JK SCIENCE

CASE REPORT

Early Anticoagulation Post LSCS in a Symptomatic Cortical Venous Thrombosis with Protein - S Deficiency

Prabhjit Kour, Sanjay Bhat*, Saurabh Sharma*, Abhinav Gupta*

Abstract

Post partum cerebral venous sinus thrombosis (CVT) in a high risk predisposed individual and its management particularly after a major surgical intervention i.e. lower segment caesarean section (LSCS) is debatable. Guidelines are there to treat after 72 hours of major surgical intervention. We report a case of CVT with generalised tonic clonic seizures as a life threatening complication in whom anticoagulation was started at 48 hours post LSCS without any complications of anticoagulation. We recommend early anticoagulation despite major surgical intervention, so as to avoid the impending life threatening complications following cerebral venous thrombosis in predisposed individuals.

Key Words

Cerebral Venous Thrombosis, Anticoagulation, Hypercoaguable State

Introduction

Cerebral venous sinus thrombosis, a rare condition with variable clinical presentation especially in high risk predisposed individuals like hypercoaguable states, dehydration, oral contraceptives, hormone replacement therapy, pregnancy and puerperium (1). The clinical scenario can be in the form of headache (95%), focal seizures with or without secondary generalization (47%), unilateral or bilateral paresis (43%) and papilledema (41%) (2,3). We report a case of CVT presenting in puerperium, 36 hours post lower segment caesarean section (LSCS) as generalized tonic-clonic seizures in whom anticoagulation therapy was initiated earliest possible (48 hours), despite the major surgical intervention.

Case Report

Twenty nine year female presented to us with history of two episodes of generalized tonic-clonic seizures, occurring 36 hours post LSCS half an hour apart. There was no preceding history of any fever, headache or trauma. Her obstetric history included two previous spontaneous abortions; first one at 10 weeks of gestation and the second one at 14 weeks of gestation. Ante-partum period of current pregnancy was uneventful and the peripartum period including LSCS was uneventful Initial evaluation revealed normal general physical and systemic examination. Laboratory investigations revealed normal serum electrolytes, normal kidney and liver function tests. Magnetic resonance imaging of brain revealed focal cortical and subcortical hyperintensities in both cerebral hemispheres predominantly in parieto-occipital regions with MR venogram revealing complete occlusion of left jugular and transverse sinuses, giving diagnosis of cerebral venous thrombosis (*Fig-1*).

Patient's bleeding time, clotting time and prothrombin time index was normal. Patient's FDP and d-dimer levels were raised; Anti Nuclear Antibodies and Anti Phospholipid Antibody were within normal ranges. Her protein C levels were normal but protein S activity, as happens normally during pregnancy and puerperium, was reduced. Protein S quantitative assay also revealed reduced levels of protein S (45 U/dl) in serum (Normalgreater than 63 U/dl).

Subcutaneous low molecular weight heparin (LMWH) was started 48 hours after her LSCS overlapped with oral Vitamin K Antagonist (VKA)- warfarin. Patient remained asymptomatic and was discharged on oral VKA with advice regarding regular INR monitoring.

Discussion

Thrombosis of the dural sinus and/or cerebral veins (CVT) is an uncommon form of stroke usually affecting young individuals (2). Despite advances in the recognition of CVT in recent years, diagnosis and management can be difficult because of the diversity of underlying risk factors and the absence of a uniform treatment approach. CVT represents 0.5% to 1% of all strokes (3). Multiple

From the Department of Obstetrics & Gynaecology and G Medicine*, ASCOMS & Hospital, Jammu- J&K India Correspondence to : Dr Prabhjit Kour. Assistant Professor, Department of Obstetrics & Gynaecology, ASCOMS & Hospital, Jammu- J&K, India



Fig.1 MR Venogram showing Occlusion of left Jugular (Red) and Transverse Venous Sinuses (Yellow)

factors have been associated with CVT, but only some of them are reversible.

Traditionally, the most frequently postulated mechanism involved in CVT in obstetric patients is a hypercoaguable state (4) associated with dehydration and anaemia. However, other factors may play an important role, such as protein S deficiency, which is common during pregnancy and puerperium (5,6). Deficiency of protein S has been reported in previous cases of CVT in obstetric patients (7,8).

According to the guidelines of the European Federation of Neurological Societies (9), the first line treatment for cerebral venous and dural sinus thrombosis is anticoagulation. The rationale for its use is to favour spontaneous thrombus resolution and to re-canalize the occluded vein or sinus, to avoid thrombus propagation, to treat underlying prothrombotic condition and to prevent complications such as pulmonary thromboembolism. Treatment is usually started with dose-adjusted intravenous heparin between 3000 and 5000 international units or body-weight-adjusted subcutaneous lowmolecular weight heparin, until the patient stabilizes. Treatment for this acute phase is followed by oral anticoagulation with warfarin to prevent recurrence and thrombosis in other parts of the body.

Our patient was treated with anti-coagulation with LMWH within 48 hours of the major surgical intervention (LSCS), though usual recommendation is to start anticoagulation after 72 hours after a major surgery for use of LMWH as per ACCP (American College of Chest Physicians) guidelines (10), overlapped with oral VKA (Vitamin K antagonist).

We recommend early anticoagulation despite major surgical intervention, so as to avoid the impending life threatening complications following cerebral venous thrombosis in predisposed individuals.

References

- 1. Cantu C, Barinagarrementeria F. Cerebral venous thrombosis associated with pregnancy and puerperium: review of 67 cases. *Stroke* 1993; 24:1880-84.
- 2. Bousser MG, Ferro JM. Cerebral venous thrombosis: an update. *Lancet Neurol* 2007;6:162-70.
- 3. Stam J. Thrombosis of the cerebral veins and sinuses. *N Engl J Med* 2005;352:1791-98.
- 4. Inglis TCM, Stuart J, George AJ, Davies AJ. Haemostatic and rheological changes in normal pregnancy and preeclampsia. *Br J Haematol* 1982;50:461-65.
- Comp PC, Thurnau GR, Welsh J, Esmon CT. Functional and immunologic protein S levels are decreased during pregnancy. *Blood* 1986;68:881-85.
- Mackinnon S, Walker ID, Davidson JF, Walker JJ. Plasma fibrinolysis during and after normal childbirth. *Br J Haematol* 1987;65: 339-342.
- Cros D, Comp PC, Beltran G, Gum G. Superior sagittal sinus thrombosis in a patient with protein S deficiency. *Stroke* 1990:21: 633- 636.
- 8. Moreb J, Kitchens CS. Acquired functional protein S deficiency, cerebral venous thrombosis and coumarin skin necrosis in association with antiphospholipid syndrome: report of two cases. *Am J Med* 1989;87:207-210.
- 9. Einhaupl K, Stam J, Bousser MG, *et al*. EFNS guideline on the treatment of cerebral venous and sinus thrombosis in adult patients. *Eur J Neurol* 2010;17:1229-35.
- Guyatt GH, Akl EA, Crowther M, Gutterman DD, Schuünemann HJ; American College of Chest Physicians Antithrombotic Therapy and Prevention of Thrombosis Panel. Executive summary: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest* 2012;141(2 Suppl):e419S-94S.