DIAGNOSIS AND MANAGEMENT OF POST PARTUM CEREBRAL VENOUS SINUS THROMBOSIS

by

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SUMMARY

This report gives an analysis and detailed description of pathophysiology and clinical management of cases of cerebral venous thrombosis during puerperium. Although a cerebral vascular accident is a less common complication of pregnancy which carries high morbidity and mortality merits, prompt diagnosis and treatments by close co-operation between obstetrician and neurophysician.

Introduction

Cerebral venous/venous sinus thrombosis is not uncommon in young females in India, being 4.5 per thousand obstetrical admissions as compared with western incidence of one per 1.5-10 thousand pregnancies (Bansal et al 1980), while cerebral arterial thrombosis is more common in western countries (Cross et al 1968; Amias, 1970). Various pathogenic hypothetical mechanisms have been put forward viz., spread of thrombosis via vertebral veins from pelvic veins during labour, damage to cerebral venous sinuses due to raised pressure at the time of bearing down and increased tendency of blood to coagulate during pregnancy and pucrperium (Kendall, 1948, 1980).

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Thrombosis of cerebral veins may occur without involvement of a large sinus. Veins on the surface of brain pass from superficial middle cerebral vein to the superior longitudinal sinus. These cortical veins lie in sulci and enter the sinus obliquely in a direction contrary the flow of blood in it, a fact which may possibly predispose to stasis and thrombosis. Occlusion and thrombois. Occlusion and occasional rupture of the tributeries of these veins leads to white and red infarction of cortex and subadjascent white matter which is being drained by these occluded veins. Eleven cases of cerebral venous/venous sinus thrombosis managed by at Dr. S.N. Medical College attached hospitals are reviewed in detail. Most of the times these cases present with convulsions and/or unconciousness at the time of/or after the delivery and mimic eclampsia. We

have reported these 11 cases out of 148 cases of eclampsia to emphasise that cerebral venous sinus thrombosis must be kept in mind as one of the differential diagnosis when such a patient is admitted at the time of confinement.

Material and Methods

Eleven cases who developed cerebral venous sinus thrombosis during puerperium have been studied. The diagnosis was mainly clinical, based on established criteria (Amias, 1970). A detailed history, thorough general physical and neurological examination followed by routine investigations (Hb, TLC, DLC, BT, CT, ESR, complete urine blood urea, blood sugar) were carried out in each case, lumbar puncture and C.S.F. examination (cell pattern and biochemical) was also done. Eclampsia, epilepsy, prehepatic, diabetic and uraemic coma, meningitis, intracranial, haemorrhage and tumors were excluded by appropriate investigations. EEG and angiographic study could not be possible due to lack of facilities.

Observations

All but 3 (32, 35, 35 years) were bellow 25 years. Ten out of 11 belonged to rural population and none had ever attended any antenatal clinic. All of them had spontaneous normal delivery/abortion at home, being attended by T.B.D. Cerebral venous thrombosis was commonly seen in primipara (5) as compared to multipara (3) and rest 3 were second para. Majority of cases (7) developed it within 10 days, 2 cases between 10-14 days, 2 cases after 3 and 6 weeks after confinment.

Focal/generalized convulsions (6), muscular weakness in half of the body (6), deterioarted level of conciousness (4), and headache were the common symptoms (Table I). Five cases had fever and only 1 patient developed aphasia. All patients were anaemic. None of the patients had oedema, albuminuria and hypertension. TLC, DLC, BT, CT, urine, blood sugar and blood urea estimations done in each case who had high ESR e.g., 75 mm 1st hour. On neurological examination only 7 patients had unilateral hemiplegia, unilateral planter extensor (2 cases) without paresis, neck rigidity was present in 2 cases. One patient was having supranuclear seventh nerve palsy. None had locked jaw. Fundoscopy revealed papilloedma in 2 cases only. Lumbar puncture and CSF examination was normal in all excepting 2 cases who had raised tension, turbid and few R.B.Cs explainable on the basis of red infarct which may follow the venous thrombosis. General nursing, antibiotics selective and guarded anticoagulant therapy along with physiotherapy could save the lives of 9 patients, even though 2 cases expired of gram negative septicaemia and sudden respiratory failure respectively.

Discussion

Cerebral venous thrombosis is more common in Inlia. Symptoms usually develops within first 10 days of the delivery. In this series more primigravida (50%) were affected while Bansal, et al (1980) have reported it to be more common in multigravida. Diabetes, obesity, hypertension, liver disease, pre-pregnancy contraceptive pills have not been found to be the precipitating factors. Clinical picture of cerebral vein thrombosis is diagnostic. A puerperal woman

TABLE I

Age in yrs.	Day of on set	Symptoms	B. P.	Oedema/ albumin	Signs	Fundus	CSF	Treatment	Prognosis and remarks
;2.	3.	4.	5.	6.	7.	8.	9.	10.	11.
16	1st	Focal convulsions, fever, unconsciousness, weakness, right half of body	120/70	Nil/Nil	Rt. hemiplegia, Rt. Supranuclear 7th N. palsy	NAD	Normal	Antibiotics general measures, physiotherapy	Improved
35	10th	Focal convulsions, Jaw stiffness	120/70	Nil/Nil	Right hemiplegia, bilateral, planter extensor	NAD	Normal	Anticoagulant mennitol, physiotherapy, general measures	Improved (ESR 75 mml/hr.)
17	6 weeks	Fever, headache, semi conscious	100/50	Nil/Nil	Planter bilateral extensor, hemiplegia	Papillo-edema	NAD	Antibiotics, anticoagulant steroids, mennitol, general measures	Expired of gram-vepeptic aemia
23	13th	Following abortion convulsion, fever	110/80	Nil/Nil	Neck rigidity +, Kernig sign	NAD ±	Turbid RBC biochemical normal, no pus cells	Antibiotics menitol, encephabol, physiotherapy	Improved
17	1st	Focal convulsions left sided weakness	110/80	Nil/Nil	Left sided, hemiplegia	NAD	Turbid haemorrhagic due to red infarct.	Antibiotics, anticoagulant, Physiotherapy, Encephabol, General measures	Improved
35	10	Focal convulsions, Jaw stiffness	130/90	Nil/Nil	Neck rigidity ±	Early palil-loedema	NAD	Anticoagulant antibiotics, mannitol, anticonvulsant	Improved

21	7	Restlessness, muscular twitching and convulsions on left side	100/60	Nil/Nil	Lt. sided hemiparasis	NAD	NAD	Anticonvulsant, antibiotics, mennitol, anticoagulant, physiotherapy	Improved
25	12	Fever, weakness Rt. half of the body	100/80	Nil/Nil	Right sided hemiplegia	NAD	NAD	Antibiotic, mennitol, steroids	Improved
32	8	Weakness left sided, aphasia, semiconsciousness	100/80	Nil/Nil	Lt. sided hemiplegia	NAD	NAD	Mennitol, lomodex, antibiotics, physiotherapy	Improved
19	10	Irrelevant talk abnormal behaviour	100/80	Nil/Nil	Unequal dilated pupils, right plantar extensor	NAD	NAD	Antibiotics, general measures	Expired (Respiratory failure)
20	20	Fever, left sided weakness, restlessness	100/80	Nil/Nil	Lt. sided plantar extensor	NAD	NAD	Antibiotics, mennitol, steroids	Improved

who suddenly develops focal/generalized convulsions, parasis, deterioration in the level of conciousness and headache without any history of hypertension, P.E.T., diabetes, epilepsy etc., cerebrospinal fluid examination is usually normal but sometimes may show raised pressure, turbidity and varying degree of RBC and WBC can be labelled as a patient of cerebral venous thrombosis. Clinical picture of cerebral venous thrombosis may vary, because the development of these symptoms depends on the site and extent of the thrombosis of the sinuses, cortical veins or deep veins and the potentialities of the collateral circulation. All degree of mono/hemiparesis, crossed hemiplegia of face and limbs without speech and visual distrubances can occur. Focal or general seizures are due to local cortical irritation caused by cerebral oedema, venous engorgement and may be followed by paralysis in due course of time.

In cerebral arterial thrombosis, patients are usually concious, seizures are rare. There is dense hemiparesis, papilloedema is absent, the CSF is normal, and neurological deficit persists longer, positive angiographic findings are more common and prognosis for life is good, while cerebral venous sinus thrombosis carries higher mortality and those who survive have very little disability. Cases of meningitis have high fever, neck rigidity, positive, Kernig's sign and CSF is full of pus cells, hence can be safely excluded. Intracranial haemorrhage due to rupture of berry aneurysm is quite rare in puerperium and can be safely excluded by absence of severe headache, marked neck stiffness and CSF full of blood even visible to naked eyes.

Other common diseases like eclampsia, diabetic coma, prehepatic coma, epilepsy,

cardiac stroke can be excluded by detailed history, clinical examination and relevant investigations.

Prophylactic antibiotics, selective and guarded anticoagulant therapy, anticonvulsants as and when needed alongwith general care of the patients saves the life of the majority of the patients. Blind anticoagulant therapy always presents a fear of haemorrhage from ruptured tributaries of affected vessels. Out of 11 cases only 2 patients (18.2%) could not be saved who died of gram negative septicaemia and sudden respiratory failure. Probable mortality given varies from 30% to 50% (Barner, 1980) Bansal et al (1980) have reported 27.5% mortality in their series of 138 cases.

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