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A RARE CASE OF PRIMARY UEDVT AND CVT IN PREGNANCY

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Abstract : Upper extremity deep vein in thromboses (UEDVT) is rare. In UEDVT, Internal jugular vein thrombosis (IJVT) is a serious event with a potentially fatal outcome. Complications include pulmonary embolism, sepsis with septic emboli to different organs and tissues as well as intracranial propagation of the thrombus with cerebral edema. As any thrombosis, Internal Jugular Vein Thrombosis is precipitated by Virchow's triad endothelial damage, alteration of blood flow and hypercoagulability. The history and examination in patients with an Internal Jugular Vein Thrombosis may be vague and misleading. Patients may present with a painful swelling of the neck but they may also be absolutely asymptomatic. The prevalence of CVT in Indian population is about 4.51000 obstetric admissions with a case fatality rate of less than 10. CVT associated with pregnancy and puerperium has a more acute onset and better prognosis than thrombosis due to other causes. Imaging procedures frequently used to diagnose an Internal Jugular Vein Thrombosis include sonography with color-coded duplex sonography, computed tomography, magnetic resonance imaging as well as magnetic resonance venography. Since the advent of newer imaging techniques angiography and other contrast studies are rarely necessary to diagnosis sinovenous thrombosis as angiography is associated with significant complications like thrombosis, risk of propagating infection intracranially in septic thrombophlebhitis, time consuming and cumbersome. Potential pitfalls of angiography include mixing of contrast and unopacified blood from the hemisphere opacification of intradural collaterals which may mimic filling of sinus. Up to date, there is no standardized treatment regimen for patients with an Internal Jugular Vein Thrombosis.

Keyword :UEDVT, IJVT, CVT, Thrombosis, Pregnancy, Puerperium.

INTRODUCTION

Thrombosis of Internal jugular vein is relatively rare condition. It is generally associated with central venous catheterization, neck surgery, trauma, endocrine abnormality, coagulation disorder, hyper coagulable status, local infection and malignancy. Cerebral Venous Thrombosis (CVT) is any thrombosis occurring in intracranial veins and sinuses, is a rare disorder affecting 5 persons per million per year with huge regional variations in developing countries; pregnancy and puerperium are the common causes of CVT in young women. Significant risk was associated with, use of oral contraceptive pills, caesarean delivery, increasing maternal age, presence of co-morbid conditions like hyperemesis, intercurrent infection and maternal hypertension. Internal jugular vein thrombosis presents with neck pain and swelling of face. Doppler USG, CT, MRI scan have been described for diagnosis of Internal jugular vein thrombosis.

CASE REPORT

25 years old P2L2, with 2 full term normal delivery, delivered 12 days back on antihypertensives developed head ache on 6th post-natal day and was referred to our hospital as CVT. She presented with c/o head ache for one week. There was no significant past history. Obstetric history was uneventful except pregnancy induced hypertension during intrapartum period during the present delivery O/E patient was conscious, oriented, not anemic, no edema legs, swelling over the right side of the neck below the right mandibular region which was tender, warm, not indurated, PR 80/min, BP 140/90mm Hg, CVS, RS, CNS was normal. Fundus examination is mandatory when CVT is associated with raised intracranial pressure and with persistent headache. Fundus examination was normal. Abdomen was Soft. In Vaginal examination uterus was bulky, lochia healthy. Patient was continued with T.Labetolol. All basic blood and urine investigations were normal. Coagulation profile normal. ECG and ECHO were normal. Doppler USG of neck showed right side Internal jugular vein thrombosis. MRV brain showed right transverse sinus, sigmoid venous sinus thrombosis and right Internal Jugular Vein Thrombosis. Since the advent of newer imaging techniques angiography and other contrast studies are rarely necessary to diagnosis sinovenous thrombosis as angiography is associated with significant complications like thrombosis, risk of propagating infection intracranially in septic thrombophlebhitis, time consuming and cumbersome. Potential pitfalls of angiography include mixing of contrast and unopacified

blood from the hemisphere; opacification of intradural collaterals which may mimic filling of sinus. Patient was started on injection heparin 5000 units IV BD along with antibiotic and other supportive measures. Then patient was switched over to tablet warfarin 7.5mg OD for six months by checking coagulation profile and maintaining INR. Repeat MRV brain after six months of initial presentation showed reduced flow with area of recanalization in right Internal jugular vein, transverse venous sinus, sigmoid venous sinus and revascularization of the right Internal jugular vein could be demonstrated with duplex sonography.

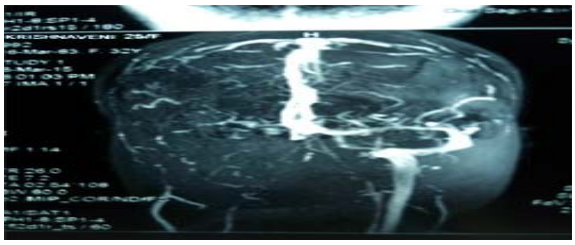

Fig-1 Before Treatment (MRV brain shows right transverse sinus, sigmoid venous sinus thrombosis and right IJV thrombosis)

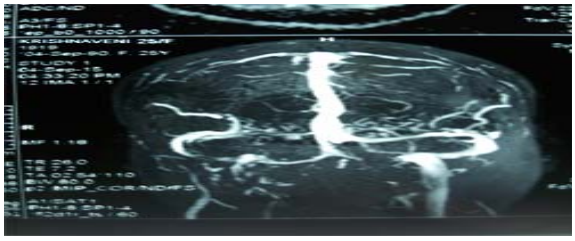

Fig-2 AfterTreatment (MRV brain after six months of initial presentation showed reduced flow with area of recanalization in right IJV, transverse venous sinus, sigmoid venous sinus)

DISCUSSION

The Internal jugular vein begins in then cranium at the conclusion of the sigmoid sinus. It exits the cranium via the jugular foramen and then courses through the anterior neck, lateral to the carotid artery, covered by the sternocleidomastoid muscle for most of its length. It concludes by joining the subclavian vein, thus forming the brachiocephalic vein. Hpercoaguability in pregnancy and puerperium can be attributed to the following pathogenic factors:

I. Increased levels of fibrinogen, factor VII, VIII and X.

II. Diminution in inhibitors of coagulant proteins S.

III. Rise in inhibitors of protein C levels.

IV. Factor V Leiden and Factor II gene mutation is a n extensively studied and proved predisposing factor in pregnancy and puerperium.

V. Platelet count and adhesiveness is also found to increase in pregnancy.

VI. Use of ART is strongly associated with UEDVT.

venous thrombosis results from activation of the coagulation cascade. Anticoagulant therapy is thus a cornerstone in its management. Heparin anticoagulation is a time honoured treatment and is advocated in all cases of CVT irrespective of the aetiology. The aims of heparin therapy are

1. To recanalise the occluded sinus or vein,
2. To prevent the extension of the thrombus,
3. To treat the underlying prothrombotic state,
4. To prevent venous thrombosis in other parts of body (deep vein thrombosis, pulmonary embolism) and
5. To prevent recurrence.

Low Molecular Weight Heparin (LMWH) are now the choice preparations for anticoagulation. As per ACOG 2011 recommends Low Molecular Weight Heparin for treatment of acute venous thrombosis. They have a more reliable dose response pharmacokinetics, better bioavailability, longer half-life, dose independent clearance, minimal requirement for monitoring, low risk for bleeding and a reduced propensity for the platelet inhibition seen with standard heparin. After the acute stage, heparin should be replaced by oral anticoagulation and considering the evidence in deep venous thrombosis, warfarin should be continued for 6-12 months aiming at an INR of two or three. Duration of warfarin is not standardized and the decision should be based on reversibility of underlying cause and anatomic issues of recanalization and collateral flow. Complete recovery is the rule in survivor. Long term prognosis and risk of recurrence are influenced by the nature of underlying disease in patient with systemic disease. These patients are closely followed up to six months and repeat MRV is done after six months of initial therapy. The goal is to prevent any recurrence and any other venous thrombosis and pulmonary embolism. Progesterone only pills is advised as a contraceptive for these patients as it does not significantly affect the lipid metabolism, hemostatic factors, liver function, blood pressure, not shown to increase the risk of thromboembolism and therefore generally allowed.

Complications of the Internal jugular vein thrombosis

Septic emboli, Pulmonary embolism, Elevated Intra Cranial pressure, facial edema, Loss of vision, Intracranial venous thrombosis

Guidelines for the management of CVT

· In patients with provoked CVT (associated with a transient risk factor – Anaemia, Preeclampsia, Internal Jugular Vein Cannulation), vitamin K antagonists may be continued for 3-6 months, with a target international normalized ratio of 2.0-3.0.

· In patients with unprovoked CVT (not associated with above transient risk factors), vitamin K antagonists may be continued for 6-12 months, with a target international normalized ratio of 2.0-3.0.

· For patients with recurrent CVT, venous thromboembolism (VTE) after CVT, or first CVT with severe thrombophilia (ie, homozygous prothrombin G20210A; homozygous factor V Leiden; deficiencies of protein C, protein S, or antithrombin; combined thrombophilia defects; or antiphospholipid syndrome), indefinite anticoagulation may be considered, with a target international normalized ratio of 2.0-3.0.

· For women with CVT during pregnancy, low-molecular-weight heparin (LMWH) in full anticoagulant doses should be continued throughout pregnancy, and LMWH or vitamin K antagonist with a target international normalized ratio of 2.0-3.0 should be continued for 6 weeks postpartum (for a total minimum duration of therapy of 6 months).

· It is reasonable to advise women with a history of CVT that future pregnancy is not contraindicated. Further investigations regarding the underlying cause and a formal consultation with a haematologist or maternal fetal medicine specialist are reasonable.

· It is reasonable to treat acute CVT during pregnancy with full-dose LMWH rather than unfractionated heparin.

· For women with a history of CVT, prophylaxis with LMWH during future pregnancies and the postpartum period is reasonable.

CONCLUSIONS:

Internal jugular vein thrombosis is an uncommon and possibly life-threatening problem. Internal jugular vein thrombosis can be occurred in different pathologies. Early

diagnosis and appropriate management is important to prevent potentially fatal complications from this condition. Thrombosis of the Internal jugular vein is probably underdiagnosed. Since the clinical presentation may be vague or misleading, a high degree of suspicion is required to make the diagnosis. The potential complications such as pulmonary embolism or intracranial propagation of the thrombus may be fatal. Whenever the thrombosis is not caused by an inflammatory process, a malignant tumor should be excluded. We recommend a therapy with intravenous antibiotics as well as a systemic anticoagulation. Ligation or resection of the internal jugular vein is reserved for patients who develop complications despite adequate medical therapy.

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