CRITICAL ANALYSIS OF CAUSES AND COURSE OF CEREBRAL VENOUS THROMBOSIS IN PREGNANCY AND PUERPERIUM

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CONTENTS

INTRODUCTION

Women in pregnancy or puerperium can suffer from several neurological disorders but a few of them can occur or deteriorate because of pregnancy. The classic cerebrovascular accident during pregnancy and puerperium is cerebral venous thrombosis.

Primary cerebral venous thrombosis occurs in puerperium in young women and is a major cause of stroke in young females in India. Puerperal CVT is 10-12 times more common in India compared to west and accounts for 10-12% cases of stroke in young with CT / MRI evidence of haemorrhagic or ischemic infarcts in brain.

Puerperal CVT is primary in contrast to the secondary CVT seen both in men and women after brain injury, infections, metastatic and non metastatic lesions.

Mortality could be high in CVT 10-30% but with supportive, symptomatic as well as specific therapy with heparin, mortality has been reduced and in most of the cases, recovery is excellent without any residual disability.

AIM OF THE STUDY

The aims of the study are

a) To find out the incidence of cerebral venous thrombosis at Government Rajaji Hospital, Madurai.

b) To analyse the symptoms, causes, course, prognosis, morbidity and mortality in cerebral venous thrombosis.

c) To study the importance of early diagnosis and early introduction of planned therapy.

HISTORY OF CEREBRAL VENOUS THROMBOSIS

1. The syndrome of intracranial venous and sinus thrombosis termed as cerebral venous thrombosis (CVT) was first recognized in early part of 18th century when Ribe (1825) described in a 45 year old man, the clinical and autopsy spectrum of superior sagittal sinus thrombosis.
2. The first ever description of superior sagittal sinus thrombosis occurring in puerperium was by Abercrambie in 1828.
3. For more than a century, several cases were clinically diagnosed as puerperal hemiplegia, puerperal aphasia and late postpartum eclampsia which proved to be CVTs at autopsy.
4. During the 1940s Sir Charles Symonds, J. Purdon Martin and H.L. Sheehan defined the clinical syndrome and made an antemortem diagnosis of CVT.
5. Srinivasan encountered 50 cases of severe CVT amongst 1000 deliveries performed per year. It has been estimated that the prevalence rate in developing countries is

approximately 10 times more than that in developed countries.

6. In 1942, Stansfield was the first to treat CVT with heparin.

7. With the widespread use of angiography, CT and then with the advent of three dimensional MR flow imaging, it has become easier to sort out other conditions and also it has been shown that the prevalence of CVT is more common than reported previously and carries a less serious prognosis.

ANATOMY

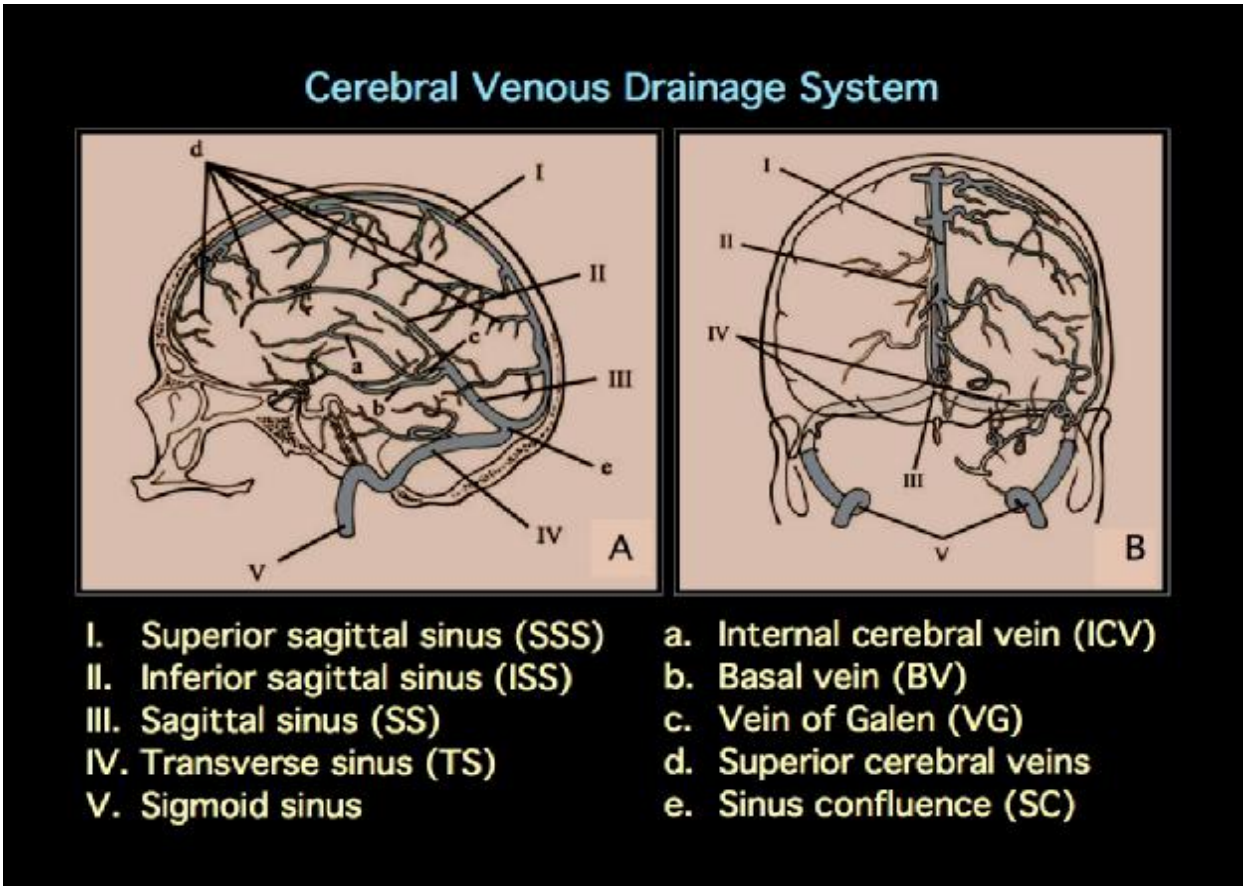
The supratentorial venous system can be divided into a superficial group and a deep group. The superficial system is important in evaluation of venous occlusion since they may demonstrate an abnormal filling sequence and flow pattern. The deep system is important in evaluating the ventricular size and mass effects.

The superficial system

The lateral cerebral convexity above the Sylvian fissure is drained by anterior frontal, central and parietal veins. These lateral veins also receive venous blood from the medial surface of the cerebrum just before they drain into the superior sagittal sinus. The largest vein above the Sylvian fissure is the Vein of Trolard. This is usually located in the parietal region.

The superficial middle cerebral veins drain the lateral surface of the cerebrum adjacent to the Sylvian fissure. The superficial middle cerebral vein usually drains posteriorly into the transverse sinus but may also drain anteriorly into the spheno parietal sinus or medially into the deep middle cerebral vein.

ANATOMY OF CEREBRAL VENOUS SINUSES



The lateral surface of the cerebrum beneath the Sylvian fissure and inferior surfaces of the temporal and occipital lobes drain directly into the transverse sinus. The largest lateral vein beneath the Sylvian fissure is the vein of Labbe.

Deep System

The deep venous system consists of

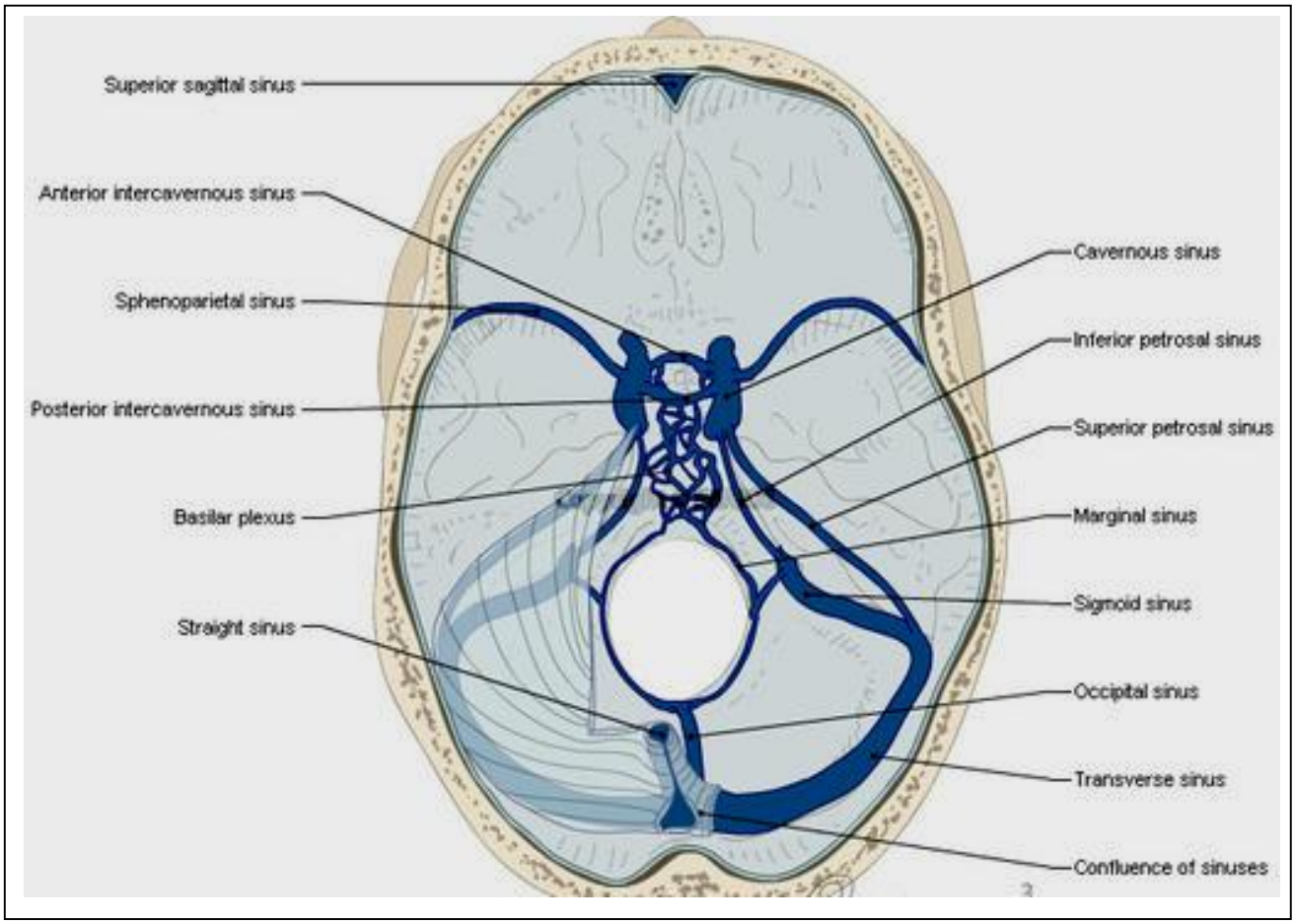
1. The internal cerebral veins
2. Basal veins of Rosenthal
3. Thalamic veins

These veins drain into the great vein of Galen and then into the straight sinus. The internal cerebral veins which lie within 2 mm of the midline are the most important deep vein since they can be used to diagnose midline shifts.

The venous sinuses of the Duramater

The sinuses of the duramater are venous channels which drain the blood from the brain and the bones of the cranium. They are situated between the two layers of the duramater and are lined by endothelium continuous with that which lines the veins.

DURAL VENOUS SINUSES

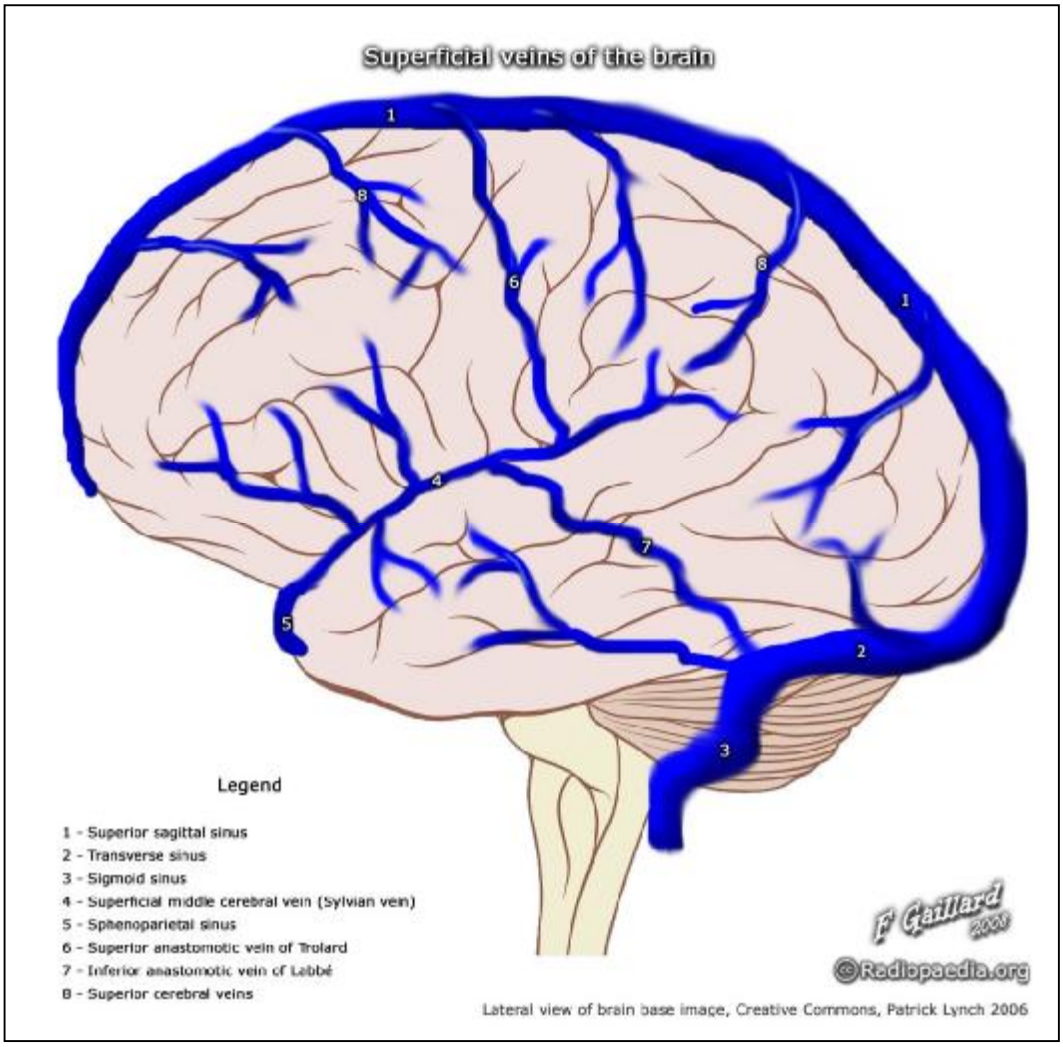


The superior sagittal sinus

This occupies the convex margin of the falx cerebri. It commences in front of the crista galli, runs backwards, grooving the inner surface of the two parietal bones and the squamous part of the occipital bone. Near the internal occipital protuberance, it deviates to one or the other side, usually the right and is continued as the corresponding transverse sinus. Its inner surface presents the openings of the superior cerebral veins, projecting arachnoid granulations and also communicates with venous lacunae situated in the duramater near the sinus. The superior sagittal sinus receives the superior cerebral veins and near the posterior extremity of the sagittal suture, veins from the pericranium.

The confluence of the sinuses is the term applied to the dilated posterior extremity of the superior sagittal sinus. It is on one side, generally the right of the internal occipital protuberance and from it, the transverse sinus of the same side is derived. It also receives blood from the occipital sinus and connects with the commencement of the opposite transverse sinus.

SUPERFICIAL VEINS OF THE BRAIN



The inferior sagittal sinus

It is in the posterior half or two-thirds of the free margin of the falx cerebri. It increases in size posteriorly and ends in the straight sinus. It receives several veins from the falx and occasionally a few from the medial surfaces of the cerebrum.

The straight sinus

It is in the junction of the falx cerebri and tentorioum cerebelli. It runs backwards and downwards, continuing the inferior sagittal sinus to the transverse sinus of the side opposite to that into which the superior sagittal sinus is prolonged. Its terminal part communicates with the confluence of sinuses. Besides the inferior sagittal sinus, it receives some of the superior cerebellar veins and at its commencement, the great cerebral vein.

The Transverse sinuses

These are of large size and begin at the internal occipital protuberance, one generally the right being the direct continuation of the superior sagittal sinus, the other of the straight sinus. Each transverse sinus passes laterally and forwards to the posterolateral part of the petrous part of the temporal bone where it curves down as

the sigmoid sinus. The transverse sinuses are triangular on transverse section and are frequently of unequal size, the one draining the superior sagittal sinus being the larger. Where they become continuous with the sigmoid sinus, they are joined by the superior petrosal sinuses and in their course they receive inferior cerebral, inferior cerebellar and diploic veins and the inferior anastomotic vein.

The sigmoid sinuses are direct continuations of the transverse sinuses beginning where the latter leave the tentorium cerbelli. Each sigmoid sinus curves downwards and medially in a deep groove on the jugular process of the occipital bone and then turns forwards to become the superior bulb of the internal jugular vein in the posterior part of the jugular foramen.

APPLIED ANATOMY

Cerebral venous circulation exhibits following anatomical characteristics that influence clinical profile and management of CVT :

a) Cerebral veins and sinuses have neither any valves nor tunica muscularis. Absence of valves permits blood flow in various directions while absence of tunica muscularis permits veins to remain dilated.

b) Intercommunication between various venous sinuses either via communicating veins (vein of Trolard, & Vein of Labbe) or through merger into each other especially at torcular Herophili, explains lack of correlation between the severity of underlying pathology and infrequent clinical symptomatology. Even recovery that is complete or with minimal sequelae, is explained by this fact.

c) Venous sinuses are located between two rigid layers of duramater. This prevents their compression when intracranial pressure rises.

d) Emissary veins from scalp, face, paranasal sinuses and ears etc., diploic veins, and meningeal veins drain into cerebral venous sinuses either directly or via venous lacunae. This explains the frequent

occurrence of CVT as a complication of infective pathologies in the catchment areas, e.g., cavernous sinus thrombosis in the facial infections, lateral sinus thrombosis in chronic otitis media and sagittal sinus thrombosis in scalp infections.

e) Superficial cortical veins drain into superior sagittal sinus against the blood flow in the sinus, thus causing turbulation in the blood stream that is further aggravated by the presence of fibrous septa present at inferior angle of the sinus. This fact explains greater prevalence of superior sagittal sinus thrombosis.

f) Arachnoid villi are located in the walls of superior sagittal sinus and drain CSF into the sinus. So, thrombosis when it develops in the sinus, especially in the posterior segment, blocks villi and leads to intracranial hypertension and papilloedema.

g) Deep cortical veins, like arterial circle of Willis, also form a venous circle around midbrain, comprising of basal vein of Rosenthal formed by the merger of anterior and middle cerebral veins, formed by the drain into internal cerebral vein posteriorly that merges into the vein of Galen. These basal veins become engorged in superior sagittal sinus thrombosis and can be demonstrated by venous

transcranial doppler ultrasonography in 80% cases.

i) Superior sagittal sinus is the commonest sinus to be involved in aseptic CVT .

Frequency of various sinuses involved in aseptic CVT

Superior sagittal sinus	-	72%
Lateral sinus (combined)	-	70%
Straight sinus	-	13%

REVIEW OF LITERATURE

Puerperal CVT is seen frequently in India. Pregnancy increases the risk of stroke in young women by 13 fold. Apart from the hypercoagulable state of pregnancy, dehydration, anaemia and protein deficiency play a part. The incidence in Madurai is 1 in 250 deliveries (K.Srinivasan, 1984[37])

Age seems to be an indeterminant factor. It is more common in multiparous women.

Labour and delivery are usually normal. A few cases have followed preeclampsia. 95% of pregnancy associated strokes in our country are CVTs.

In India, the onset of symptoms is most commonly between 5-10 days postpartum with a few cases occurring within 3 days following delivery, whereas in Western Countries, 95% of the cases occur more than one week after child birth. Two reasons have been suggested for this problem. First, water restriction for the first few days after delivery which is a common midwifery custom in our hot country results in dehydration. This is the most important factor as judged from the time of onset and the relative sparing of women

delivered in Institutions. Second, diet during the III trimester for Indian village women is unusually high in calories and fat. This factor could affect coagulation.

The report by C. Prakash & Surinder Singh[12] recorded 21 cases of venous and sinus thrombosis which had occured in puerperium over a period of four years. 14 cases had evidence of cerebral venous thrombosis and 7 of sagittal sinus thrombosis. The age of the patients varied from 18-45 years.

There were only two cases of primipara, the rest were multipara. Delivery was full term normal in 19 cases and breech presentation in 2 cases. Evidence of puerperal sepsis was present in 2 patients and one had signs of thrombophlebitis in the leg. The interval between the delivery and onset of symptoms varied from 3 to 28 days.

Headache was present in 12 patients, convulsive fits in 8, unconsciousness in 6, aphasia in 5 cases.

Blood pressure was within normal limits in all the cases. Fundus examination revealed papilloedema in 7 patients, two of these patients later developed optic atrophy.

In Janaki et al series[38], the incidence of vascular thrombosis was 1.03% of total medical admissions. The group of vascular thrombosis consisting of arterial and venous thrombosis was peculiar to pregnancy and puerperium. Janaki has reported arterial thrombosis as unlikely, unless cardiovascular and renal causes are present. The maximum number of cases was in the third decade. All the cases were of low socio economic status and were from rural areas around Delhi. All but 2 patients were delivered at home by untrained personnel.

In Maru et al series[28] 64 cases of cerebral venous thrombosis (CVT) in pregnancy and puerperium were evaluated. CVT made up approximately half of the young strokes and 40% of strokes occurring in females. Majority of them were below 25 years of age, multipara women from rural areas who were delivered at home by untrained dais. While 79.6% cases occurred during first two weeks after delivery, the incidence of antepartum CVT was also higher at 7.8%. Majority of the patients had meningoencephalitis (70.32%) presentation. Others in the spectrum had either acute fulminant type (18.76%), neuropsychiatric (6.25%) or pseudotumour cerebri like

presentation (4.86%). Patients with neuropsychiatric and pseudotumour cerebri like presentation had a better prognosis while those with acute fulminant type had a poorer outcome. The patients with bilateral hemorrhagic infarcts and diffuse cerebral edema on CT scan had a poorer prognosis. A lower mortality rate (18.75%) could be achieved with early diagnosis and quick institution of planned therapy.

ETIOLOGY

Kendall et al[39] considers the condition as primary cause and as a result of local vessel damage during labour. This may be assisted by haematologic changes which occur during pregnancy and which render the pregnant woman susceptible to a hypercoagulable state. Clotting factors V, VII, VIII, IX, XII and fibrinogen levels increase whereas Antithrombin III, tissue plasminogen activity and protein levels decrease.

Platelets become hyperaggregable. The underlying reasons for these hematologic changes are unclear but they may be hormone related. Damage to vessel wall occurs as a result of increased intracranial venous pressure which takes place as result of increased

intra abdominal tension.

These hematological changes are however common to all pregnancies and do not explain the selective involvement of only a few.

Estanol and colleagues studied prothrombin time, partial thromboplastin time, thrombin time, platelet count, fibrinogen level, thrombin generation index, antithrombin III, protamine test in several puerperal women but found no difference between the CVT group and the control group.

The cause of predilection of cerebral veins and dural sinuses is also obscure. **Stasis** of blood due to reduction in venous tone during pregnancy, **damage** to the sinus endothelium due to straining and breath holding during labour and low grade **septicaemia** during puerperuim have all been implicated. Further studies are however required in this field.

According to C. Prakash and B.C.Bansal[12],

Anatomical factors which predispose to thrombosis are

1. Lack of pumping action of muscles in the intracranial sinuses which promotes stasis of blood in cerebral sinuses .
2. Trabeculations within the sinuses.

Physiological factors that predispose to thrombosis are

1. Changes in blood flow. Eg. Stasis or hyperviscosity of blood as in dehydration, CHF, polycythaemia.
2. Changes in coagulability of blood. Eg. Pregnancy and pueperium , APLA, protein C & S alteration, antithrombin III deficiency.

Martin has advocated an explanation of retrograde venous embolism from pelvic veins. It is suggested that a fragment of clot may be carried to superior sagittal sinus from pelvic veins by way of vertebral venous system, these veins being devoid of valves. This fragment may then act as a starting point for a clot which forms in the sinus. The upward propulsion of the fragment is facilitated by act of straining which raises intra abdominal pressure.

PATHOLOGY

Cortical vein thrombosis usually present as a cord like swelling with minimal or absent hemorrhagic infarction of the brain. In superior sagittal sinus thrombosis, sinus is distended and appears blue. Cortical veins are also swollen and may rupture at some places giving rise to infarction and intracerebral hemorrhage.

When venous flow is obstructed, a transudative cerebral edema develops, it may even lead to transtentorial herniation with notching of uncus of temporal lobe. The clinical symptoms and the pathological changes however, depend on the extent of venous thrombosis and the presence of involvement of anastomotic draining veins. When the latter are patent, a localised thrombosis is unlikely to produce permanent pathological changes.

However when the involvement is extensive, congestion occurs in grey and white matter and results in perivenous haemorrhages. Slowing of circulation is further aggravated by rapidly forming oedema. The capillaries are then damaged and the process of haemorrhagic necrosis perpetuates. The thrombus may extend into other veins and may undergo organisation. Later during

the course, recanalisation of the organised thrombus may result in reestablishment of venous flow.

Microscopy shows typical changes of hemorrhage, but specific feature appears to be " profuse leukocytic invasion" because of patent arteries allowing in flow of inflammatory cells.

CLINICAL PROFILE

The clinical spectrum of thrombosis of the cerebral venous system is very broad. Signs and symptoms vary according to the veins and venous sinuses involved.

Usually a young woman reports with headache, focal or multi focal seizures, fleeting neurological deficits and declining sensorium during the II or III week following full term normal delivery. Headache is caused by inflammation within the veins or sinuses or by obstruction to absorption of CSF through arachnoid villi which project into the sinuses. The headache varies in severity and location and resists simple analgesics. The headache markedly improves once anticoagulation is begun.

Classically, a seizure occurs at the height of headache which is then followed by paresis or aphasia or there is sometimes a silent

development of a Babinski sign or weakness. These parenchymal signs are because of haemorrhagic venous infarction of cerebral cortex sometimes causing significant mass effect. Recurrent convulsions and progressive deficits suggest spread of clot through the valveless network of cortical veins. Rapid propagation of clot does not allow drainage via collateral veins. Recurrent convulsions progressive deficits and declining level of consciousness are poor prognostic factors. Monoplegia brachial or crural, hemiparesis with leg more affected than arm, intact language despite hemiparesis are common symptoms but never dense and generally regressive.

Bilateral papilloedema with symptoms of raised intracranial pressure occurs in those with large sinus thrombosis blocking CSF absorption and in those with mass lesions due to cortical and deep vein infarcts.

Cortical deficits like agnosias, apraxias, cortical blindness and aphasias occur which is recognized only in the milder illness with good sensorium and in most of the patients clear up rapidly before documentation. Moderate hypotension often due to peripheral circulatory failure may also be seen. Deep vein thrombosis is not

frequent and is usually seen in the left leg. It may prove to be a warning sign of impending CVT. The onset could be early or late in puerperium.

The clinical picture and pathology of CVT as compared with eclampsia have many common features and yet obstetricians adopt entirely different lines of treatment when the seizures or stupor and coma occur in late pregnancy or early puerperium. Seizures occurring within 48-72 hours after delivery or in the anternatal period would favour eclampsia but seizures after 48-72 hours are unlikely to be due to eclampsia. Srinivasan studied 76 women with eclampsia and 10 with PIH**.** Plasma fibrinogen was above 400 mg% in 50 of them ESR was also elevated. EEG was possible in 20 patients and showed no abnormalities. The CSF studies in 15 patients was normal. Thus CVT and eclampsia are differentiated only by the time of occurrence and demonstration of focal signs.

In cerebral arterial occlusion, the patient is conscious, seizures and features of raised intracranial tension are rare. Mortality is insignificant but the residual disability is severe often with dense hemiparesis. In contrast, those with CVT are very ill with seizures,

focal signs, and impairment of consiousness with features of raised intracranial tension. Mortality is still high 20 - 30%. But in those who survive, improvement is rapid in a few weeks and the recovery usually complete. It is therefore possible to clinically diagnose cerebral arterial occlusion and confirm it by angiography.

Prognosis

According to C Prakash and BC Bansall[12], early clinical diagnosis, three dimensional MR flow imaging studies, and early institution of therapy specially heparin or thrombolytic therapy followed by oral anticoagulants have improved the prognosis of CVT. Srinivasan[37] observed that mortality has been reduced from 50.6% to 10% in the last three decades.

Factors adversely affecting prognosis are early appearance of the convulso-paralytic state, impairment of consciousness and presence of haemorrhagic infarcts demonstrated by CT or MRI. Usually recovery is either complete or associated with minimal neurological deficit because of recanalisation and dissolution of thrombosis. The risk of future recurrence appears to be very infrequent, rather unknown.

INVESTIGATIONS

Bleeding time, clotting time and prothrombin values are normal. Blood urea, sugar, creatinine and electrolyte values help to treat associated metabolic disorders. Plasma fibrinogen is raised well over the control values in all the patients. ESR is raised. Raised levels of beta lipoproteins, triglycerides, platelet adhesive index with fall in fibrinolytic activity have been stressed by Chopra and Bansal in 1978.

Significantly low haemoglobin and PCV values are seen in only 25% of cases.

Lumbar puncture is done in those with stable vital signs to exclude meningitis. Pressure is raised, proteins slightly elevated. Subarachnoid haemorrhage may be seen in some cases.

EEG may show only non-specific changes in many cases but shows lateralizing, slow and sharp waves in infarcts. Serial EEG helps to assess progress of lesions.

NEURO IMAGING

The basic problem in neuroimaging is the anatomic variation of the cerebral venous system. One patient may have the diagnosis

confirmed by one imaging procedure whereas another patient may require one of the three types of imaging-CT, MRI or angiography.

COMPUTED TOMOGRAPHY.

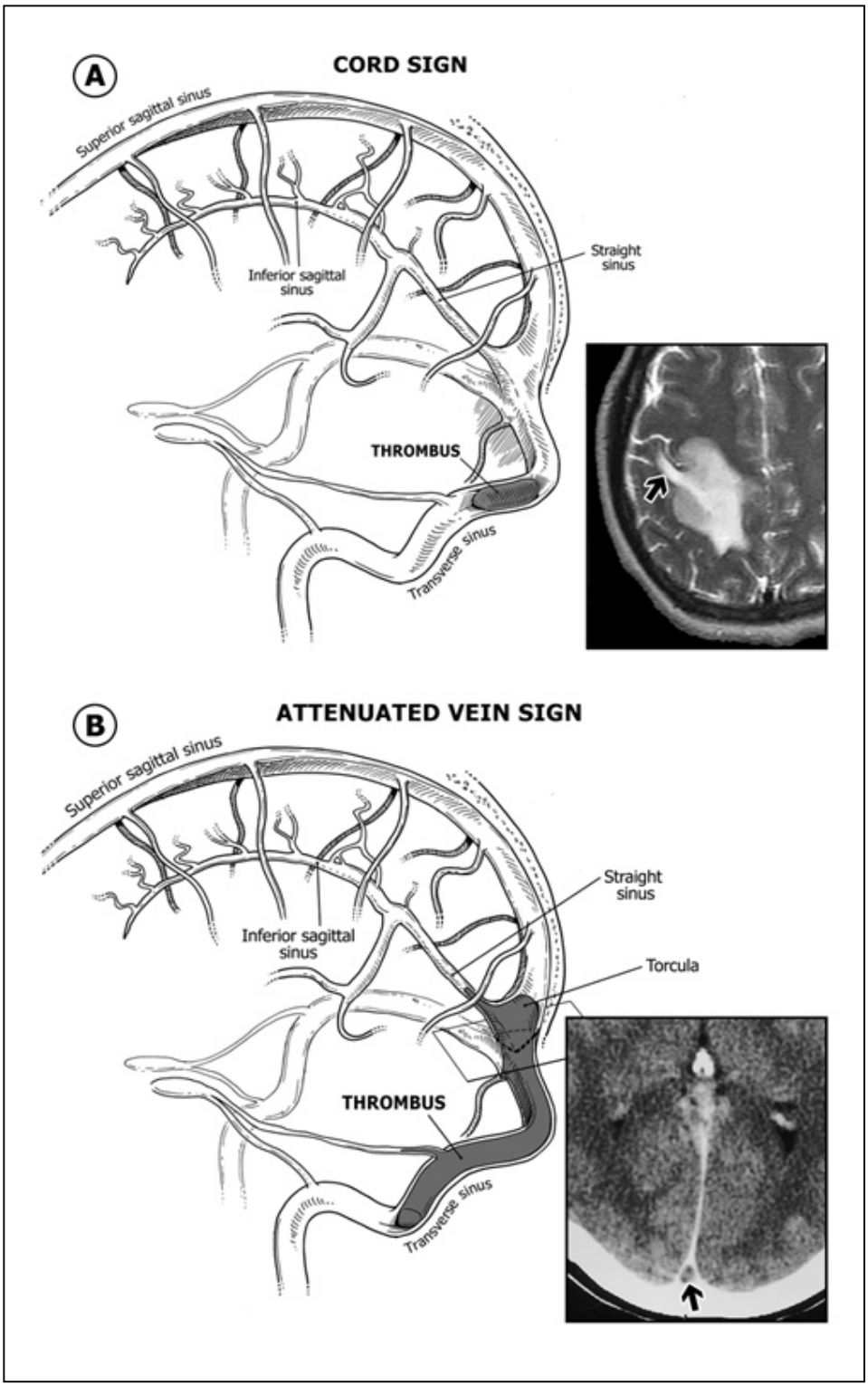
CT is usually the first neuroimaging study to be performed. CT is more accessible, less expensive and has much shorter scanning time than MRI. CT is also an excellent method of detecting mass lesions and acute bleeding including subarachnoid blood.

CT with and without contrast injection is recommended when CVT is suspected. More often CT reveals normal brain or non-specific changes. The small ventricles and hypodense regions suggest cerebral oedema.

Plain CT scan identifies both ischemic and hemorrhagic infarcts with 'Cord Sign' i.e. hyperdense thrombosed vein.

With contrast CT scan, the best direct evidence for CVT is the ‘empty delta sign' or ‘ empty triangle sign’ in the posterior third of superior sagittal sinus thombosis. The collateral vessels in the wall of the sinus enhance with contrast whereas the concealed blood within the sinus does not. This sign is seen only after the collaterals develop within the dura i.e. within 5 days following clot formation

SIGNS OF CVT IN CT BRAIN



and before three weeks. One must be careful because early bifurcation of superior sagittal sinus may cause a false empty delta sign. In severely anemic patients, CT may miss a hemorrhagic lesion due to low oxyhemoglobin.

Low attenuated areas indicate arterial and high attenuated areas surrounded by low density areas may suggest sinovenous occlusion.

S D Treadwell et al[2] reported that CT, which involves exposure to ionising radiation, may cause considerable anxiety in view of the potential hazard to the developing fetus. The potential effects of ionising radiation on the fetus include death, malformation, growth retardation, mental retardation and cancer induction. Risk is assessed on the basis of dose, and absorbed dose is measured in rad or gray, Gy (100 rad = 1 Gy). Risk estimates are derived from the survivors of high-radiation doses from atomic explosions in Hiroshima and Nagasaki, using linear extrapolation to low levels of radiation exposure. The maximum estimated fetal absorbed dose of ionising radiation is ~50 mrad for a head CT, 10 mrad for cerebral angiography, and 1.0 mrad for a chest radiograph.

To put this into context, natural background exposure at sea level is about 300 mrad per year. Estimation of the excess risk for cancer until the age of 15 is 1 in 17,000 per 100 mrad of fetal exposure. The estimated increase in lifetime risk of developing cancer after fetal exposure from a head CT is 0.07%. As approximately 1 in 4 people will develop cancer at some time in their life, this excess risk is extremely small. Up until 10 weeks of pregnancy, the threshold for detecting an increased risk of congenital malformation is above 5000 mrad. It should be emphasised that there is no direct evidence that fetal exposure to ionising radiation used in diagnostic imaging causes cancer or birth defects. Analysis suggests that pregnant women exposed to less than 5000 mrad have no additional risk to the fetus compared with women receiving background radiation alone.

RADIO NUCLIDE SCAN

Radio nuclide scans may show uptake in areas of venous infarction. Dynamic radionuclide brain scanning is non invasive and may provide useful information regarding the posterior sagittal and transverse sinuses. Resolution may be enhanced by photographic or computerised summation of counts during the sinus phase of isotope

angiogram. Resolution is not adequate for visualisation of cortical veins, the deep venous system or intra luminal filling defects in the patent sinuses. Because the absence of a lateral sinus may be a normal anatomical variant, non visualisation of a sinus must be taken in context.

Radiographic criteria for sinus occlusion are:

1. Non visualisation of the sinus on an isotope angiogram with a normal sinus groove for the sinus demonstrated by x-ray films of the skull.

2. Non visualization of the sinus on an isotope angiogram with normal or increased visualisation of the sinus on static images.

3. Abrupt termination of radio nuclide activity within the sinus, leaving a stump proximal to the point of termination "Stump sign".

The radionucleotide scan, because of its lack of specificity has entered the antique list.

MAGNETIC RESONANCE IMAGING

Angiography was-the 'gold standard' for the diagnosis of CVT till recently. Advances in MRI technology have led to the

THE DIAGNOSTIC INVESTIGATION – MRI SCAN

introduction of MR Angiography (MRA). It is now believed that MRA can replace cerebral angiography in cerebral sino-venous occlusive disease. MRI and MRA when used together have a high degree of sensitivity and specificity in the diagnosis of CVT. From day 1-5, MRI shows isointense signal in T1, hypointense in T2 and from day 5-15 , hypointense signals in T 1 and T2.

MRI is particularly useful in thromboses of midline sinuses and veins. The patient may need to be repositioned in order to study the lateral sinuses properly.

It also seems useful as a follow up instrument for documentation of thrombus regression, recanalisation and venous collateralization. Cost and logistic problems especially when patient is in intensive care unit are the main limiting factors.

In S D Treadwell et al study[2], MRI does not involve ionising radiation, and no adverse effects on the developing fetus have been documented, although any long term effects are yet to be determined. Recent guidance from the American College of Radiology suggests that pregnant patients can undergo MRI scans, provided that the potential risk/benefit ratio warrants the study, the

information cannot be acquired by other non-ionising means (eg. ultrasonography), and the information cannot wait until the patient is no longer pregnant. MR contrast agents cross the blood–placenta barrier easily, and no data exist to assess the rate of clearance of contrast agents from the amniotic fluid cycle, or the potentially toxic effects to the fetus. Guidelines suggest that administration of gadolinium-based MR contrast agent should be avoided unless overwhelming potential benefit to the patient or fetus that outweighs the theoretical risks can be demonstrated.

ANGIOGRAPHY

Carotid angiogram was routinely done before CT scan era to exclude tumors, aneurysms and AV malformation, to confirm carotid or middle cerebral artery block in a few cases.

The venous system can be well visualised by both conventional angiography and digital subtraction angiography.

The diagnosis of an isolated cortical vein thrombosis is difficult and is best accomplished when a hyperdense MR imaing signal compatible with a clot correlates with a non filling segment of vein on angiography. Poorly filling or non filling segments of the

anterior position of superior sagittal sinus can be seen in normal patients and does not indicate a thrombosis in that region. In addition to non filling veins and sinuses, there are often cortical collateral vessels often referred to as "cork screw veins".

Sometimes the diagnosis of CVT is accidentally established when angiography is done to investigate the possibility of subarachnoid haemorrhage or late postpartum hypertensive encephalopathy.

Angiography is neither sensitive nor very specific.

AUTOPSY:

Ellen found thrombosis of cortical veins, superior longitudinal sinus, and encephalomalacia as a common feature. Thrombosis of superior sagittal sinus, cortical veins and a large blood clot in the subarachniod space with necrosis of underlying brain was a feature of the 5 out of 7 fatal cases. King and Martin and Sheehan have also reported similar findings.

In the autopsy study of 98 cases of CVT at NIMHANS Bangalore over a period of ten years, puerperal CVT was the cause in 50% . Primary CVT due to dehydration, cardiac lesions, nephrotic

syndrome, congestive cardiac failure etc. accounted for 12%, and CVT secondary to pyogenic or TB meningitis was confirmed in 12% and the rest did not have any obvious cause.

RECURRENCE WITH SUBSEQUENT PREGNANCIES

The follow up of cases is inadequate and yet there is no method of predicting whether a woman who has once recovered from cerebral venous thrombosis will develop the same with subsequent pregnancies. Five patients from the Srinivasan series became pregnant again within 18 months and delivered normally without recurrence of thrombosis. Since most of the patients are multiparous and in view of the high mortlity rate, Srinivasan thinks that these patients should be advised sterlisation .

Van der stege et al[30] from Sophia Hospital, The Netherlands have described an uncomplicated pregnancy and puerperium in a 34 year old patient who had previous puerperal superior sagittal sinus thrombosis. Heparin was given 3 weeks antepartum and oral anticoagulants 3 months postpartum.

Six studies have addressed this issue with a total of 855 women under observation of whom 83 became pregnant of their

CVT. 88% of pregnancies ended in a normal birth, the remaining being prematurely terminated by voluntary or by spontaneous abortion. There were no incidence of recurrent CVT and only two cases of deep venous thrombosis. (Srinivasan 1983[37], Preter et al 1996[31], Lamy et al 2000, Ferro et al 2004[18] and 2007[2], Mehraein et al 2004[19])

TREATMENT

The overall objectives of therapy are :

1. To correct the basic pathological process that caused the problem.

2. To prevent extension of thrombus into patent vessels after treatment is instituted.

3. To control increased intracranial pressure until the involved vessels are recanalised or until collateral channels develop.

SUPPORTIVE THERAPY

Supportive care of unconsious patient is essential. Maintenance of airway, control of seizures, fluid and electrolyte balance, care of skin, bladder and bowel help in recovery.

SPECIFIC THERAPY

Anticonvulsant therapy and measures to reduce cerebral oedema are given. Mannitol and frusemide are used as diuretics. Mannitol helps to reduce intracranial hypertension and brain shift in severe cases. Since gastric bleeding could occur in cerebral or brain stem infarction or due to administered drugs, antacids can be given through Ryle's tube routinely. Aspirin 300 mg thrice daily may be used in some patients to reduce platelet adhesiveness.

STEROIDS

Corticosteroids may not reduce the hydrostatic edema and may inhibit fibrinolysis but they reduce CSF secretion and reduce the vasogenic oedema (Gevits 1974).

Corticosteroid should be used with caution because of their inhibition of fibrinolysis and increased risk of gastro intestinal haemorrhage in heparinised patients.

A case control study showed that steroids in the acute phase of CVT did not improve outcome (Canhao et al[3] 2008).

ANTICOAGULANTS

Anticoagulation has been a controversial treatment for 50 years. The argument against heparin is that it could promote bleeding into an already haemorrhagic infarction and there by precipitate death. In addition there is also risk of profuse uterine bleeding in puerperium. The argument in favour of heparin is that it stops propagation of clot and the risk of intracerebral bleed is well worth taking especially if CT does not show haemorrhagic infarction.

Treatment of sinus venous thrombosis with heparin is controversial. Many investigators have opposed its use because of the frequent occurence of intracranial haemorrhage in sinus venous thrombsis. In a randomised placebo controlled study in 20 patients (10 heparin, 10 placebo), after 3 months, 8 of the heparin treated patients had a complete clinical recovery and 2 had slight residual neurological deficits. In the placebo group, only 1 patient had a complete recovery, 6 patients had neurological deficits and 3 patients died. An additional retrospective study on the relation between Heparin treatment and intra cranial heamorrhage (ICH) in

SVT patients was based on 102 patients, 43 of whom had an ICH, 7 of these patients were treated with dose-adjusted, intravenous heparin after the ICH . Of these 27 patients, 4 died (mortality 15%) and 14 patients completely recovered.

Of the 13 patients that did not receive heparin after ICH, 9 died (mortality 69%) and only 3 patients completely recovered. So it was concluded that anticoagulation with dose adjusted intravenous heparin is an effective treatment in patients with SVT and that ICH is not a contraindication to heparin treatment.

Anticoagulation with heparin is with initial high doses and low doses later for 2 weeks (Bousser 1985[4], Srinivasan 1984[37], WHO Report 1989, Brucker et al 1998, Ferro et al 2007[2],Ganesh Dangal et al[1]). Heparin 10,000 units is given IV every 6 hours on day I and every 8 hours on II day and the frequency is reduced depending upon clotting time. Heparin can be discontinued in 2-3 weeks. Long term oral anticoagulants are needed only in those with deep vein thrombosis or suspected pulmonary embolism. With the use of heparin, mortality has been reduced from 50 - 60% in the earlier years to 10% at present.

Nagaraja et al 1995[35] included 51 women with puerperal CVT in India, initial treatment was with intravenous unfractionated heparin 5000 IU every 6th hourly. In heparin group all patients recovered, in control group 2 patients died and 1 had residual paresis at 6 months.

Heparin is indicated in those with increasing stupor or neurological deficit, high plasma fibrinogen, leg vein thrombosis or suspected pulmonary embolism. Heparin is life saving even in haemorrhagic infarcts because the cause of such infarct is venous thrombosis.

Heparin is contraindicated in those with gross liver disease, active peptic ulcer, haematoma and subarachnoid haemorrhage.

With anticoagulation, recanalisation of sinuses which have rigid walls can be followed by MR imaging.

In rare cases of heparin induced thrombocytopenia, warfarin can be started along with heparin and continued beyond 3 days when heparin is discontinued. Middel drop et al 2000 recommended warfarin for one year after the thrombotic episode. There after they need anticoagulant protection in high risk situations.

The effect of heparin which can usually be seen with in the first day of treatment, may be too slow to help the sub group of patients with rapidly progressing thrombosis which involves large parts of the cerebral venous system and which rapidly leads to diffuse brain swelling and multiple haemorrhages. In such cases intravenous thrombolytic therapy or local application of a thrombolytic agent may be indicated.

FIBRINOLYTIC AGENTS

Recently t PA and streptokinase have been used in a few cases with encouraging results with and without heparin. (Schutta 2001).

In five cases reported by Di Rocco et al. 2003, in whom urokinase and streptokinase were used recovery was complete in all patients, improvement was evident within one week and post treatment sinus patency was proven by angiography in four of the cases.

SURGICAL TREATMENT

Krayenbhul and Hunt have suggested that in some selected cases decompressive craniotomy or excision of necrosed brain might be useful. But with the limited experience of Srinivasan any such

procedure leads to fulminating systemic or cerebral venous thrombosis and the results are unfavourable.

In patients with impending herniation, hemicraniotomy can be life saving.(Stenfini et al 2001[26])

MATERIALS AND METHODS

This prospective study was under taken at Government Rajaji Hospital attached to Madurai Medical College, Madurai during the period of October 2009 – October 2010.

During this period our hospital has recorded13,164 obstetric admissions. 52 consecutive patients admitted with impairment of consciousness, seizures or focal neurological deficit were evaluated.

The inclusion criteria for the patients :

Following criteria were utilized in selecting patients for this study. Antenatal or postnatal patients admitted with complaints of

1. Headache
2. Impaired consciousness
3. Seizures
4. Focal neurological deficit

The exclusion criteria for the patients

The following patients were excluded from the study

1. Known epileptic patients

2. Post partum eclampsia (Occurrence of illness within 48 hours of delivery and absence of focal neurological deficits.)
3. CNS infection
4. Head injury.

History :

A detailed history regarding age, parity, time of occurrence, mode of delivery, various symptoms were taken from close relations and also from the patients if she was conscious or taken retrospectively from her. Any past history of hypertension, DVT, PIH, thrombophilias, APLAS were elicited.

Clinical Examination :

A thorough general examination and central nervous system examination was done.

Blood and urine were sent for all investigations related to cerebral venous thrombosis like renal function test, hematological examinations and APLA. Fundus examination and MRI with MRA/MRV were done for all patients.

The diagnosis was made by correlation of clinical features, the time of occurrence of illness and MRI with MRA / MRV scan findings.

Neurophysician opinion was obtained for all patients and were treated with anticonvulsants, antiedema measures and anticoagulants and patients were followed up till their discharge.

ANALYSIS OF THE STUDY

Incidence

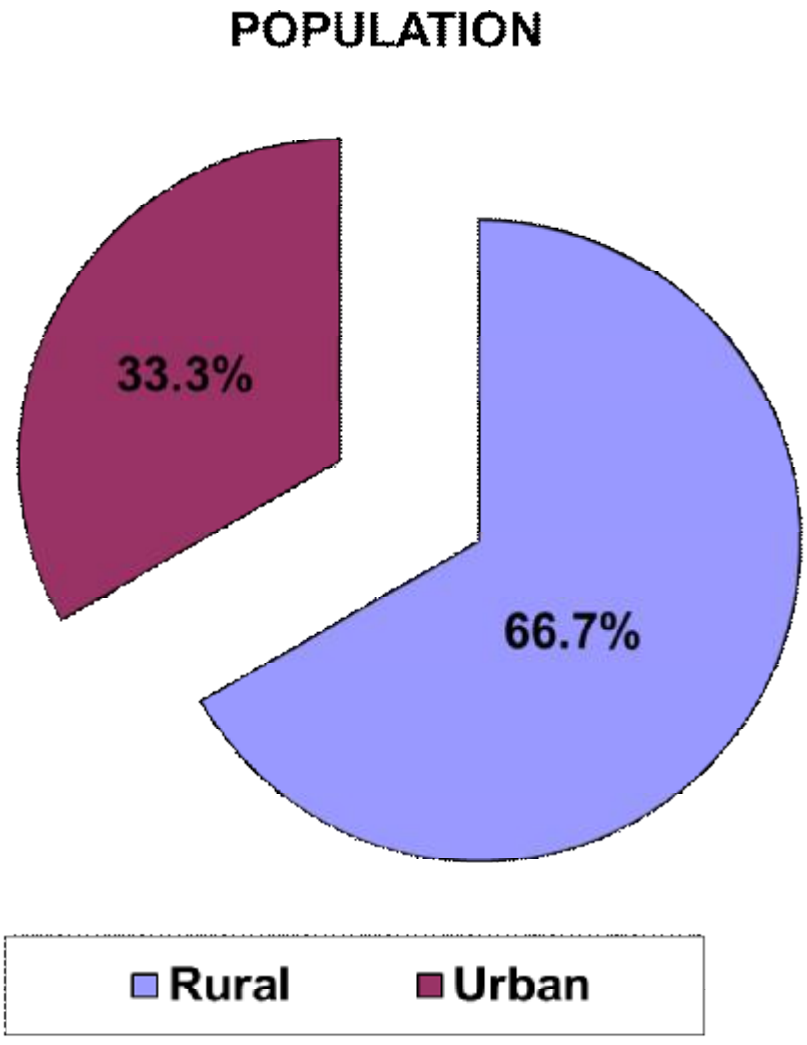
The incidence at Government Rajaji Hospital, Madurai was 3.9 per 1000 obstetric admissions compared to Western incidence of 1 in 3000 obstetric admissions.

A large series of 38 cases with angiographically proven CVT observed during 1975 to 1982 has been recently reviewed. (Bousser et al, 2000)[24] . The authors stressed that the incidence is falsely low as diagnosis of CVT is extremely difficult unless a high degree of clinical suspicion is confirmed with investigations.

Gates reported 66 cases collected from teaching hospitals in Australia. In 15 of 29 fatal cases who underwent autopsy, the diagnosis of CVT was not suspected before death.

A total of about six hundred cases have been reported so far from India and in majority the diagnosis was made on clinical grounds.

In a series of 80 cases at Nagpal, the diagnosis was confirmed at autopsy in 32 cases. The diagnosis of cerebral venous occlusion


POPULATION
33.3%
66.7%
Rural
Urban

was missed in 28 % out of 32 cases amongst the 8500 unselected medical autopsies in a period of 14 years.

POPULATION

Table1:

Population	***No.of cases***	***%***
Rural	36	66.7%
Urban	16	33.33%

36 out of the 52 patients (66.7%) belonged to rural population where people lived under unhygienic conditions and were predisposed to infections.

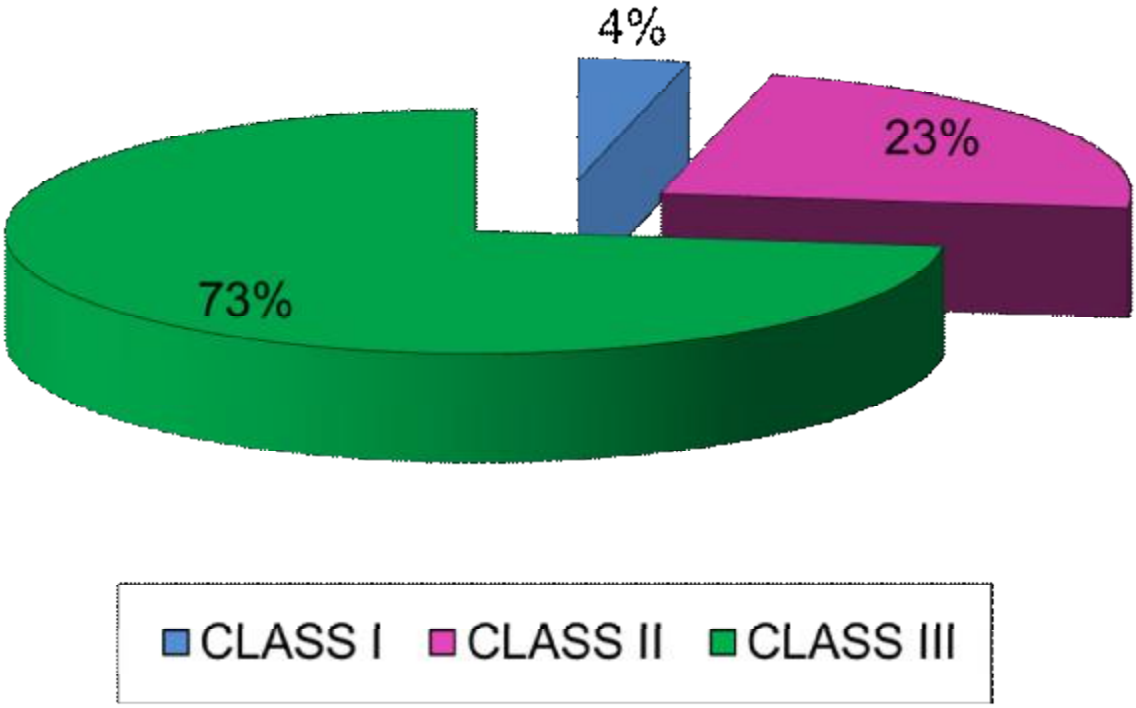
Moreover 70% of Indian population still live in village and this might be a reason for rural predominance. The occurrence in the rural population was twice of that in the urban population. 16 patients (33.33%) belonged to the urban population.

SOCIO ECONOMIC STATUS

Table 2 :

Status	*Class III*	*Class IV*	*Class V*
No. of cases	2	12	38
%	4	*23*	*73*

SOCIO ECONOMIC STATUS



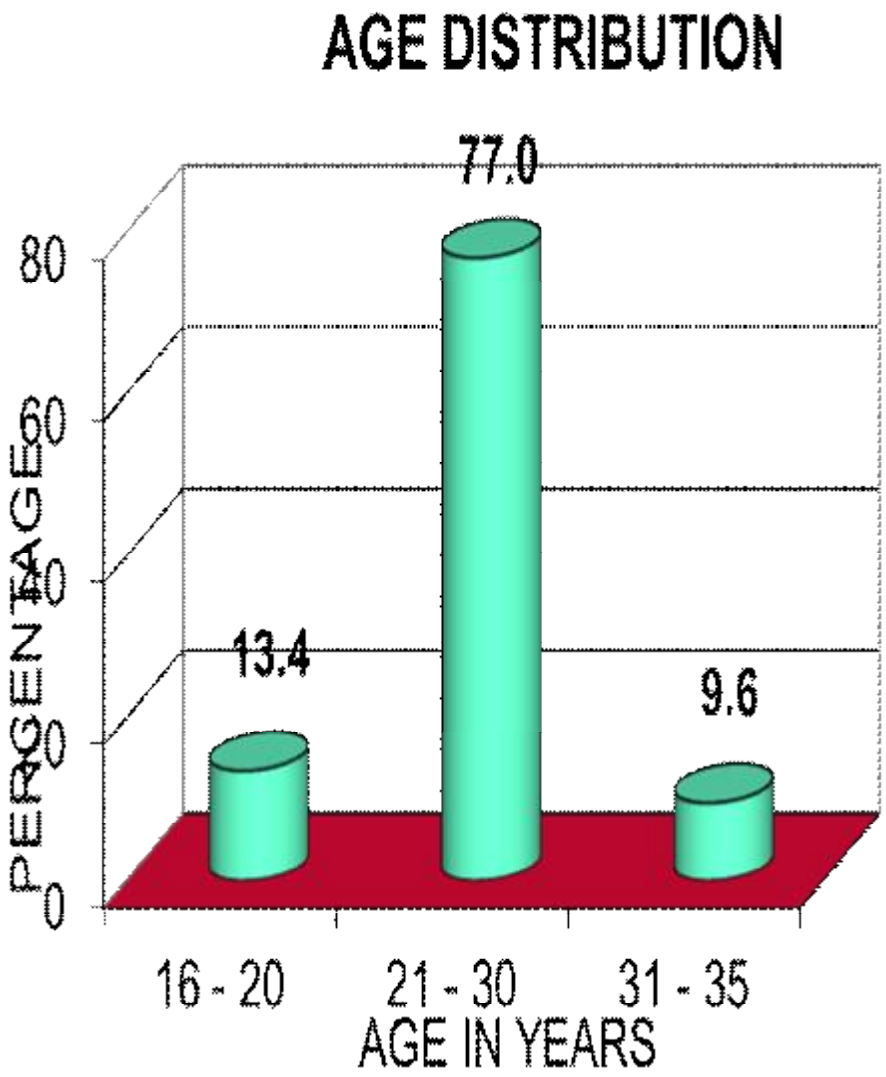
Anaemia and Protein deficiency are widely prevalent in the low socio economic group and these are important predisposing factors. Almost all the patients belonged to low socio-economic group with 38 out of 52 patients (73%) of them belonging to class V status.

AGE

Table 3:

Age in Years	**No.of cases**	%
16-20 years	7	13.4
21-30 years	40	77
31-35 years	5	9.6

The incidence varied from 18-35 years. The maximum age incidence, 40 out of 52 patients (77%) was in the III decade and correlates with the largest number of deliveries in this period. The least age at which CVT occurred was 18 years and no case was seen above the age of 35 years in this study.


AGE DISTRIBUTION
77.0
13.4
9.6
80
60
40
20
0
PERCENTAGE
16 - 20
21 - 30
31 - 35
AGE IN YEARS

PARITY

Table 4:

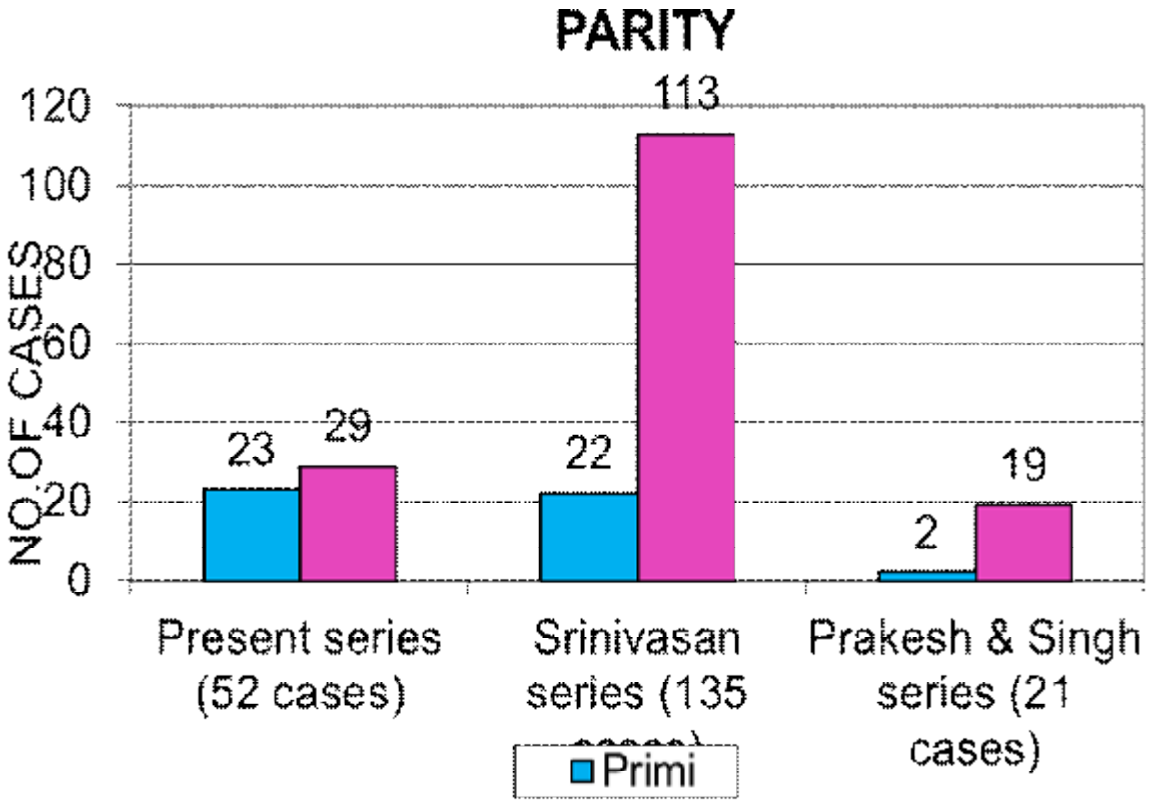
Parity	Present Series 52 cases	Srinivasan series 135 cases	Prakesh & Singh series 21 cases
Primi	23 (44.3%)	22	2
Multi	29 (55.7%)	113	19

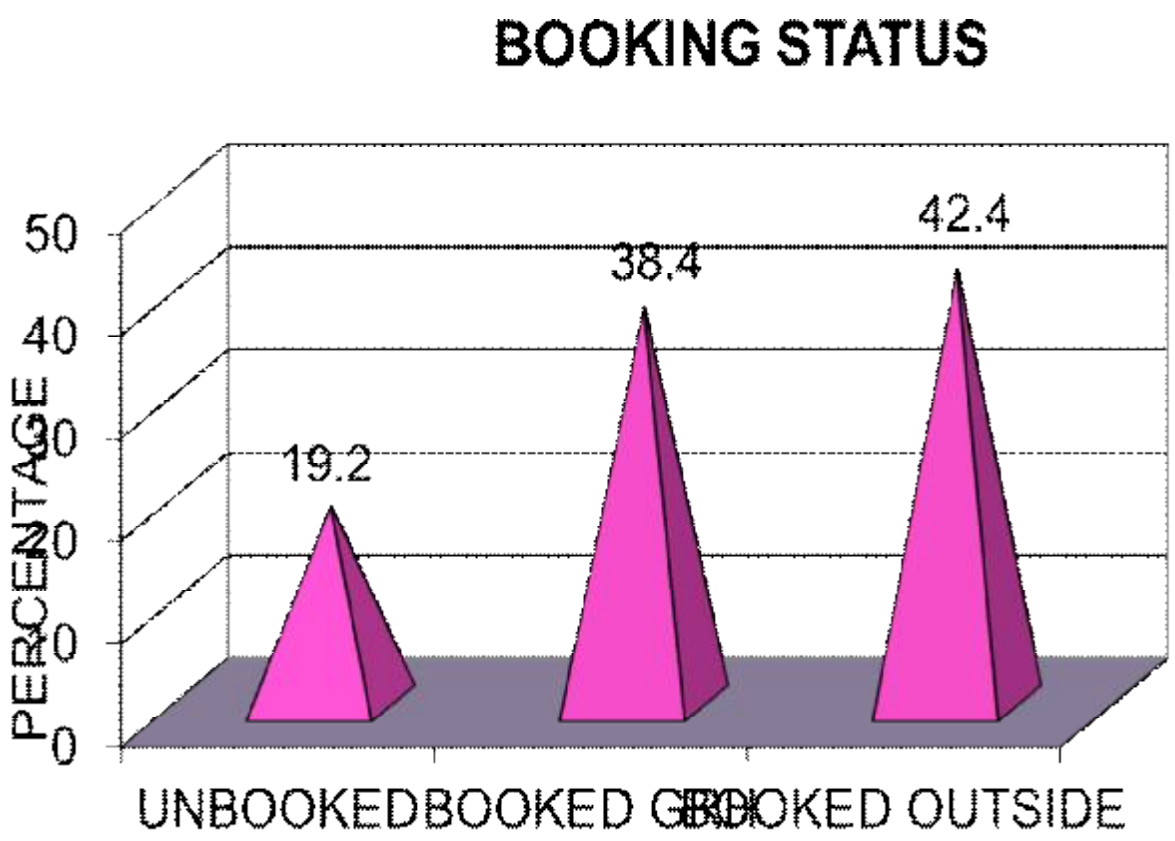
The disease occurred in 29 multiparous women out of 52 patients (55.7%). This observation is in correlation with the previous studies.

BOOKING STATUS

Table 5:

Booking Status	No.of cases	Percentage
Unbooked	10	19.2
Booked		
GRH	20	38.4
Outside	22	42.4


PARITY
120
100
80
60
40
20
0
NO.OF CASES
23
29
22
113
2
19
Present series (52 cases)
Srinivasan series (135
Prakesh & Singh series (21 cases)
Primi


BOOKING STATUS
50
40
30
20
10
0
PERCENTAGE
19.2
38.4
42.4
UNBOOKED
BOOKED OUTSIDE

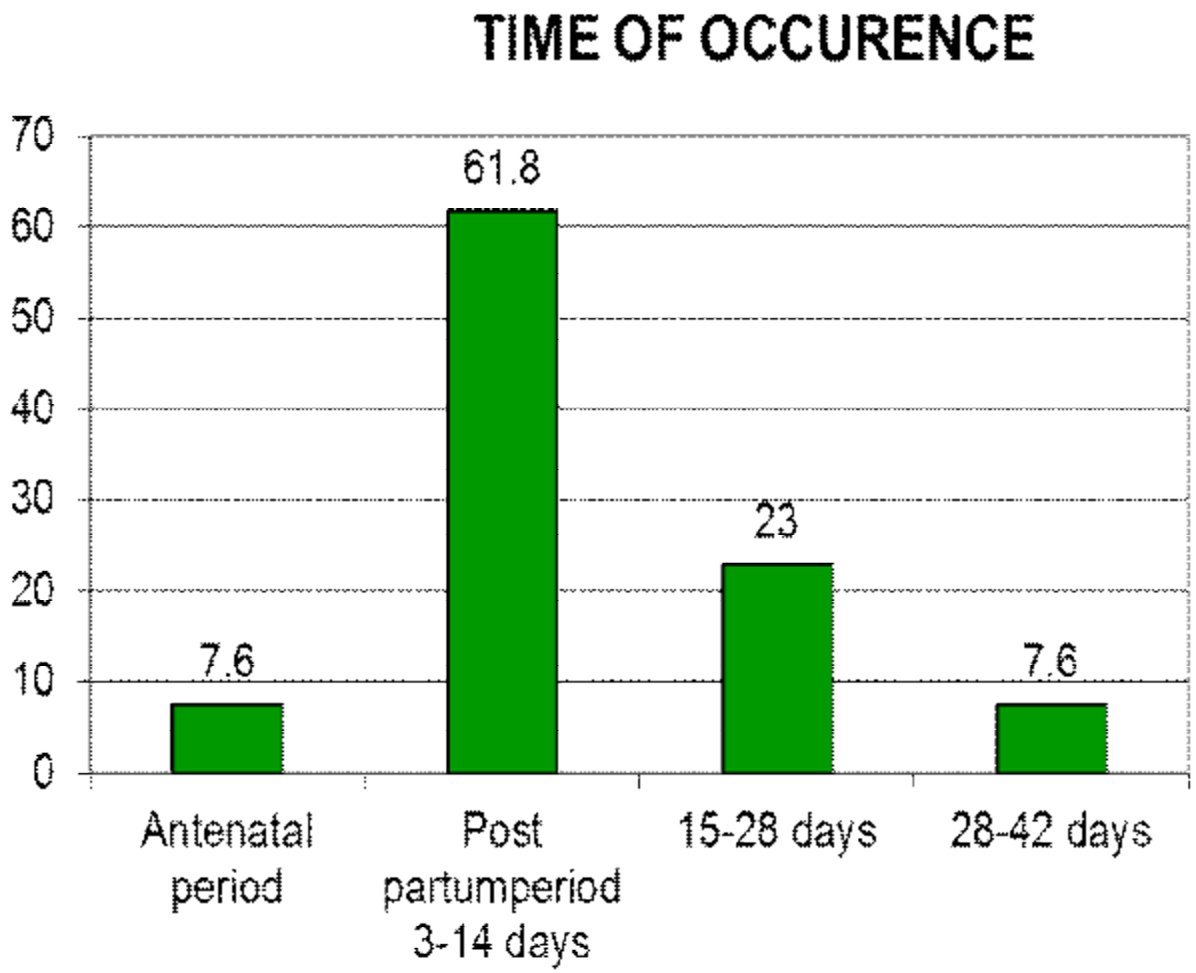
Majority of the cases, 30 out of 52 patients (61.6%) were unbooked and booked outside which implies that mothers with improper antenatal care are more affected.

TIME OF OCCURRENCE

Table 6:

Antenatal Period	***Postpartum Period***		
	3-14 days	15-28 days	28-42 days
4 7.6	32 61.8	12 23	4 7.6

The maximum incidence, 32 out of 52 cases (61.8%) were seen in the first two weeks of puerperium. 4 cases (7.6%) developed CVT in the antenatal period but majority of the cases 49 out of 52 cases (92.4%) were associated with puerperuim. Out of the 4 antenatal cases, one had normal vaginal delivery; one died within 40 minutes of admission at 7 months of gestation and the other two recovered completely and are continuing pregnancy with anticoagulants. The maximum coagulability of blood is seen during this period, and also Indian village women are not inclined to maintain good hydration


TIME OF OCCURENCE
70
60
50
40
30
20
10
0
61.8
23
7.6
7.6
Antenatal period
Post partumperiod 3-14 days
15-28 days
28-42 days

during this period.

PLACE OF DELIVERY

Table 7:

Place of Delivery	No.of cases	Percentage
Home delivery	4	8.4
Private hospital	6	12.5
Govt Rajaji Hospital	20	41.6
Other Govt. Hospital & PHC	18	37.5

The disease occurred in 20 patients (41.6%) who delivered at Government Rajaji Hosipital, Madurai and in 28 (58.4%) patients who delivered at other places. The lower incidence with institutional delivery may be due to strict aseptic precautions during delivery, avoidance of dehydration and maternal education.

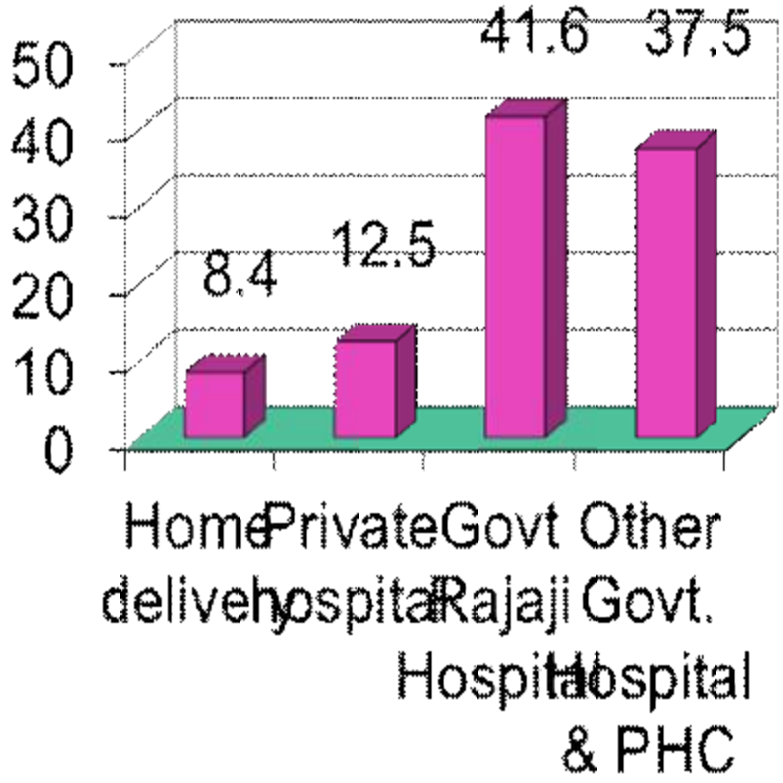
MODE OF DELIVERY

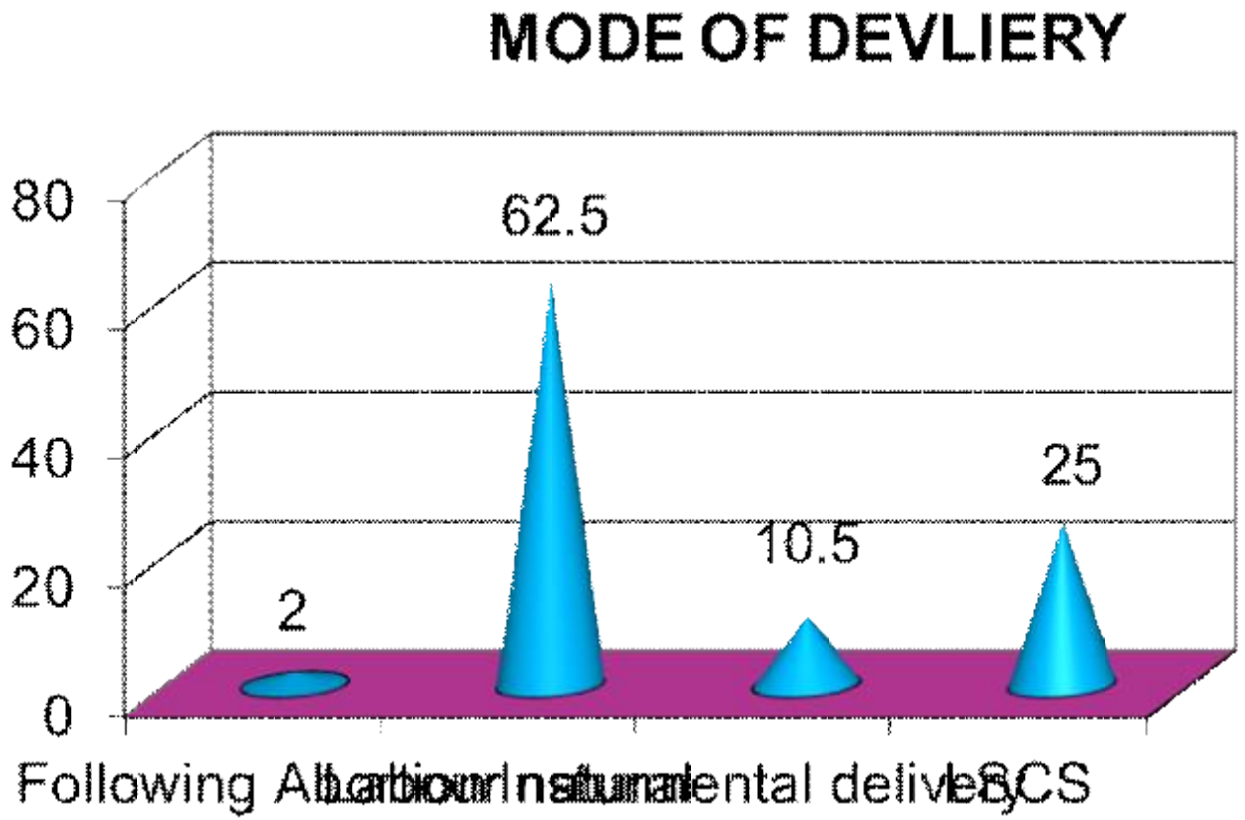
Table 8:

Mode of delivery	No.of cases	Percentage
Following Abortion	1	2
Labour natural	30	62.5
Instrumental delivery	5	10.5
LSCS	12	25

The maximum number of cases, 30 out of 48 (62.5%) occured

PLACE OF DELIVERY




MODE OF DEVLIERY
80
60
40
20
0
62.5
25
10.5
2
Following Abortion
Instrumental delivery
LSCS

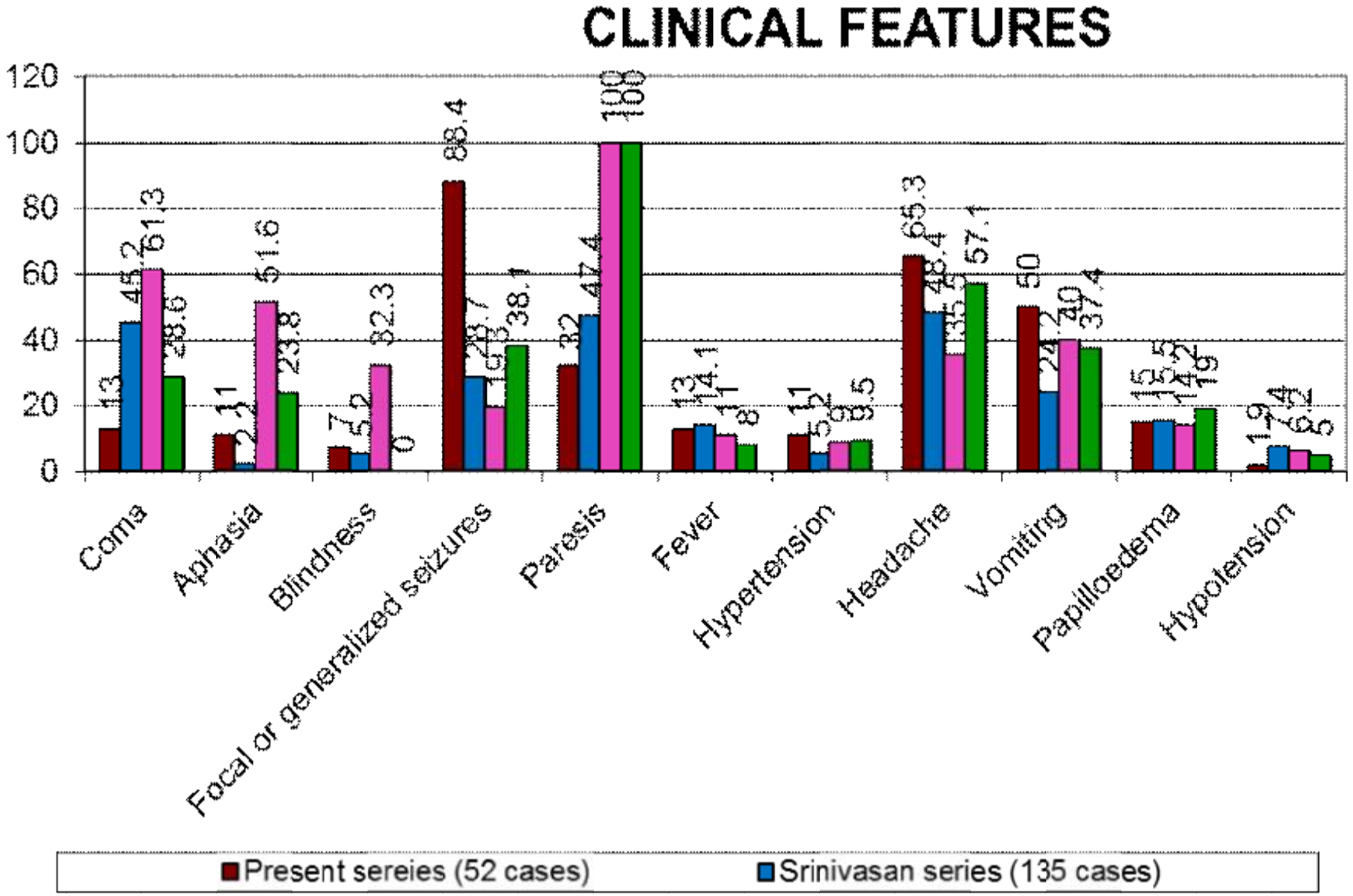
following full term normal delivery. The reason may be that these patients lack the close observation and care given to those after surgical intervention.

CLINICAL PROFILE

Table 9:

Clinical features	Number	Present Series 52 cases %	Srinivasan Series 135 cases %	Cross et al Series 31 cases %	Prakash and Singh 21 cases %
Coma	8	13	45.2	61.3	28.6
Aphasia	6	11	2.2	51.6	23.8
Blindness	4	7	5.2	32.3	5
Focal or generalized seizures	46	88.4	28.7	19.3	38.1
Paresis	17	32	47.4	100	100
Fever	7	13	14.1	11	8
Hypertension	6	11	5.2	9	9.5
Headache	34	65.3	48.4	35.5	57.1
Vomiting	26	50	24.2	40	37.4
Papilloedema	8	15	15.5	14.2	19
Hypotension	1	1.9	7.4	6.2	5

Classically the disease is observed in women presenting in a semistuporous condition with headache and focal or generalized convulsions followed by monoplegia or hemiplegia with or without


CLINICAL FEATURES
120
100
80
60
40
20
0
13
45.2
61.3
28.6
11
2
51.6
23.8
7
5.2
32.3
0
88.4
28.7
15.3
38.1
32
47.4
100
100
13
14.1
11
8
11
5.2
8.5
65.3
48.4
35
57.1
50
24.2
40
37.4
15.5
15.5
14.2
19
1.9
7.4
6.2
5
Coma
Aphasia
Blindness
Focal or generalized seizures
Paresis
Fever
Hypertension
Headache
Vomiting
Papilloedema
Hypotension
Present sereies (52 cases)
Srinivasan series (135 cases)

aphasia and occasionally bilateral involvement of the lower limbs.

RESULT

Table 10:

RESULT	No.of cases	Percentage
Complete recovery	38	73
Incomplete recovery	6	11.5
Dead	8	15.5

Inspite of the alarming clinical picture, recovery was rapid and remarkable. 38 out of the 52 patients (73%) recovered without any neurological disability. 6 out of 52 patients (11.5%) were discharged with minimal residual paresis. Total mortality was 15.5% (8 cases), most of the patients dying within two weeks of onset of illness.

Heparin was used in 49 patients (94.2%) and 3 patients (6.1%) in whom heparin was used died.

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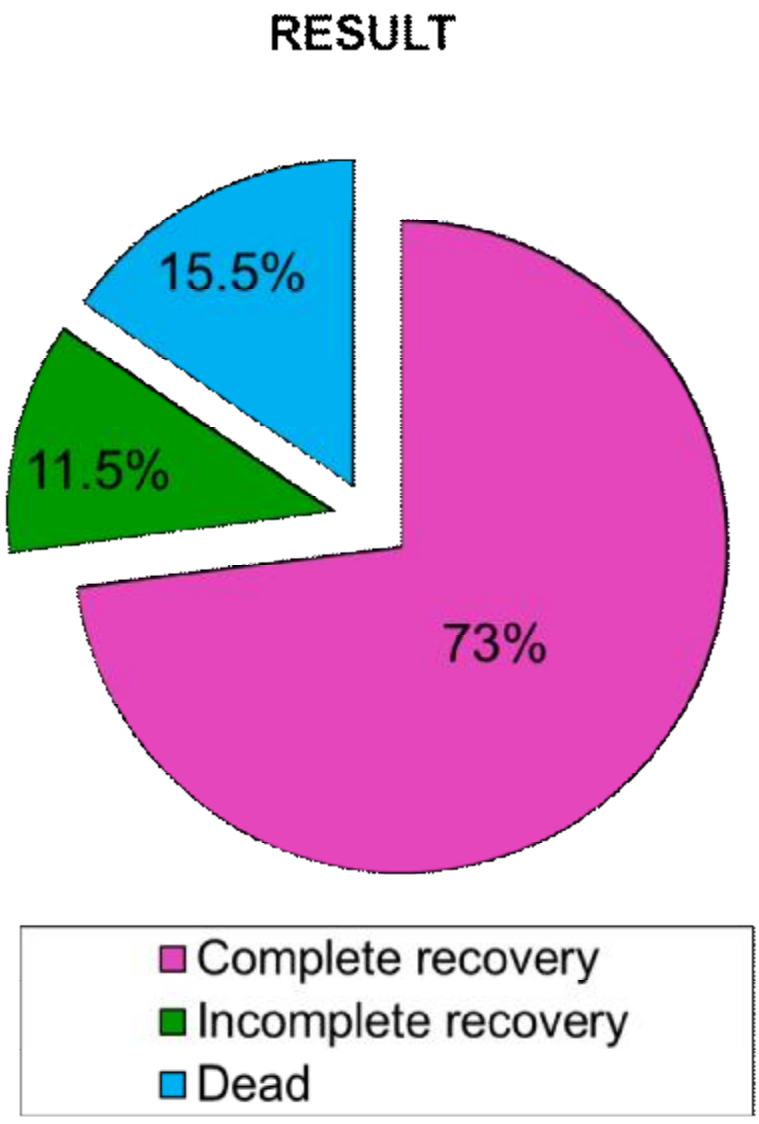

RESULT
15.5%
11.5%
73%
Complete recovery
Incomplete recovery
Dead

FREQUENCY OF VENOUS SITES

Table 11:

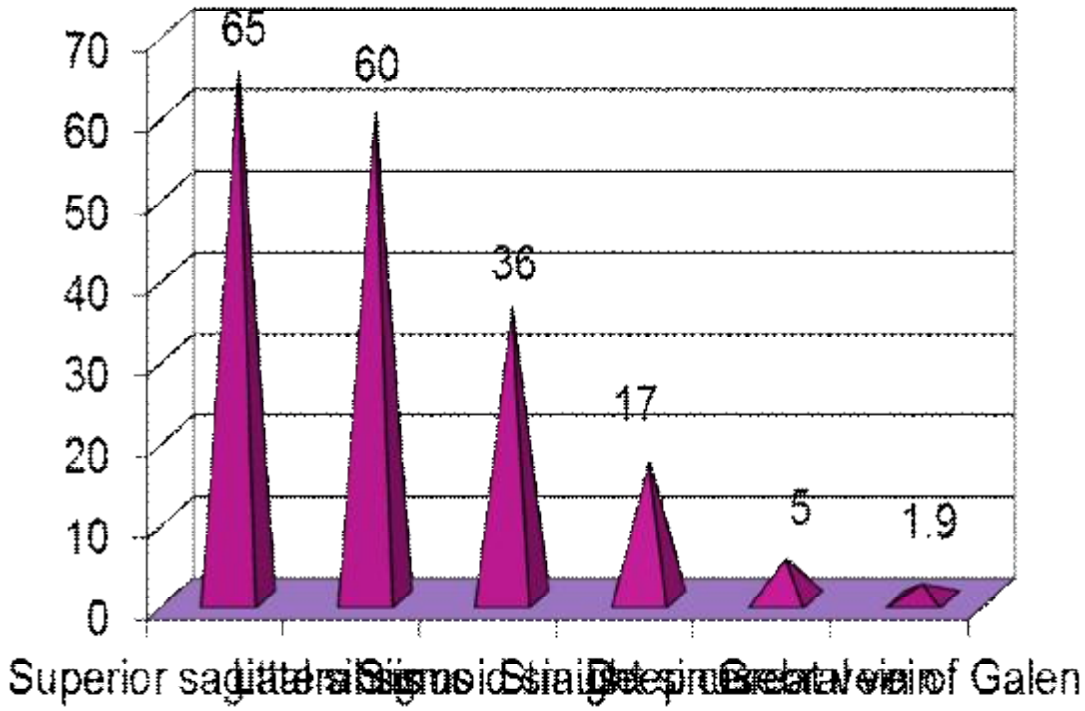
Frequency of venous sites	**No. of cases**	**%**
Superior sagittal sinus	34	65
Lateral sinus	32	60
Sigmoid sinus	19	36
Straight sinus	9	17
Deep cerebral vein	3	5
Great vein of Galen	1	1.9

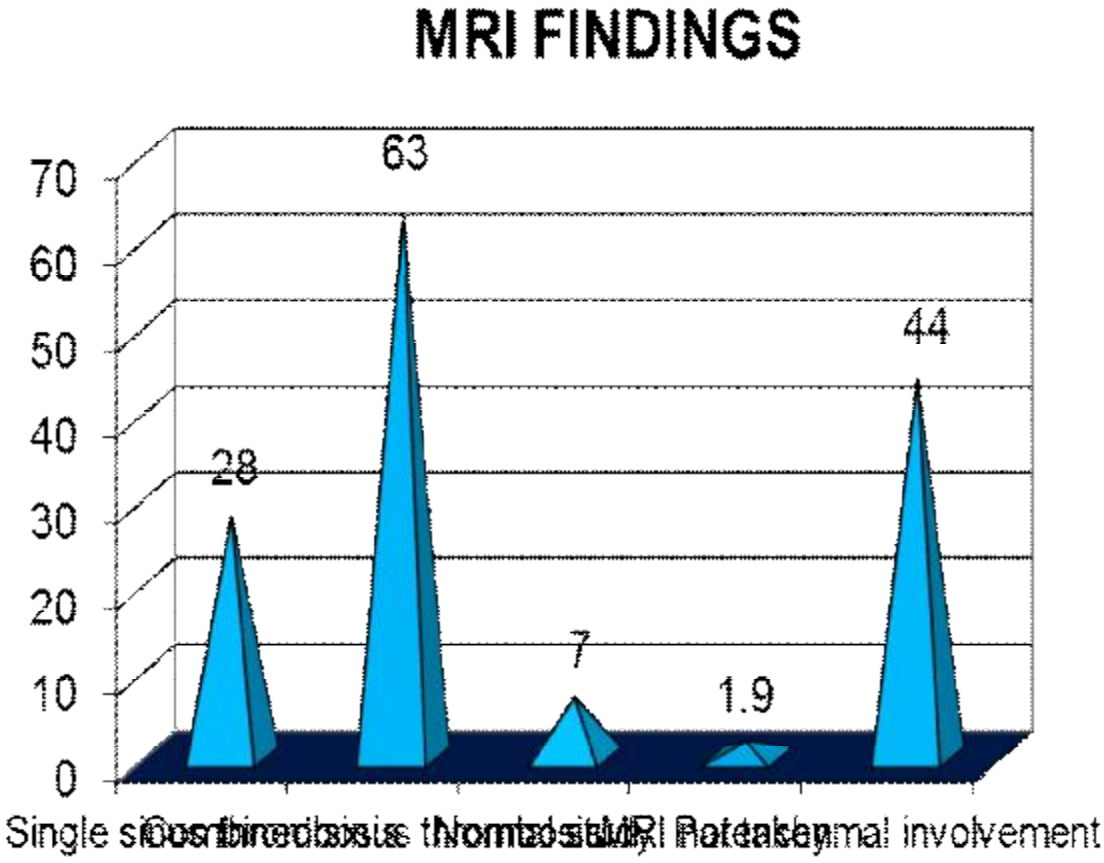
MRI FINDINGS

Table 12:

	No.of cases	Percentage
Single sinus thrombosis	14	28
Combined sinus thrombosis	33	63
Normal study	4	7
MRI not taken	1	1.9
Parenchymal involvement	23	44

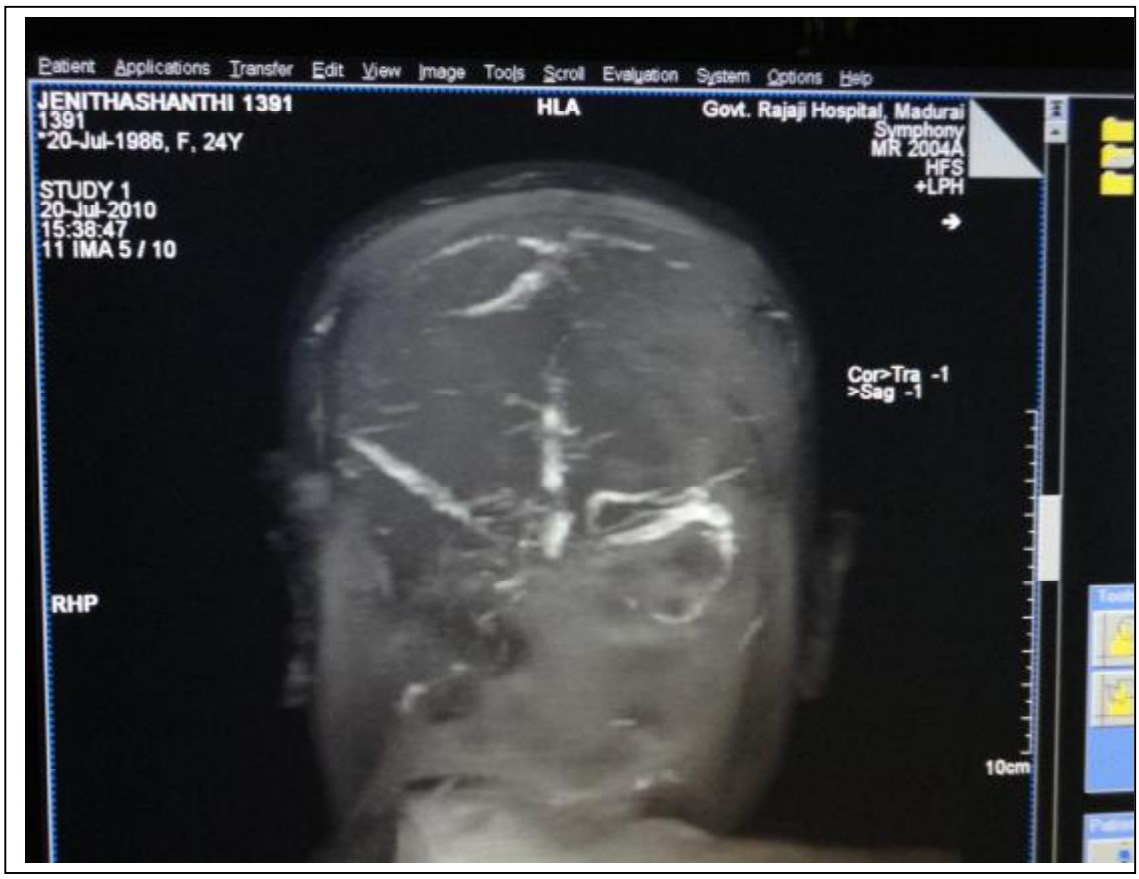
FREQUENCY OF VARIOUS SITES



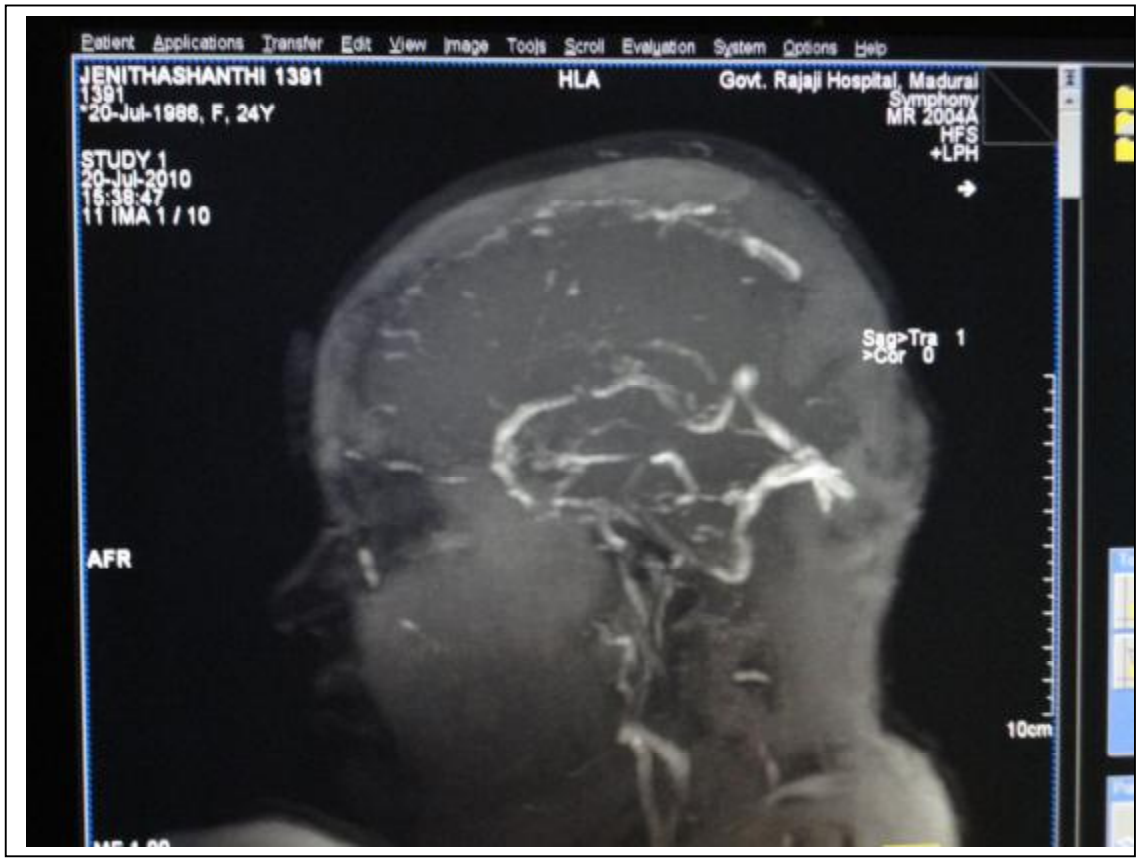

MRI FINDINGS
70
60
50
40
30
20
10
0
63
44
28
7
1.9
Single s
Combined
Normal MRI
Extra-axial involvement

Superior saggital sinus was the most common site, 34 out of 52 cases (65%) to be involved. Combined sinus thrombosis, 33 out of 52 cases (63%) was more common than single sinus involvement.

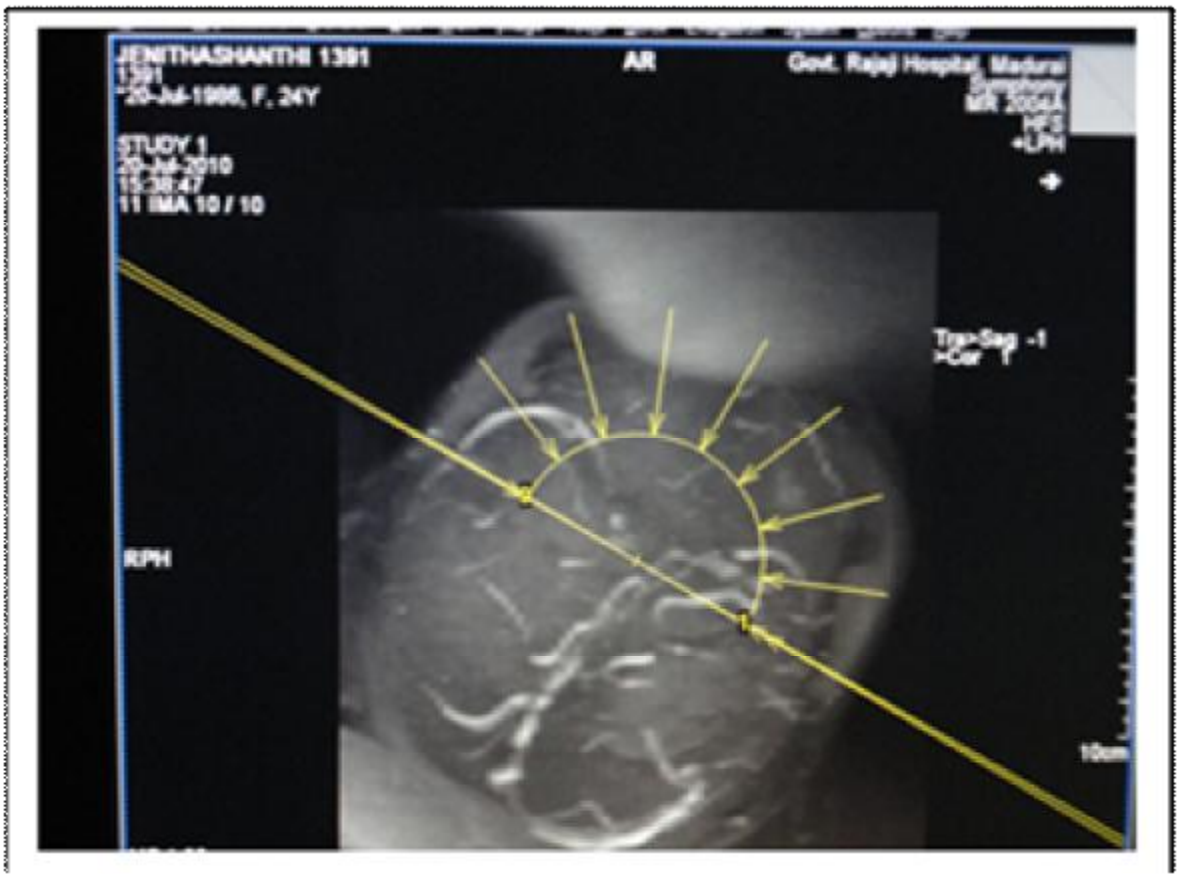
Name: Jenitha Shanthi,
Age : 24 years
Parity: Multi IP No. 54216



Sub acute venous thrombosis
of B/L transverse sinus and sigmoid sinus

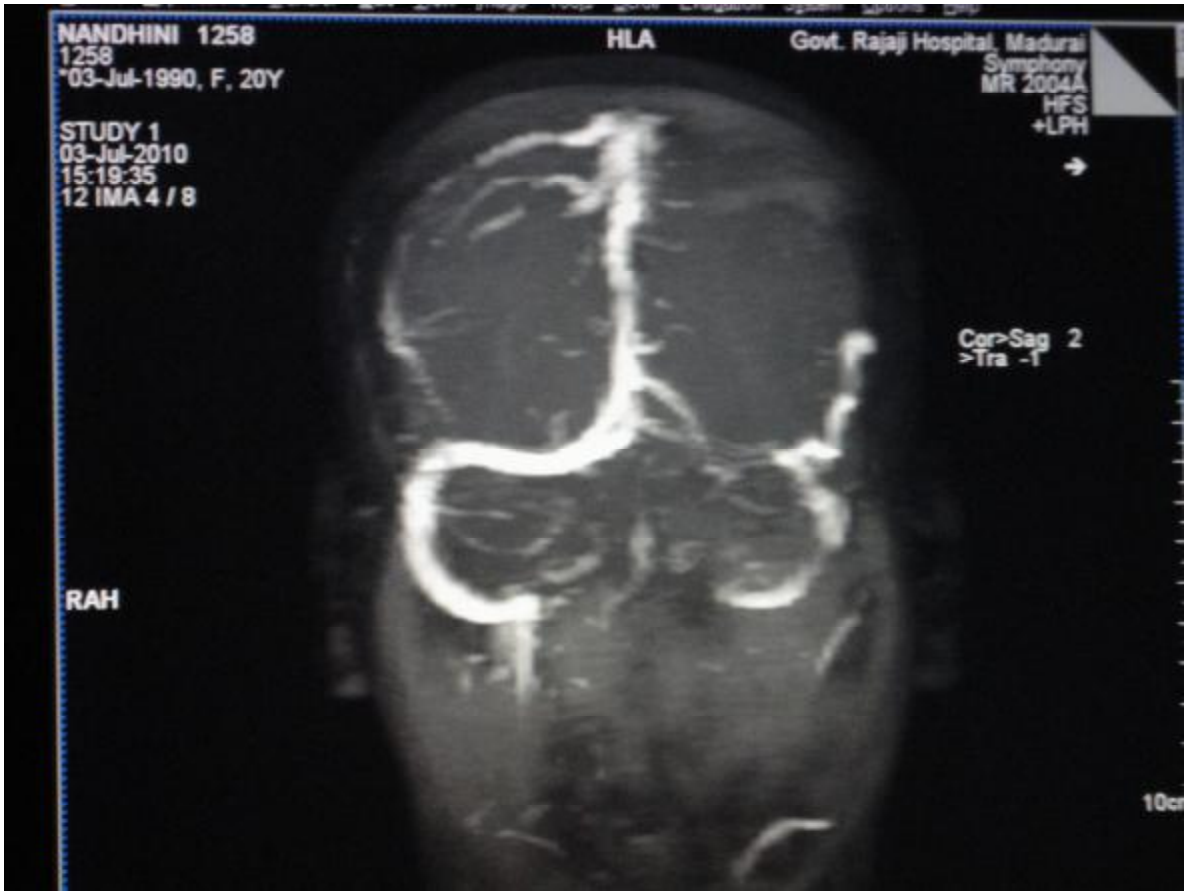


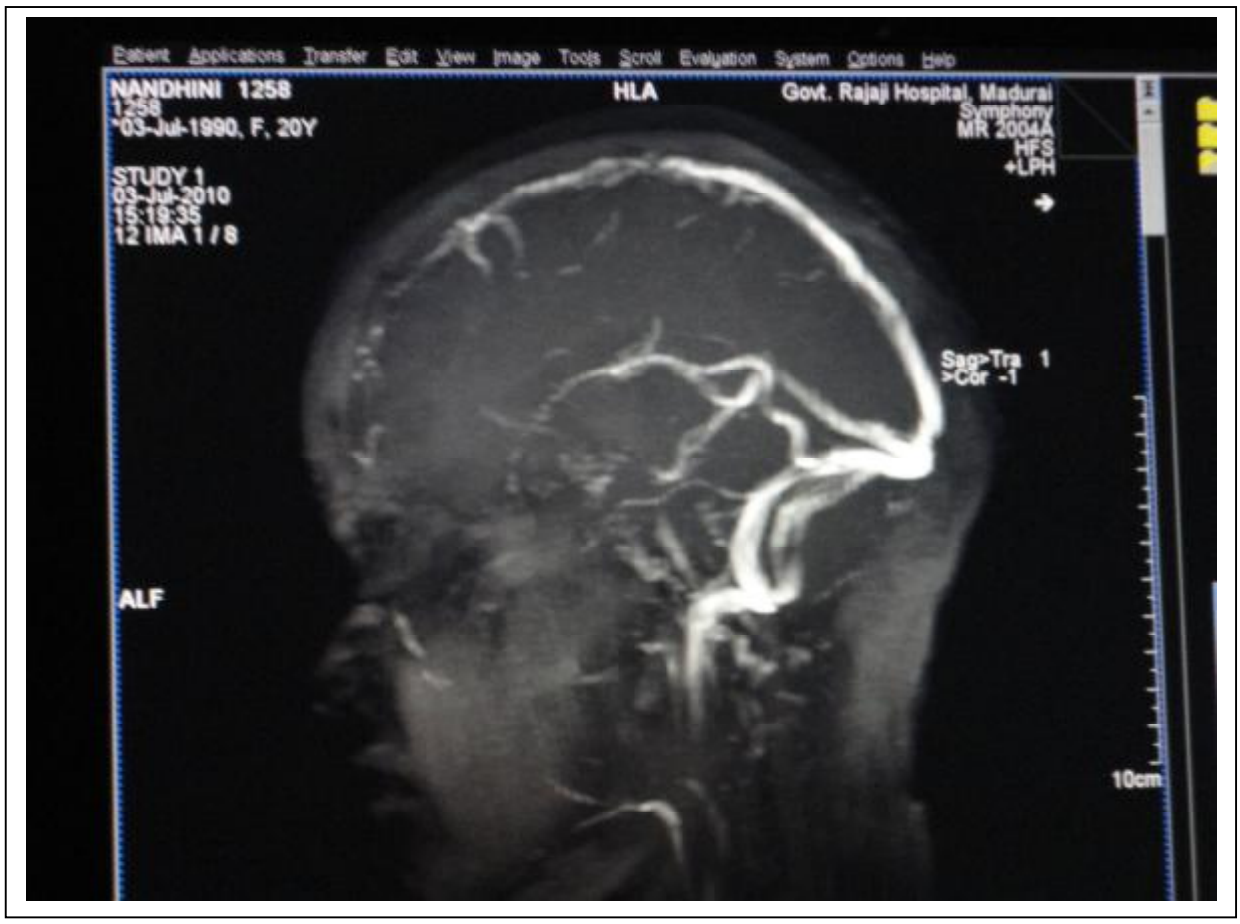
Extensive subacute venous thrombosis of superior sagittal sinus



Deep cortical vein, thalamostriate and medullary vein thrombosis

Name : Nandhini
Age : 20 years
Parity : Primi
IP No. : 49736





Non visualization of right transverse sinus,
right sigmoid sinus and straight sinus

DISCUSSION

During the 1 year of observation, there were 13,164 obstetric admissions and 52 of these were admitted with CVT. 48 patients had delivered and 4 were in the antenatal period. 35 out of the 48 patients had full term normal delivery and 12 delivered by LSCS. One patient had spontaneous abortion. This correlates with Srinivasan et al[37] and Maru A et al series[28].

In cerebral venous thrombosis, symptoms are caused by obstruction of the cortical veins or the superior longitudinal sinus. Impairment of CSF absorption causing raised intracranial tension or obstruction of the draining veins results in regional cerebral infarction with focal signs or seizures. Disorders of consciousness alone might be due to deep cerebral vein thrombosis. Extensive peripheral venous thrombosis, prolonged unconsciousness or status epilepticus are bad prognostic signs.

MODE OF PRESENTATION

Level of consciousness :

Derangement of consciousness was seen in 8 patients while 44 patients were fully conscious at the time of admission and remained so throughout the illness.

Headache:

34 out of 52 patients had headache at the time of admission.

Fits:

46 patients had convulsions, being generalized in 41 patients and focal in 5.

Paralysis:

17 patients presented with paralysis 8 had hemiplegia right or left, 9 patients had hemiparesis.

Aphasia was present in 6 patients, fever was present in 7 cases.

Neuropsychiatric illness:

2 patients presented with symptoms of psychosis.

Blood pressure:

Blood pressure above 140/90 mm of Hg was seen in 6 patients.

Fundus examination:

The fundus was normal in 44 patients but 8 patients had papilloedema.

All the above mentioned clinical profile correlated with the following studies: Srinivasan et al[37], Preter et al[31], Agostoni et al[20], Wasay et al, Maru A et al[28].

Investigations:

Anaemia with haemoglobin less than 8gm% which is a

predisposing factor for CVT was present in 16 patients. Correlates with Stolz E, Valdueza JM, Grebe M et al series[7.]

Leucocytosis with total count above 10,000 were present in 32 cases.

APLA was positive in 3 patients.

47 patients had thrombotic features on MRI and MRA/MRV scan. Out of which 23 had parenchymal brain lesion. 4 patients had normal study. Correlates with Idbaih A studies[8], Cakmak S et al [16], SD Treadwell[2].

Treatment:

49 patients with CVT were treated with heparin and only 3 people in whom heparin was used died. Use of heparin reduces the mortality. This correlates with Ganesh Dangal studies[1] , Srinivasan et al series[37], Bousser and Ferro et al[4].

Corticosteroids in the form of Dexamethasone were used in patients who were severely ill on admission. These patients were either unconscious at admission or became unconscious, soon after. These patients fared better on the whole. Recovery of consciousness was quicker and recovery from paralysis was also sooner. This

correlates with Canhao P et al study[3].

Prevention:

Over the years the incidence of CVT and mortality due to CVT has not declined much. Lack of antenatal and perinatal care is an important contributing factor for maternal death. Prevention could be done by

a) Better maternal health education

b) Improvement of social status of women

c) Early identification and reference of high risk pregnant women to tertiary care centre.

d) Comprehensive antenatal and perinatal care.

SUMMARY

1. The incidence of CVT in Government Rajaji Hospital, Madurai is 3.9 per 1000 obstetric admissions.
2. The rural population (66.7%) was affected more than the urban population(33.33%), the ratio of the affected rural to urban population being 2 : 1.
3. 73% of cases belonged to the low socio economic group.
4. The age incidence varied 18-35 years. The maximum age incidence (77%) was in the III decade.
5. The disease is more common in multiparous (55.7%) than primiparous(44.3%) women.
6. 61.5% of cases occured in the first two weeks of puerperium. This observation favours the fact that the disease is due to hypercoaguability of blood and the maximum coaguability of blood is seen during the first 10 postpartum days.
7. 62.5% of cases were seen after full term normal delivery.
8. The occurrence of the disease was less with institutional deliveries indicating that the disease is common where proper asepsis is not observed.

9. Fatal cases tend to be deeply unconscious from the onset, had extensive paralysis and numerous fits.
10. Those with parenchymal involvement, thrombophilia, APLAS had greater risk to be left with neurological sequelae.
11. MRI and MRA/MRV is used as first line diagnostic tool in cases of high clinical suspicion.
12. Heparin reduces the mortality and residual disability.
13. Corticosteroids are beneficial by ameliorating the symptomatology since they reduce the vasogenic oedema.
14. Mortality is as high as 15%.

CONCLUSION

1. Primary cerebral venous thrombosis occurs in puerperium in young women and is a major cause of stroke in young females.
2. Apart from hypercoagulable state, anaemia, lack of aseptic precautions during delivery and prolonged dehydration after delivery are the major causative factors.
3. MRI / MRA / MRV has revolutionized neuro diagnosis and has a firmly established place in the early diagnosis of CVT.
4. Early initiation of specific treatment with heparin helps to avoid mortality in most cases resulting in excellent recovery without any residual disability.

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PROFORMA

CRITICAL ANALYSIS OF CAUSES AND COURSE OF CEREBRAL VENOUS THROMBOSIS IN PREGNANCY AND PUERPERIUM

NAME : AGE: HOSPITAL NO. :

DOA : DOD: DIAGNOSIS :

Socioeconomic status : Class i / ii / iii / iv / v

Booking status : Booked / Unbooked

Obstetric Code : G P L A

Past History : HT / DM / PT / Heart Disease /Epilepsy / DVT / PIH / Thrombophilia / Diarrhoea / CVT / SLE / APLAS

Obstetric History : Mode of Delivery / Place of Delivery

Clinical Presentation :

Seizure : Focal / Multifocal / Generalized

Conscious Level : Semiconscious / Unconscious / Coma

Fever/ Vomiting / Headache / Giddiness

Blindness / Cranial Nerve Palsies

Paresis / Aphasia

Hypotension/ Hypertension

Time of occurrence

Antenatal Period	Postnatal period		
	3 – 14 days	**15 – 28 days**	**29 – 42 days**

General Examination:

Anaemia Oedema Hydration Febrile / Afebrile

PR BP CVS RS

Examination of Central Nervous System:

Higher Function :

Cranial Nerves :

Motor system :

Sensory System :

Investigations :

i) Hb : ii) Urine Albumin : iii) TC :

Sugar : DC :

Deposits : ESR :

iv) Blood Sugar : v) Sr. Creatinine : vi) BT :

Urea : Sr. Electrolytes : CT :

vii) APTT :

viii) Platelet count :

ix) Fundus :

x) APLA :

xi) MRA / MRV :

DIAGNOSIS :

Treatment :	**Drugs**	**Dose**	**Duration**
	Antibiotics		
	Mannitol		
	Dexamethazone		
	Phenytoin		
	Heparin		
	Acitrome		
	Physiotherapy		
Recovery :	Complete / Incomplete		
	Days		

Morbidity / Mortality :

ABBREVIATIONS

GB	:	GRH Booked
OB	:	Outside Booked
UB	:	Unbooked
P	:	Primi
M	:	Multi
AN	:	Antenatal period
PN	:	Postnatal period
LN	:	Labour Natural
LSCS	:	Lower Segment Caesarean Section
UD	:	Undelivered
C	:	Conscious
SC	:	Semiconscious
UC	:	Unconscious
HT	:	Hypertension
APLA	:	Anti Phospholipid Antibodies
PE	:	Papilledema