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# Post-partum Cavernous Venous Sinus Thrombosis (CVST): A Case Report

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**Abstract:**

***Cerebral venous sinus thrombosis (CVST) is one of the most serious and fatal disorder of intracranial venous pathways characterized by clot formation in Cerebral venous sinuses. Generally, CVST is a fulminating process with a high rate of mortality (1 in 3 patient) secondary to a spreading bacterial infection specially over head and neck region where coagulase positive Staphylococcus (90%) is mostly responsible. Individuals with hypercoagulable state i.e., pregnancy or puerperium and malignancy can also suffer from this form of thrombosis. Here we are presenting such a case, who developed CVST on the 5th day of puerperium with features of persistent headache and eye problems. The diagnosis was confirmed with MRI in combination with Magnetic resonance venography (MRV). She responded well to low molecular weight heparin (LMWH) and was discharged on long term oral anticoagulant and regular follow-ups. Her subsequent pregnancy was uneventful without repeat attack of CVST.***

***Key words: Cavernous Venous Sinus Thrombosis (CVST), Magnetic resonance venography (MRV).***

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## Introduction:

Cerebral Venous Sinus Thrombosis (CVST) is very serious disorder with a high mortality approximately 30%.[1] Cavernous sinuses are paired interconnected trabeculated cavities located on either side of sella turcica, superior to sphenoid sinus and posterior to optic chiasma- receive venous drainage from face, orbit, sinus or brain through valve less veins.[2] Incidence of CVST is about 3-4 in a million in USA[1] and 1% to 5% of all cases of stroke worldwide. It can affect people of all races; usual age range is between 25 and 35 and more common in women (1.79:1).[2] Common causes are pregnancy, puerperium and hypercoagulable states as SLE, taking oral contraceptive pills (OCP) (20% higher risk), inherited prothrombotic tendencies as factor V Liedan mutation, Protein C & S, antithrombin deficiency (10-15% cases)[3]. Staphylococcal infection (90%), fungus-aspergillous or rhinosporius, diabetes mellitus, intracranial hypertension or history of head injury can also be risk factors. CVST less common now a days because of widespread aggressive antibiotic use.[4] A patient with persistent headache (90% cases) with an unilateral eye swelling rapidly progressing to bilateral involvement within 24-48 hours with or without features of cranial nerve involvement is a typical feature of CVST. Other differential diagnosis need to be ruled out along with combined consultation with an opthalmologist and a neurologist[5] and also by some relevant investigations as blood test features (raised total count with neutrophilic leucocytosis, CSF findings normal and fundoscopic examinations). Contrast enhanced CT (CECT) or MRI along with MRV of head is the most sensitive and specific imaging technique to diagnose CVST from.[5] Prompt treatment with low molecular weight heparin and mannitol can save life. Sometimes steroids provide symptomatic relief. Broad spectrum antibiotics with early and aggressive high doses for 2-4 weeks needed to cover wide spectrum of infective agents.[6] Anticoagulants in acute stage is not advised as might lead to intracranial haemorrhage or haemorrhagic infarct as also thrombectomy or streptokinase. All patients need long term treatment with oral anticoagulants.[7] Delay in prompt diagnosis and treatment might jeopardise patient's life. Among those who survives a few might have persistent seizures or long term other health problems of

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headache or some degree of vision loss, coagulation problem (pulmonary embolism) or infection (meningitis or sepsis or lung infection/pneumonia) or even pituitary insufficiency or hemiparesis or visual steal syndrome. Fortunately, incidence has markedly reduced now with the advent of effective antimicrobial & anticoagulants use.[8]

**Case Summary**

Our patient M, a 22 year old African woman who worked as a shop assistant, has been transferred from local clinic to tertiary hospital of South Africa in a state of early labour with a history of pregnancy for 31 weeks and pregnancy induced hypertension. This was her 1st pregnancy and a planned one. She was married for 7 months and did not use any form of contraceptives including OCP. She was neither smoker nor alcoholic nor any high risk behaviour. She had regular menstrual cycle with average flow and duration and was in regular antenatal check-ups where all standard screening tests were normal till 29 weeks, when her blood pressure (BP) was found to be 140/95 on more than 2 occasions but there was no proteinuria. Ultrasound and routine tests for pre-eclampsia protocol revealed no abnormality. Anti-hypertensive medication has been started. She felt well, her blood pressure were controlled, urine remained free of protein. At 30 weeks when she had premature contraction, was admitted to local clinic where steroid and tocolytics started but unfortunately at 31 weeks she went to preterm labour as evidenced by effacement and dilatation of cervix of 4 cm. As neonatal facilities were lacking in local clinic she was transferred to tertiary hospital when her cervix was found already 6 cm dilated with a single fetus presented by vertex and BP 130/76 mmHg. Labour was allowed to progress, membrane ruptured with clear fluid, a clip electrode was applied to fetal scalp for continuous fetal monitoring. Spontaneous delivery occurred after 3 hours, a female baby of 1.4 kg in apparently good condition (transferred to NICU) was delivered. Her Blood Pressure remained stable throughout labour, only needed entonox for labour analgesia. She was transferred to postnatal ward where she preferred to stay near the baby. Her condition remained stable including BP, urine output, lochial discharge with unengorged breasts as breast milk was collected by pump regularly for baby.

On 5th day of postpartum she felt unwell, initially nauseated then vomited small amount, twice and also dysuria. On examination, temp 38.2$^{O}$c, urine protein 1+, pulse and BP slightly raised. Urine sample sent for culture, antibiotics started. On next day she developed headache mainly over crown of head and occipital location. Headache was persistent, throbbing in nature, accompanied with photophobia. On 7th postpartum day, dysuria resolved, urine culture negative but pyrexia and headache persisted. She was in positive fluid balance, no signs of dehydration and face seemed puffy with periorbital oedema, GCS 15/15, normal speech and no oedema of hands, legs or sacrum. Temp remained 38.2$^{O}$c and cardiovascular system, respiratory system no abnormality noted. Extremities normal with no signs of deep vein thrombosis (DVT) or thrombophlebitis, no engorged breast. Abdomen slightly distended consistent with recent pregnancy and delivery. A neurologist and an opthalmologists were asked for their opinion. She was reluctant to move her head but can put her chin to chest, Kernig's sign negative. Pupillary reflexes to light and accommodation were equal and symmetrical though some restriction to upward and lateral eye movement. There was no loss of visual acuity and no nystagmus. Fundoscopic examination of optic disc difficult because of photophobia, it revealed no papilledema, no signs of hypertensive retinopathy and disc margins on both sides blurred medially. Systemic examination of cranial nerves, ears and tympanic membranes normal. Tendon reflexes equal and symmetrical. Ultrasound for DVT and thrombophlebitis was negative, liver function tests all were within normal range. EMI, skull X-ray, EEG did not reveal any pathology. ANA, double stranded DNA & anticardiolipin antibody done to exclude SLE. With persisted nausea and headache- MRI along with MRV advised- findings consistent with cavernous sinus thrombosis with blood clots in transverse and sigmoid sinuses. There were prominent collateral channels draining blood downwards to veins of Sylvian fissure. No aneurysm or arteriovenous malformation showed. The internal cerebral vein and inferior sagittal sinus were dilated.

**Fig.-1 :** *MRI with MRV findings of CVST:*

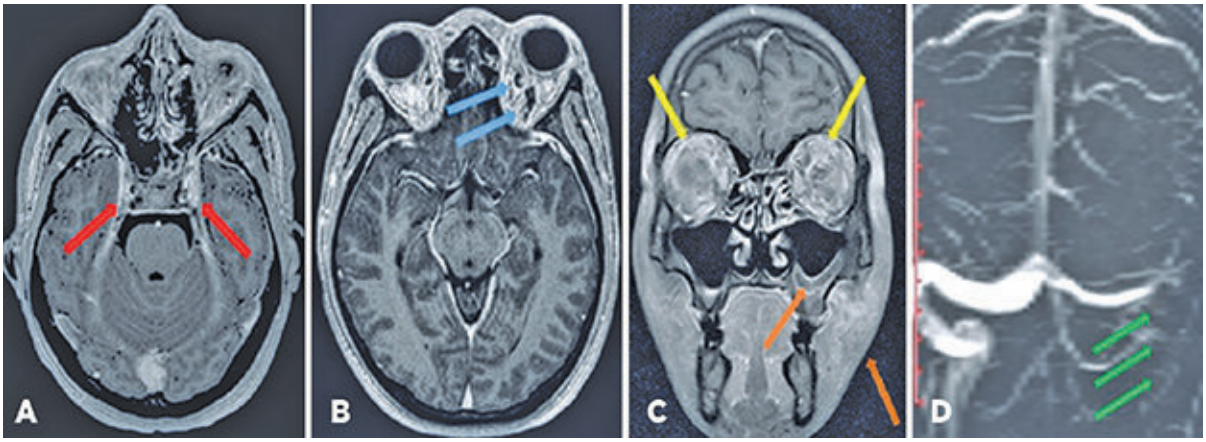


**Fig.-2 :** *MRI with MRV findings of CVST :*

Neurosurgeon also called on findings of MRV, joint decision was to start low molecular weight heparin and mannitol infusion to improve cerebral blood flow and to reduce cerebral oedema. She was on close supervision. Her condition found to be stable on 3rd day. As good collateral circulation established itself so neurosurgeon opine not to use any anticoagulants or streptokinase nor proceed for thrombectomy. Ophthalmologist excluded any visual defect. Her condition gradually resolved over the next 6 days when she was discharged home with specific advice of oral anticoagulants for one year but to avoid oral contraceptives. She attended obstetric as well as medical unit where she was found entirely symptom free. Ophthalmological examination findings normal with slight blurring of disc margin. She was advised to avoid pregnancy for at least 2 years and to use barrier methods as hormonal or intrauterine systems not suitable for her and to repeat MRI along with MRV before next pregnancy and to continue oral anticoagulants for the same duration with INR follow up.

**Discussion:**

Cerebral venous thrombosis is one of the fatal complications of puerperium, symptoms usually appeared in 1st 3 weeks of postpartum in most cases with severe headache being common complaints CVST itself can be fatal and percentage of mortality increases when associated with thrombosis at other sites. Sometimes it is confused with cerebral thrombophlebitis secondary to infection in middle ear, face or skull. Frequently found to be associated with pre-eclampsia specially postpartum.[8] It should be suspected in any previously healthy woman who develops a syndrome of paresis, headache, vomiting or nausea, convulsions, speech and eyesight problems and confusion during pregnancy or after delivery. Thrombosis of Cavernous sinus is specially associated with severe persistent headache, nausea, vomiting and a neuro palsy without any identified aetiology and development of acute bilateral papilledema. The face and scalp found markedly congested. Lumbar puncture might show a slight increase in CSF pressure otherwise no significant finding to differentiate it from meningitis. An EEG, CTs scan, or EMI is not always helpful. The diagnosis is confirmed by MRI and MRV. The treatment is not well documented. As the role of anticoagulants are doubtful in acute phase as it can lead to intracerebral haemorrhage or haemorrhagic infarction.

Puerperium is a very vulnerable time as mothers are prone to various types of infective and non-infective complications and some of those can be fatal. Most cases of pyrexia and headache are related to obstetrics and genitourinary tract infection[9]. Literature review showed Yaqiang Li et al, in case report of CVST presenting with rapidly progressive dementia but 5 days of anticoagulant treatment-symptoms improved, then fully healed with no signs of recurrence within 1 year. Another case study, by Mohamed Abdullahi Hasan et al showed, a 35 years old lady, para 7 developed similar headache with vomiting at 16 days postpartum but recovered fully with oral anticoagulant of LMWH(Enoxaparin).

Prognosis is variable- varies from total recovery like our patient to survival with residual morbidity or death. Perspective study shows independent survival rate

approximately 80%. The risk of recurrence of CVST in next pregnancy or puerperium fortunately is low.

**Conclusion:**

CVST may present with extremely varied signs and symptoms. Diagnosis is confirmed by MRI along with MRV. It is potentially life threatening if undiagnosed or delay in diagnosis but remains a treatable disorder. So, CVST should be considered in every woman with neurological symptoms in pregnancy and or puerperium. There should be broad differential diagnosis in females experiencing severe headaches with seizure and or visual field problems on postpartum period. Maternity Healthcare workers should enhance the care for these type of patients by seeing causes rarer than postpartum eclampsia or meningitis and conducting tests that could differentiate them. It is important for early diagnosing and managing for prevent complications and death.

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