/mm^3	Raised
Platelet count	458,000/mm^3	Raised
Serum urea	27.8 mg/dL	Normal
Serum creatinine	0.71 mg/dL	Normal
Serum sodium	139 mEq/L	Normal
Serum potassium	3.07 mEq/L	Low
Serum calcium	8.0 mg/dL	Mildly low
AST	72 IU/L	Raised
ALT	65 IU/L	Raised
Total protein	5.74 g/dL	Low
Serum albumin	3.34 g/dL	Low
LDH	376 U/L	Raised
PT	20.3 s (control 14 s)	Prolonged
INR	1.5	Raised
aPTT	30.7 s (control 32 s)	Normal
VZV IgM (ELISA)	Positive	Recent VZV infection

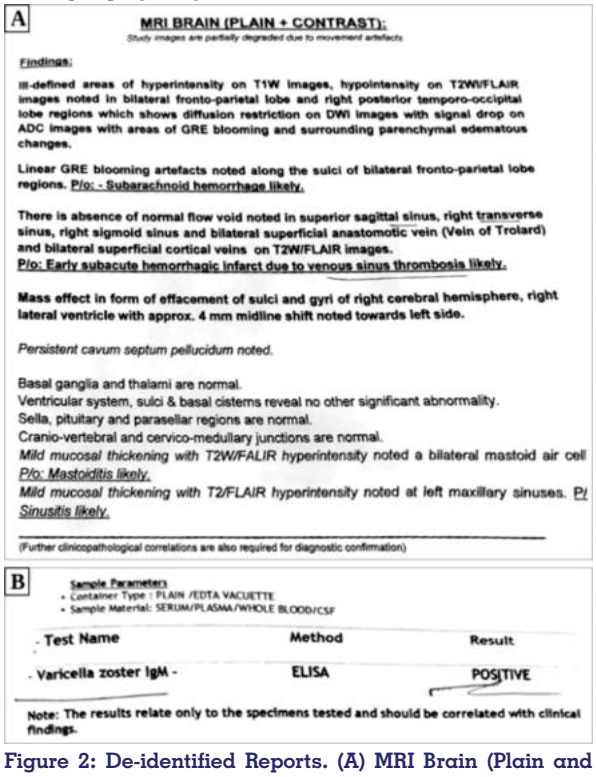
AST: aspartate aminotransferase; ALT: alanine aminotransferase; LDH: lactate dehydrogenase; PT: prothrombin time; INR: international normalized ratio; aPTT: activated partial thromboplastin time; VZV: varicella-zoster virus.

Contrast MRI of the brain, performed the day after the VZV IgM report, was partly degraded by movement artifacts. Ill-

defined lesions, hyperintense on T1-weighted and hypointense on T2-weighted/FLAIR images, were seen in both frontoparietal lobes and the right posterior temporo-occipital region, with diffusion restriction, signal drop on ADC maps, blooming on gradient-echo (GRE) images and surrounding edema. Linear GRE blooming along the sulci of both frontoparietal regions suggested subarachnoid hemorrhage. Normal flow voids were absent in the superior sagittal sinus, right transverse sinus, right sigmoid sinus, both superficial anastomotic veins (veins of Trolard) and the superficial cortical veins, and the radiological impression was early subacute hemorrhagic infarction due to venous sinus thrombosis. Effacement of the sulci and gyri of the right hemisphere and of the right lateral ventricle was present, with an approximately 4 mm midline shift to the left. Mild mucosal thickening of both mastoid air cells and the left maxillary sinus, and a persistent cavum septum pellucidum, were also noted (Figure 2A).

A diagnosis of varicella-associated CVST with hemorrhagic venous infarction was made. Treatment comprised intravenous acyclovir 10 mg/kg (750 mg) by slow infusion every 12 hours for 14 days, unfractionated heparin as an intravenous bolus of 80 U/kg followed by a continuous infusion of 18 U/kg/h, mannitol for cerebral edema, intravenous levetiracetam 1 g every 8 hours, and empirical intravenous meropenem and vancomycin while bacterial infection was being excluded, together with head-end elevation and electrolyte monitoring.

Under serial neurological assessment and supportive intensive care, the presenting neurological manifestations resolved completely, and he was discharged with a Glasgow Coma Scale score of 15/15, with advice on regular follow-up and monitoring for bleeding complications. Written informed consent for publication of this case report and the accompanying images was obtained.


A

MRI BRAIN (PLAIN + CONTRAST):

Study images are partially degraded due to movement artefacts

Findings:

Ill-defined areas of hyperintensity on T1W images, hypointensity on T2WI/FLAIR images noted in bilateral fronto-parietal lobe and right posterior temporo-occipital lobe regions which shows diffusion restriction on DWI images with signal drop on ADC images with areas of GRE blooming and surrounding parenchymal edematous changes.

Linear GRE blooming artefacts noted along the sulci of bilateral fronto-parietal lobe regions. P/o: - Subarachnoid hemorrhage likely.

There is absence of normal flow void noted in superior sagittal sinus, right transverse sinus, right sigmoid sinus and bilateral superficial anastomotic vein (Vein of Trolard) and bilateral superficial cortical veins on T2W/FLAIR images.
P/o: Early subacute hemorrhagic infarct due to venous sinus thrombosis likely.

Mass effect in form of effacement of sulci and gyri of right cerebral hemisphere, right lateral ventricle with approx. 4 mm midline shift noted towards left side.

Persistent cavum septum pellucidum noted.

Basal ganglia and thalami are normal.
Ventricular system, sulci & basal cisterns reveal no other significant abnormality.
Sella, pituitary and parasellar regions are normal.
Cranio-vertebral and cervico-medullary junctions are normal.
Mild mucosal thickening with T2W/FALIR hyperintensity noted a bilateral mastoid air cell P/o: Mastoiditis likely.
Mild mucosal thickening with T2/FLAIR hyperintensity noted at left maxillary sinuses. P/ Sinusitis likely.

(Further clinicopathological correlations are also required for diagnostic confirmation)

B

Sample Parameters
- Container Type : PLAIN /EDTA VACUETTE
- Sample Material: SERUM/PLASMA/WHOLE BLOOD/CSF

Test Name	Method	Result
Varicella zoster IgM -	ELISA	POSITIVE

Note: The results relate only to the specimens tested and should be correlated with clinical findings.

Figure 2: De-identified Reports. (A) MRI Brain (Plain and Contrast): Absent Flow Voids in the Superior Sagittal, Right Transverse and Right Sigmoid Sinuses, Veins of Trolard and Cortical Veins, with Hemorrhagic Venous Infarcts, Sulcal Subarachnoid Hemorrhage and 4 mm Midline Shift. (B) Serum VZV IgM by ELISA: Positive.
Source: Hospital records; patient identifiers removed.

DISCUSSION

This patient had thrombosis of three dural sinuses together with the superficial anastomotic and cortical veins, multifocal hemorrhagic venous infarcts, sulcal subarachnoid blood and mass effect, in temporal association with untreated chickenpox and a positive VZV IgM. Siddiqi et al.[6] first described CVST with primary varicella in two young patients, and Gayathri et al.[7] reported a 23-year-old Indian man who, 15 days after chickenpox, developed thrombosis of the superior sagittal, transverse and right sigmoid sinuses with a right frontoparietal hemorrhagic infarct, and recovered with heparin, acyclovir and oral anticoagulation. The interval and sinus distribution in our patient are almost identical, and the additional cortical vein thrombosis explains the bilateral, non-arterial pattern of the hemorrhagic lesions. In the review by Wu et al.[5], venous sinus thrombosis after VZV encephalitis occurred mainly in young men, most often involved the transverse sinus, and improved in all patients given anticoagulation, as did our patient.

Two mechanisms may link varicella with thrombosis. VZV infects the cerebral vessel wall and causes an inflammatory vasculopathy of large and small vessels.[3] Acute varicella also induces transient autoantibodies; Josephson et al.[8] found lupus anticoagulant, anticardiolipin and anti-protein S antibodies more often during acute varicella, with low free protein S and increased thrombin generation in children who developed thrombosis. A thrombophilia panel drawn during the acute illness should therefore be interpreted cautiously and repeated after recovery.

A positive serum VZV IgM confirms recent infection but not central nervous system invasion. In VZV vasculopathy, cerebrospinal fluid anti-VZV IgG with intrathecal synthesis is a more sensitive marker than VZV DNA by PCR.[9] Lumbar puncture is often deferred when there is mass effect, as in this patient, so the association here rests on the timeline, serology and imaging. Thrombosis was inferred from loss of flow voids; non-arterial multifocal hemorrhagic lesions in a young adult should prompt magnetic resonance or computed tomographic venography for confirmation. The mild mastoid and maxillary mucosal thickening raised the alternative of septic CVST, which justified empirical broad-spectrum antibiotics.

Anticoagulation is the mainstay of CVST treatment, and intracerebral hemorrhage caused by venous outflow obstruction is not a contraindication.[4] Intravenous acyclovir is recommended for VZV infection of the central nervous system,[10] and combining it with heparin treated both the trigger and the thrombosis. Male sex and hemorrhage on admission imaging, both present here, independently predicted death or dependence in the International Study on Cerebral Vein and Dural Sinus Thrombosis,[11] justifying intensive monitoring.

From a public health perspective, varicella vaccine is not part of India's Universal Immunization Programme. About one in five Indians aged 16–20 years and one in ten aged 21–40 years lack antibodies to VZV,[2] leaving a sizeable adult population at risk of severe disease. The vaccine is more than 95% effective against severe varicella,[1] and catch-up vaccination of susceptible adolescents and adults, including health-care workers, deserves wider use.

CONCLUSION

Chickenpox in adults can be complicated by extensive CVST with hemorrhagic venous infarction. In a patient with recent varicella and new neurological symptoms, early MRI with venous imaging is warranted, and hemorrhagic infarction should not delay anticoagulation. Antiviral therapy, anticoagulation and control of cerebral edema should proceed together, and susceptible adolescents and adults should be offered varicella vaccination.

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