



Cerebral venous thrombosis in association with oral contraceptive use

VD Maheswari ¹, Manju Maheswari ², Ajay Nair ²

^{1,2} Department of medicine, and ³ Department of Gynecology and Obstetrics, S.M.S. Medical College and Hospital, Jaipur

Key words : oral contraceptive, cerebral venous thrombosis

Introduction

Cerebral venous/sinus thrombosis (CVT/CST) is characterized by intravascular clotting in the venous system of the brain, including the dural sinuses, cortical veins, and galenic system of deep veins ¹. CVT carries a high mortality and may result in neurological sequelae ². Seventy percent of the cases result from a hypercoagulable state ³. Oral contraceptives, even in low dose estrogen formulations induce a hypercoagulable state and thereby predispose to the development of CVT ^{4,5}. We present a case of CVT occurring in association with the use of oral contraceptives.

Case report

A 43 year old housewife presented with sudden throbbing headache with projectile vomiting for last 3 days. A day before admission she developed blurring of vision and diplopia with slurring of speech, followed by two episodes of generalized tonic-clonic seizures. There was no history of fever, trauma, or motor weakness. There was no past history of diabetes, hypertension, tuberculosis, ear discharge, head injury, or episodes of seizures in the past. Her menstrual history revealed polymenorrhea for which she had been prescribed a popular oral hormonal contraceptive MALA-D (Levonorgestryl 0.15 mg + ethynyl estradiol 0.03 mg). She had been taking this oral hormonal

contraceptive regularly for the last 2 years without medical supervision. On examination, she was drowsy and irritable, though responding to verbal commands. Central nervous system (CNS) examination showed neck rigidity with absent Kernig's sign and the presence of left lateral rectus weakness. Fundus examination showed bilateral papilledema. Rest of the CNS examination and that of the other systems were unremarkable except that her blood pressure was 190/110 mmHg. Hemoglobin was 9.3 g/dL, while white cell counts and ESR were normal. Kidney and liver function tests, bleeding time, clotting time, and prothrombin time were normal. Except for low density lipoprotein level of 159.4 mg/dL her lipid profile was also normal. CT scan showed multiple hyperdensities in the left parietal cortex, suggestive of hemorrhagic infarct. MRI angiography was normal while MRI venography revealed thrombosis within the left transverse and sigmoid sinuses with hemorrhagic venous infarcts in the left temporo-occipital region (Figures 1 and 2). Antinuclear antibody, LE cell, antiphospholipid antibody, antiphospholipid antibody and VDRL were negative. Patient could not afford the tests for familial hypercoagulable states. She was treated with antiepileptics, antihypertensives and low molecular weight heparin. She recovered completely and was discharged on oral anticoagulants. At 6 months followup, she was normotensive without antihypertensives and was free of convulsions.

Discussion

The association of oral contraceptives with CVT suggests that it would be worthwhile to take a personal and family history of venous thrombosis (CVT/DVT) when prescribing oral contraceptives and for patients with such

Paper received on 13/02/2004 ; accepted on 17/11/2004

Correspondence :

Dr. V. D. Maheshwari

Professor, Department of Medicine

3/70, Jawahar Nagar, Jaipur 302 004.

a history counseling about alternative methods of contraception should be considered ^{4,6}. Regular followup of the patient on oral contraceptive for drug induced hypertension, deep vein thrombosis, and other cardiovascular or cerebrovascular complaints may go a long way in preventing such complications.

References

1. Bousser MG. Cerebral venous thrombosis: nothing, heparin or local thrombolysis ? *Stroke* 1999;30:481-3.
2. de Bruijn SFTM, Buddo M, Teunisse et al. Long term outcome of cognition and functional health after cerebral venous sinus thrombosis. *Neurol* 2000;54:1687-9.
3. van Gijn J. Cerebral venous thrombosis: pathogenesis, presentation and prognosis. *J Soc Med* 2000;93:230-3.
4. Martinelli I, Sacchi E, Landi G et al. High risk of cerebral vein thrombosis in carriers of a prothrombin gene mutation and in users of oral contraceptives. *N Engl J Med* 1998;338:1793-7.
5. Buccino G, Scoditti V, Pini M et al. Low estrogen oral contraceptive as a major risk factor for cerebral venous and sinus thrombosis. *Italian J Neurol Sci* 20;231-5:1999.
6. de Bruijn SFTM, Stam J, Koopman MMW et al. Case control study of risk of cerebral sinus thrombosis in oral contraceptive users who are carriers of hereditary prothrombotic conditions. *BMJ* 1998;316:589-92.