A DISSERTATION ON

"CLINICAL PROFILE OF POSTPARTUM CEREBRAL VENOUS THROMBOSIS AND ITS MANAGEMENT"

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I solemnly declare that this dissertation entitled **‘‘CLINICAL PROFILE OF POSTPARTUM CEREBRAL VENOUS THROMBOSIS AND ITS MANAGEMENT”** is a bonafide record of work done by me in the Department of Obstetrics and Gynecology at Government Mohan Kumara Mangalam Medical College Hospital under the guidance and supervision of Prof.Dr.S.S.Subha, M.D. D.G.O., Professor and Head of the Department, Department of Obstetrics and Gynecology, Government Mohan Kumaramangalam Medical College & Hospital, Salem, TamilNadu.

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PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled "**CLINICAL PROFILE OF POSTPARTUM CEREBRAL VENOUS THROMBOSIS AND ITS MANAGEMENT"** of the candidate **Dr.U.Kalaiyarasi** with registration Number **221716702** for the award of M.S., Degree in the branch of Obstetrics and Gynecology. I personally verified the urkund.com website for the purpose of plagiarism check. I found the uploaded thesis file contains from introduction to conclusion pages and result shows **16** percentage of plagiarism in the dissertation.

CONTENTS

INTRODUCTION

Cerebral Venous Thrombosis (CVT), the thrombosis of dural venous sinuses and cerebral veins was recognized since the early 19th century[1] but still remains a diagnostic and therapeutic challenge It often occurs in young individuals more common in females[2]. CVT will occur at any time from infancy to old age most reported cases were women with puerperium.[3] Onset of symptoms may be acute, subacute or chronic[4]. Cerebral venous infarction will be the most serious consequence of cerebral venous thrombosis, venous infarctions are often multifocal, bilateral affecting both grey matter and sub cortical white matter.

Patient of CVT will present with headache, seizure, papilloedema, altered sensorium and focal deficits due to thrombosis of intracranial veins and sinuses leading to haemorrhagic infarctions and increased intracranial tenstion[2]. This potentially life threatening event require early clinical suspician and prompt treatment, as the above disease will usually present in combinations ranging from syndrome of raised intracranial pressure to deep altered sensorium. CVT has distinct subgroup of cerebrovascular disease in India and it is a emerging cause of mortality in women of reproductive age group[3]. Most of the cases are seen in post partum period . CVT cases reviewed from Asian countries is showed of differences in risk factors, profile and outcome in these patients as compared to European studies. Largest cohort of CVT patients from Europe (n=624) reported that 50% of these cases were related to OCP pills, 6% were due to pregnancy and 14% were secondary to puerperium. Cantu from Mexico reported 59% cases due to Pregnancy puerperium. But , International Study on Cerebral Vein and Dural Sinus Thrombosis(ISCVT)[4] showed only 20%

cases are obstetric CVT, compared to reports from Mexico, India showing much higher frequency[5,6].

Cross et al[7] noted: “Usually recovery is rapid and complete if patient survives the acute episode”. Three fourth of cases of CVT in puerperium reported by him, survived with good recovery. It was considered as rare disease in preimaging era based on autopsy studies CVT is now on limelight due to its increased frequency despite awareness & better imaging modalities. Computerised tomography scan commonly shows haemorrhagic infarctions with or without “cord”, or “empty delta” sign[7]. MRI and MRV, when used in doubtful situations can clarify the diagnosis by showing thrombosed sinus of cortical veins[8,9]. Infact after the introduction of MRV, many of the patients earlier diagnosed as idiopathic raised ICT have been noted to have sinus thrombosis giving rise to syndrome of raised intracranial pressure without localization.

Pregnancy, puerperium, hormonal contraceptives and hormonal replacement therapy are the gender specific risk factors[10]. The puerperium is the known pre-existing risk factor because of its prothrombotic state[11]. CVT in puerperium is now recognised and feared complication as it accounts for 12% maternal mortality. Since, the direct cause of maternal mortality are brought down, there is growing interest on this non-obstetric cause. These factors prompted us to study the clinical profile and to evaluate the clinical, etiological, radiological characteristics, management and outcome of this disease among patients admitted with CVT in GMKMCH, a major tertiary care hopsital for the people in and around Salem district.

AIM AND OBJECTIVE OF THE STUDY

To observe the

- Demographic profile,
- Predisposing factors,
- Spectrum of clinical manifestation
- Neuroimaging,
- Management and
- Prognosis of Cerebro Vascular Thrombosis,

which occur during puerperium of the patients admitted in our hospital.

REVIEW OF LITERATURE

Cerebral venous thrombosis or sino-venous thrombosis, as the name implies is a condition which involves cerebral venous sinuses and veins together or independent of each other with thrombotic event of varied temporal evolution. The clinical presentation is varied ranging from syndrome of raised ICT without localization to seizures, focal deficits and deep altered sensorium[2,3.] Some patients may even present as behavioral disturbances as the predominant clinical manifestation, confusing the picture with post partum Psychosis. Strokes resulting from cerebral venous thrombosis usually affects young persons particularly women in reproductive age group, and carry a high mortality if not managed adequately[12]. The term Primary or Idiopathic Cerebral Venous thrombosis is used when no specific etiological factor is evident. 'Secondary' sino-venous thrombosis results from a variety of causes that include injury, infection, hematological disturbances, dehydration etc[13].

Historical Background

The wide spectrum of clinical features in cerebral venous thrombosis, the varied and changing etiological factors and the apparent "rarity" of the condition had made advances in knowledge slow and uneven. Periods of relative neglect has been interspersed with burst of enthusiastic discussion. The earliest reference to cerebral venous sinus thrombosis was that of Ribes in 1824[13]. He described in detail the clinical and post mortem findings of 45 yr old man who had thrombosis of superior sagittal and lateral sinuses, subdural effusion and metastatic carcinoma in the brain. The first case of puerperal venous thrombosis was reported by John Abercrombie[14] in 1828. His patient, a 24 year old woman, developed headache, delirium and initially

right sided than generalized seizures at the beginning of second week after delivery. Autopsy showed ischemic and haemorrhagic infarcts with thrombosed and sclerosed cortical veins. Quinke and Nonne identified the clinical syndrome of pseudo tumor cerebri (a term coined by latter) as a clinical counterpart to sinus thrombosis. Kalbag and Woolf, Sir Charles Symonds and others gave a precise clinical description of CVT after 1940. After introduction of CT scan and recently used MRI with MRV diagnosis of CVT has become simpler as these imaging modalities are quite sensitive in detecting CVT. Several large series with confirmation of diagnosis by angiograms, surgical exploration, and autopsy and recently with CT and MRI studies have been reported from Indian subcontinent[3,15].

Epidemiology

The true incidence of CVT is unknown. Ehlers and Courville found only 16 superior sagittal sinus thrombosis in a series of 12,500 autopsies (0.12%)[16]. Towbin found CVT in 9% of 182 consecutive autopsies[17]. However, with the more recent reports of large clinical series, the true incidence of CVT is probably considerably higher than that derived from autopsy series. People of all age groups may be affected by CVT but there is preponderance in young women because of specific causes like use of oral contraceptives, pregnancy and puerperium. Puerperal CVT has been reported to account for upto 15-20% of 'young stroke". It is the commonest cause of stroke in young women in India. 50% of strokes in Indian women are related to pregnancy and puerperium and 95.5% of these are due to CVT[18]. In Western countries, the incidence of CVT related to pregnancy and puerperium ranges from 1 in 1666-10,000 pregnancies. Risk factors like hyper homocysteinemia**,** OCP use, alcoholism, procoagulant state are increasingly recognized in addition to the

conventional risk factors like postpartum state.

It was estimated that more than 12% of maternal mortality occurs due to Cerebro Vascular Accidents. During puerperium women have a hypercoagulable state which explains the higher incidence of CVT in this period. According to the report published by Kittner *et al*, the average risk of being affected by haemorrhagic stroke during pregnancy is 2.5 times higher than non pregnant status and the risk further increases during puerperium. They also reported that the risk of ischemic stroke is 8-9 times greater in puerperium[19].The risk of CVT increases along with increasing maternal age particularly after 35 years.Conditions peculiar to pregnancy being significant risk factors were anaemia, postpartum haemorrhage, Hypertensive disorders of pregnancy, and postpartum infection[20].

A.INCIDENCE

Since CVT related to puerperium is not very common, it is difficult to arrive at an absolute risk above that of non pregnant group. In 1995, Sharshar T *et al* from France reported that rate of CVT due to ischaemia/infarction was 11 per100 000 deliveries with the puerperal period carrying the most risk. They also reported that the risk of haemorrhagic stroke was 9 per100 000 deliveries, again with the maximum occurrence in the puerperal period[21].Large population based studies are required to establish the incidence exactly, and reported incidence rates vary considerably.

Incidence of pregnancy-related stroke was calculated per 100 000 deliveries. It varied from 4.3–210 cases per 100 000 deliveries[22,23]. The recent population study in USA analyzed the inpatient data from, 1000 hospitals. Through this study 2850 cases of CVT in pregnancy and puerperium were analysed, showing This study

reported the incidence of CVT related to pregnancy as 34.2 per 100 000 deliveries and women have 3 fold risk of CVT during puerperiun than other women[24]. Wiebers and Whisnant reported 13-fold increased risk of stroke in pregnant females during the course of pregnancy and puerperium[25].

Most studies have reported CVT due to ischaemia and haemorrhage in more or less similar proportions[26,27].But Jaigobin and Silver have reported the incidence of ischaemic stroke as 18 / 100 000 deliveries and haemorrhagic stroke as 8/100000 deliveries. The prevalence of cerebral vein thrombosis is 4.5/ 1000 obstetric admissions in India. In our country CVT in the early puerperal period is 10 times higher than the Western countries

A study on pregnancy associated thrombo embolic disorders was conducted in Taiwan and showed that 73% of CVT occurred in the post partum period[28]. The data from USA showed that around 90% CVT related to pregnancy occurred either during delivery or puerperium[29] .Lanska *et al* also have published a similar report[30].The mortality due to CVT in pregnancy and puerperium has been estimated to be 10-13%[31,32].

B.RELEVENT ANATOMY OF CEREBRAL VENOUS SYSTEM:

Relevant Venous Anatomy [33]

The cerebral venous system contain of cerebral veins that drain into dural sinuses which then empty the blood into two internal jugular. Embryologically, the entire drainage system is subdivided into

- **an outer and superficial segment** which drains the scalp, underlying muscle, and tendons

- intermediate segment which drains the skull, diploe and dura matter

- cerebral segment, consisting of the veins that drain the brain. The cerebral segment may be further subdivided into a superficial cerebral group of veins and a deep cerebral group of veins. The superficial cerebral veins coalesce on the Pial surface draining out the blood from the outer 1 or 2 cm. of cortex and the underlying white matter. Venous blood in these vessels travels in a centrifugal direction and ultimately terminates in one of the dural sinuses. The deep cerebral veins serve to drain blood in a centripetal direction away from deep white matter, the basal ganglia, and the diencephalons. Tributaries draining many of the deep structure of the cerebrum join veins in the lateral angles of the ventricles and form a sub ependymal plexus. The veins of this plexus empty into the internal cerebral veins, which join the great cerebral vein of Galen.

Dural venous sinuses:

There are 2 groups of dural venous sinuses, superior and inferior.

- Superior group - collects major part of the blood of the brain.

  -includes superior sagittal sinus (SSS), inferior sagittal sinus, transverse sinus, straight sinus and sigmoid sinus.

- The inferior group - drains the basal and medial parts of under surface brain, the orbits and the sphenoparietal sinus collects at the cavernous sinus. Cavernous sinuses connect with the lateral sinuses via superior and inferior petrosal sinuses and with the pterygoid plexus.

The superior sagittal sinus courses over the superior border of falx cerebrijoined by the straight sinus to form the Tocular Herophili. From this confluence venous blood drains into the 2 lateral sinuses which drain into jugular bulbs and then into the Superior vena cava.

There are several anatomic variations of the dural sinuses. The most important are atresia of the anterior part of the superior sagittal sinus, duplication of the SSS mainly in its posterior part, asymmetry of the transverse sinus with dominance of right transverse sinus and aplasia or hypoplasia of the posteromedial segment of the left transverse sinus. Due to these variabilities, the angiographic diagnosis of cerebral vein or dural sinus thrombosis can be challenging.

Cerebral veins:

Cerebral veins include superficial venous system, deep venous system and posterior fossa veins.

- Superficial cerebral veins course over the surface of brain. They drain the major part of cerebral cortex. They have no valves and have several anastamosis.

-These superficial cortical veins drain into the superior sagittal and lateral sinus. These smaller veins show considerable variation in number and location except 3 large ones namely the large vein of Trolard, large vein of Labbe and the vein of Rosenthal.

- The deep cerebral veins drain the inferior frontal lobe, most of the deep white matter of cerebral hemisphere, the corpus callosum, basal ganglia and upper

brainstem. It includes the internal cerebral vein and basal veins of Rosenthal that join to form the Great vein of Galen which drains into the straight sinus. These venous systems are more constant in size and course.

- The posterior fossa has large variations in venous drainage patterns: those in the anterior drain into petrosal sinuses, those in the upper third drain into the vein of Galen system and those in the posterior drain into the lateral sinuses.

- Superior sagittal sinus and the lateral sinuses are the most commonly affected sinuses in CVT, followed by the straight sinus and the cavernous sinus .

- Thrombosis of Galenic system or isolated involvement of cortical veins is infrequent.

The following figures (I) clearly depicites the anatomy of cerebral venous system.

FIGURE I - VENOUS ANATOMY OF BRAIN

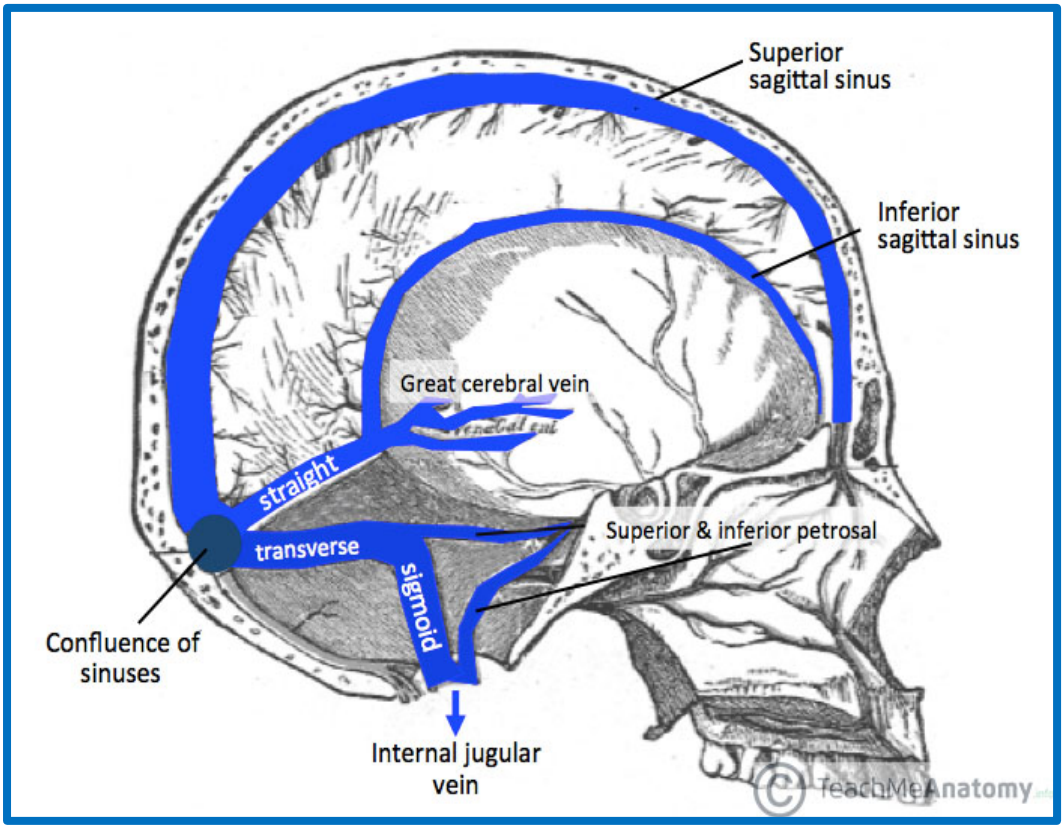
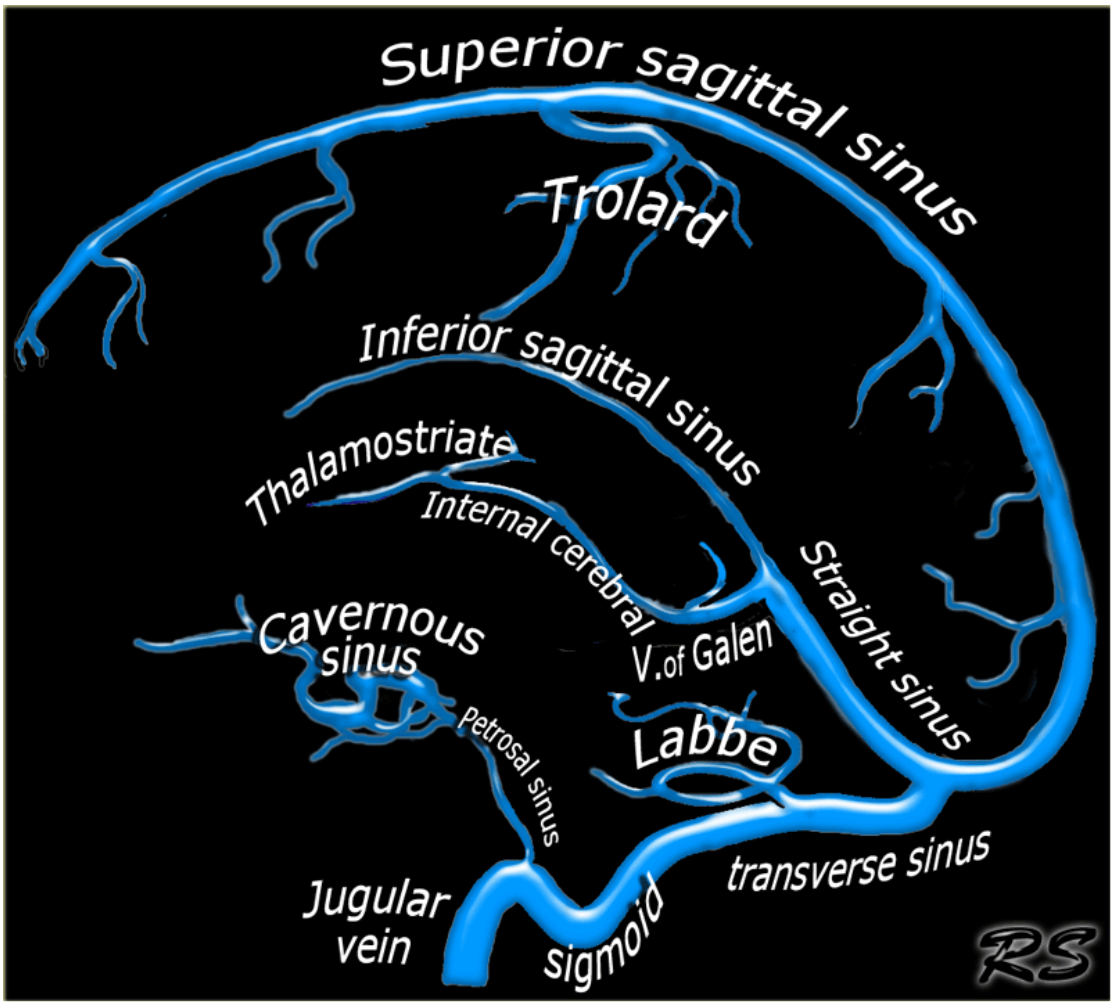


FIGURE II-INTRACRANIAL VENOUS ANATOMY OF DURAL SINUSES & CORTICAL VEINS



C.ETIOPATHOGENESIS

Various theories have been put forward regarding pathogenesis of CVT, particularly in relation to puerperal CVT.

Martin-Batson theory of embolic thrombosis[35,36]:

In understanding the pathogenesis of puerperal CVT studies of Batson (1940), and extension of the results of the study by Martin (1941) are milestones. Batson in experimental work on monkeys and human cadavers showed that pelvic veins have anastomosis with cerebral plexus of veins. Though he demonstrated anatomical connection in human cadavers, positive proof of functional conduct in live patients has not been shown. Based on this data Martin argued that thrombi from pelvis of parturient women under circumstances of raised intra-abdominal pressure could pass into vertebral plexus and then to intracranial sinuses. Once the thrombus reaches SSS, where blood flow is slow, it acts as a nidus for further thrombosis. The Martin-Batson theory does not explain the fact that SSS is most frequently involved although the vertebral plexus of veins communicate with the occipital and petrosal sinuses and not SSS. It also fails to explain the delayed onset of symptoms.

Kendall's theory of local damage:

Kendall (1948)[37] put forward his hypothesis of local damage in the sinus. He suggested that damage to the sinus endothelial lining occurs during the periods of breath holding and straining which may occur during the second stage of labor. The opposition to this hypothesis is that while most of the female population become pregnant and delivers and many of them repeatedly, less than 0.04% of them develop thrombosis of the SSS.

Theories of hyper coagulability:

Sinclari[38] (1902) was perhaps the first to demonstrate that plasma fibrinogen levels increase up to 150% of normal in the last trimester of pregnancy and attributed it to the increased tendency to thrombosis at this time of puerperium. In addition to humoral factors contributing to hyper coaguable state.Summarizing hyper coagulability in the form of increased levels of plasma fibrinogen, factor II,VII,VIII,X.XII and XIII, decreased fibrinolytic activity, increased platelet count and adhesiveness, and increased phospholipids occur in normal puerperium and may contribute to CVT[39,40]. Stasis and endothelial damage may also play a role. Thus one or more of the above factors may be responsible for puerperal CVT.

In addition to above mentioned abnormalities, other factors held responsible for hypercoaguable state are anemia and dehydration (Kalbag and Woolf[39]; Srinivasan and Natarajan[43]. Apart from anemia relatively recently recognized important factors causing hypercoaguble state leading to CVT are factor V leiden mutation, anti-cardiolipin and lupus anticoagulant antibody, increased resistance to protein C and decreased level of protein S, and antithrombin III deficiencies[40,41,42].

As stated earlier pregnancy associated hypercoagulability is the most important predisposing factor for CVT. After parturition, hypercoagulability worsens further due to volume depletion and trauma.

The risk of peripartum CVT increases with maternal hypertension, high maternal age, operative delivery, hyperemesis, dehydration and infections.In a report from India it has been observed that 112 cases out of 138 were under 30 years of age. Increased risk of CVT during postpartum period is attributed to bad obstetric practices

like delivery conducted by untrained persons and fluid restriction after delivery. Cantu et al has reported that cases of puerperal CVT show significantly higher incidence of anemia and raised ESR.

Obstetric CVT has an acute onset and better prognosis than thrombosis due to other causes. Peripartum CVT shows diversity of clinical features, mode of onset and neuroimaging signs due to which the diagnosis is often overlooked or delayed. The rate of death from peripartum CVT varies from 2% to10%. Studies have reported that mortality is significantly lower for peripartum CVT.When maternal deaths occur, they usually result secondary to intracerebral haemorrhage.

D.PATHOPHYSIOLOGY:

Two major mechanisms [43] occurring in pathophysiology of cerebral venous thrombosis are,

1. Thrombosis of either cerebral veins or dural sinus, resulting to cerebral lesions.

2. Thrombosis in dural sinus, leading to disturbance of CSF absorption and raised intracranial pressure.

- Thrombosis of cerebral veins or sinuses causes increased venular and capillary pressure. As local venous pressure continue to raise, reduced cerebral perfusion results in ischemic injury and cytoxic edema causing to disruption of blood brain barrier resulting to vasogenic edema and venous and capillary rupture culminates in parenchymal hemorrhage.

- Obstruction of cerebral sinuses causes decreased cerebrospinal fluid absorption, resulting increased intracranial pressure, which aggravates

venular and capillary hypertension and causing parenchymal haemorrhage, vasogenic and cytotoxic edema.After venous occlusion large areas of brain may be functionally and metabolically disturbed, but not irreversibly. Reversibility is very typical of venous lesions, reflected by a favourable clinical recovery and vanishing lesions on neuroimaging[44].

Occlusion of one of the larger venous sinuses is not likely to cause localized tissue damage unless there is involvement of cortical veins or the Galenic venous system since alternate drainage routes will suffice .Thrombosis in cerebral veins, with or without dural sinus thrombosis causes multiple venous infarcts[45] .

E.TYPES OF CEREBRAL VENOUS THROMBOSIS:

Intracranial venous thrombosis can be classified based on etiology as

1. Septic - involves cavernous sinus, infrequent nowadays

2. Aseptic - Dural venous thrombosis

- Deep venous thrombosis and

- Cortical or Superficial vein thrombosis

F.CAUSES OF CEREBRAL VENOUS THROMBOSIS

❖ **Hypercoagulability**

The following physiological changes taking place in pregnancy leads to a hypercoagulable state.

- Reduced level of von Willebrand factor
- Increased level of fibrinogen

- Increased resistance to the action Protein C
- Reduced level of protein S
- Plasminogen activator inhibitors (PAI) 1 and 2 levels raised
- Hyperprolactinaemia causing platelet aggregation

❖ **Venous stasis**

Stasis of blood in the venous system is mainly due to the following factors.

- Pelvic vessels are being compressed by the enlarged uterus
- Restricted mobility,mostly bed ridden during pregnancy and puerperium

❖ **Endothelial injury**

- Vascular injury occuring in parturition
- Virchow triad which constitutes Hypercoagulability, Venous stasis, Endothelial injury predisposing to venous thromboembolism[46.]
- Extensive anatomical, physiological and biochemical changes takes place throughout pregnancy which involving all important organs.
- During puerperium coagulation and fibrinolytic systems are subjected to major changes leading to prothrombotic state. It is the physiological preparation for pregnancy. These alterations are responsible for the pathogenesis of complications that occur during puerperium such as venous thromboembolism[29].
- Apart from this, there is also progressive resistance to the action of Activated protein C, which is an anti coagulant.
- Protein S level starts decreasing around 10-11 wks and the decrease progresses

throughout pregnancy[47].

- Concentrations of PAI 1 and 2 - increased during pregnancy which will interfere with Fibrinolysis[29].

- The physiological increase of prolactin concerntration during pregnancy continues till puerperium,which occurs a preparation for delivery and breast feeding. This physiological hyperprolactinaemia can cause platelet aggregation which is mediated through ADP stimulation[48.]

- The anatomical alterations in pregnancy like compression of great vessels by the enlarging gravid uterus lead to venous stasis and increase the risk of thrombus formation. Doppler analysis of the venous system during pregnancy has shown progressive decrease in flow velocity. The flow velocity in the femoral vein at term is one third of that recorded during early months[49].

❖ **MISCELLANEOUS**

Other etiological factors related to pregnancy associated stroke are,

1. .Arterial dissection, as a consequence of straining during second stage of labour

2. Disseminated intravascular coagulation due to obstetric complications causing intracerebral haemorrhage.

3. Stroke related to the use of anaesthetic drugs in pregnancy**.**Because there is no pressure gradient, ventricular dilatation and hydrocephalus rarely occurs.

G.RISKFACTORS

Risk factors that have been recognized to cause CVT in puerperium are the following[50]

- Increased maternal age
- Asian race
- Hypertensive disorders of pregnancy
- Heart disease complicating pregnancy
- Smoking
- Diabetes Mellitus
- Systemic lupus erythematous & other connective tissue disorders
- Haemoglobinopathies
- Vascular headache
- Substance abuse especially cocaine
- Caesarean section & Instrumental delivery
- Fluid & electrolyte disorders especially dehydration
- Acquired & Inherited thrombophilias
- Multiple pregnancy
- Multiparity

- Postpartum infections

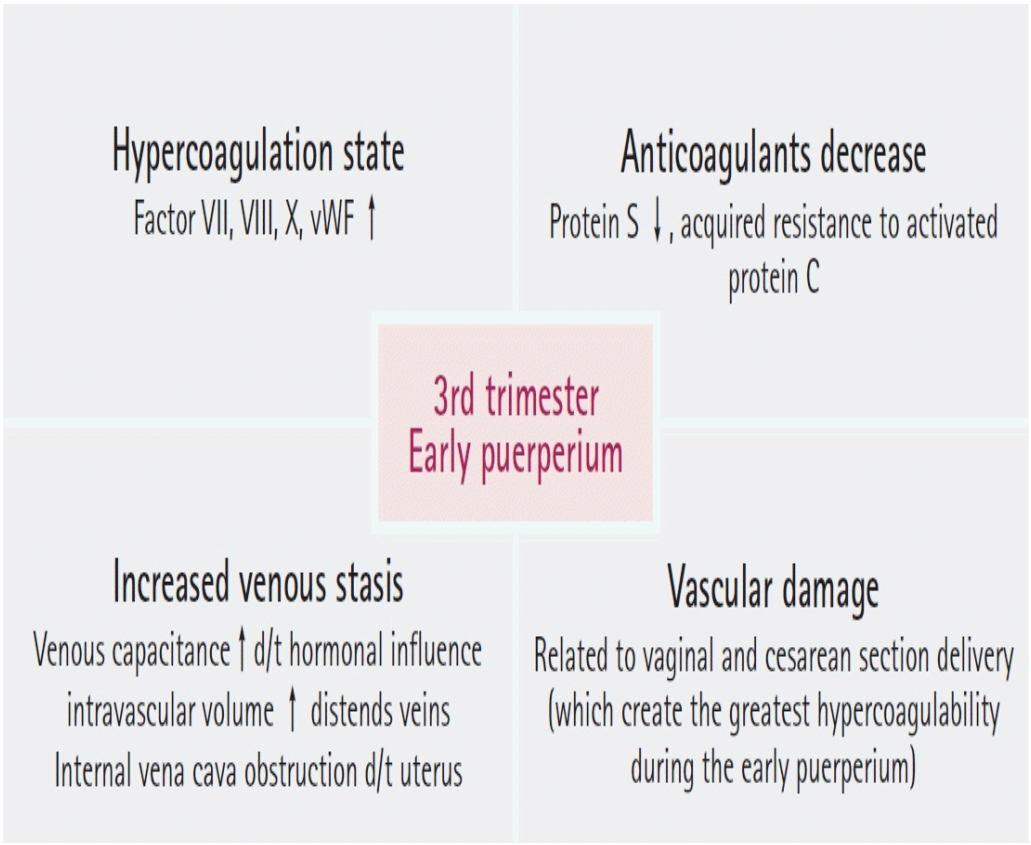
- The blood loss during parturition may lead to infection and dehydration which will worsen the pro coagulant condition[15].

- Operative vaginal delivery and caesarean section will increase the risk of CVT because surgically induced tissue damage causes reduced protein C concentration which is an potent anti coagulant. Prolonged bed rest after surgery decreases the blood flow in the lower limbs leading to venous stasis[29].

- Normal pregnancy itself is not associated with endothelial injury. But during the course of vaginal or abdominal delivery, minimal damage to pelvic vessels may take place. This may increases the risk of developing venous and arterial thromboembolism[29].

- Puerparal CVT usually presents with any of thses risk factor in combination.

- Any of these risk factors may affect the puerperium usually in combination.



H.CLINICAL FEATURES

Studies have found that puerperal CVT shows more acute course and early stabilization when compared to CVT in non pregnant population. Mental status changes are more common with peripartum CVT[51]. Clinical features depend on occluded vein and also the propagation of clot in vascular channel.

❖ Headache:

It is the most common symptom in CVT. It may be due to stretching of nerve fibers in the walls of occluded veins, raised intra cranial pressure or local inflammation surrounding the clot. It can be of a thunderclap type mimicking subarachnoid haemorrhage[52].

- Focal NeurologicalDeficit:

  Transient or long standing due to venous infarct

- Encephalopathy and Convulsions

- Rare clinical presentations:

Migraine with aura, pulsatile tinnitus, isolated psychiatric disturbances, isolated cranial nerve palsy, Sub arachnoid haemorrhage. Some patients may present with psychotic features before manifestations of raised intracranial pressure or focal deficits sets in. Monoplegia (brachial or crural), hemiparesis with leg more affected than arm, intact language despite right hemiparesis are all common but generally regress without residual deficits.

The following figure III, clearly shows the anatomic locations of different venous sinuses with associated symptoms[53.]

FIGURE III - ANATOMIC LOCATIONS OF DIFFERENT VENOUS SINUSES WITH ASSOCIATED SYMPTOMS

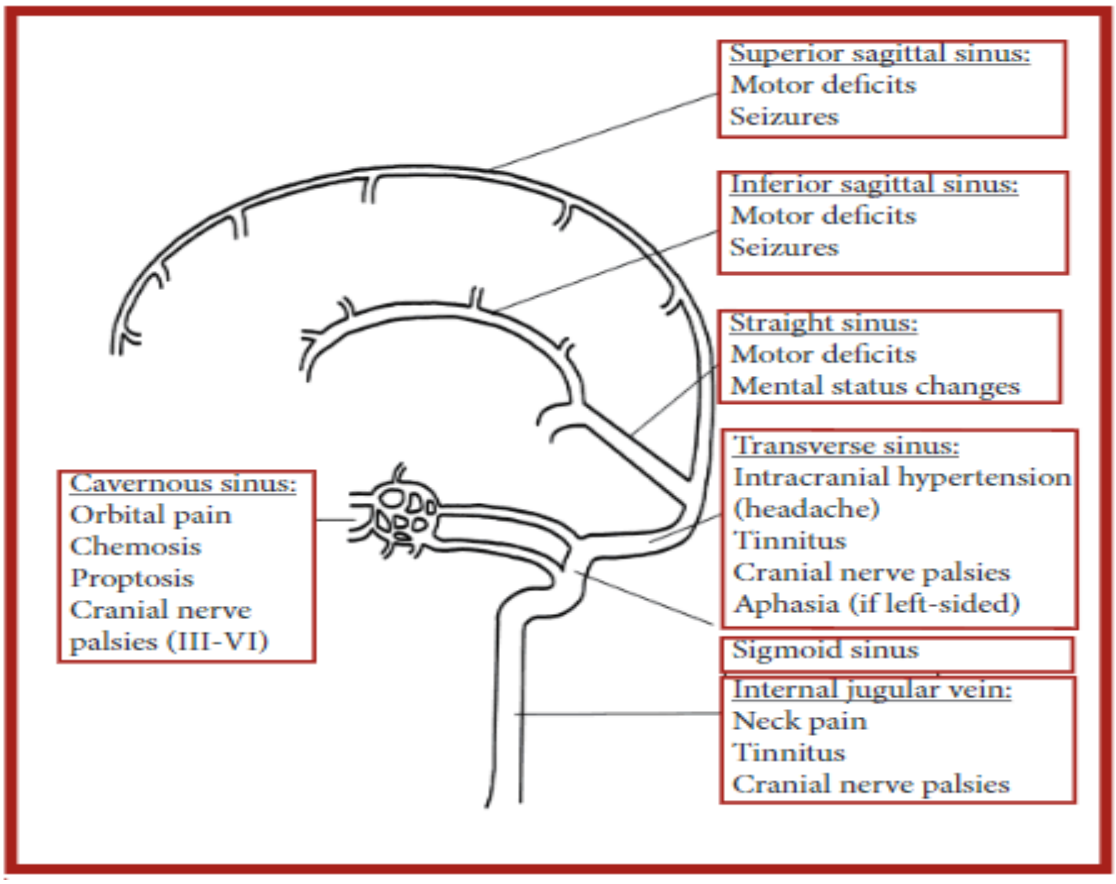


Table 1 - Neurological signs and symptoms in CVT and their frequency (%)[54]

New onset headache	96%
Focal neurological deficit	46%
Paresis of one or more limbs	40%
Convulsions (generalized)	37%
Convulsions (focal)	10%
Papilloedema	40%
Altered consciousness - GCS<14	39%
Coma – GCS<5	15%
Isolated intracranial hypertension	20%
Brainstem/ cerebellar signs	12%
Dysphasia	22%
Visual defects	10%

I.STROKE SYNDROMES OF PUERPERIM

Here is the place to discuss the cerebro vascular events that are specific to puerperium

I.1.ECLAMPSIA AND POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

❖ Studies have shown that upto 5% of pregnancies are complicated by preeclampsia, otherwise known as Pre Eclamptic Toxemia (PET) and 1 in 200 of these women develops eclampsia[55].

❖ PET is a hypertensive disorder specific to pregnancy and it is complicating around 10% of all pregnancies. Gestational hypertension is described as a sustained systolic BP of 140 mmHg or more and/or diastolic BP of 90 mmHg or more which is evident on two occasions minimum 6 hours apart but within 7 days.

❖ National Institute of Health Working Group has classified hypertension in pregnancy as

1. Chronic hypertension
2. Gestational hypertension
3. Pre eclampsia and eclampsia
4. Pre eclampsia superimposed on eclampsia

Incidence of Pre eclampsia and eclampsia varies about 25%-45% of pregnancy-associated Cerebro vascular accidents[29]. The development of PET is manifested by

hypertension, albuminuria and edema. There is generalised occurence of endothelial dysfunction and vasospasm involving all the major organs of body.

❖ Preeclampsia may also manifest before 20 weeks of gestation. This is manifested mainly in thrombophilias, multiple pregnancy and molar pregnancy. Sometimes it may also seen in the postpartum period[29]

❖ Eclampsia is the onset of seizures and/or unexplained coma

during pregnancy or puerperium in a patient with features of pre eclampsia. Incidence of eclampsia in developing countries varies from 1/100 deliveries to 1/1700. The seizures are usually generalized tonic clonic type[56].

❖ Eclampsia is more common in primigravida. The peak incidence is in the teenage years and early 20s.

❖ Sharshar *et al* reported that both haemorrhagic and non-haemorrhagic stroke can occur in eclampsia[21].Studies have reported that patients with pre-eclampsia and eclampsia carry a greater risk for CVT. It has been observed that the most common cause of death in patients with eclampsia is intracerebral haemorrhage.

❖ The risk of ischaemic stroke related to Preeclamptic toxaemia persists even after the postpartum period. Stroke Prevention in Young Women Study suggest that in those with a history of preeclampsia or eclampsia during their pregnancy, the risk of non-pregnancy-related ischaemic stroke in their later period of life is 60% high[57].

❖ However it has been observed that hypertension alone cannot be responsible for the CVA, as intracerebral haemorrhage is relatively uncommon even with sustained elevated blood pressure. James *et al* have published a study stating that more than 75% of patients had diastolic pressure below 105mmHg before the event of

stroke[58].

Pathophysiology

❖ The exact pathophysiology of Pre Eclamptic Toxemia remains uncertain.But endothelial dysfunction and vasospasm appears to have an important role, suggesting relationship between PET and atherosclerosis[59].

❖ The basic pathology is an abnormal cytotrophoblast invasion in the spiral arterioles which is known as defective placentation. This leads to placental ischaemia. The placenta secretes vasoactive substances and leads to endothelialdysfunction[59].

❖ In patients with pre eclampsia plasma rennin activity andangiotensin level are lower than normal. But there is increased responsiveness to the pressor effect of angiotensin[59].

❖ Altered immunological response in the mother and oxidative stress also have a role in the pathogenesis of PET[40].

❖ Around 10% of patients present with Haemolysis, Elevated Liver enzymes and Low platelet count. The name HELLP syndrome was first introduced by Louis Weinstein in 1982. It is associated with multi-organ failure resulting from endothelial damage, fibrin deposition and platelet aggregation. .

❖ History of hypertension, heart disease or CVT in first degree relative carries a higher risk for developing preeclampsia/eclampsia. It supports the role of possible genetic risk factors.

❖ The cerebral autoregulation is maintained between a mean arterial pressure range of 60–150 mm Hg. Chronic hyperventilation in pregnancy may alter this mechanism. Many studies have reported that disturbance in the cerebral autoregulation leads to increased cerebral perfusion pressures which results in baro trauma and damage to the vascular channels[29].

❖ In patients with PET, increased vascular permeability causes thirds

pacing of intravascular fluids. This leads to haemoconcentration and reduced intravascular volume. There is activation of the coagulation cascade with micro-thrombi formation. All these factors contribute to the overall picture of reduced tissue perfusion and greater risk for CVT.

Clinical Features

❖ Most of the patients present with classical features of preeclampsia like high blood pressure, subcutaneous edema and albuminuria.

❖ In eclampsia, patients present with neurological features like headache, vomiting, convulsions and altered level of consciousness. Eclampsia sometimes presents with sudden-onset focal neurological deficit consistent with CVA.

❖ CT or MRI studies in patients with eclampsia revealed arterial ischaemic events or cerebral haemorrhage.

❖ The prognosis is poor in cases with intracerebral haemorrhage. This may be because of that cerebral haemorrhage occurs usually in patients with severe pre-eclampsia, uncontrolled severe hypertension and intense endothelial dysfunction. Jeng JS *et al* reported that haemorrhagic lesions are more common in Asian women than Western women and this carries a higher mortality.

❖ In addition to ischemia and haemorrhage, reversible posterior encephalopathy syndrome [PRES] can also occur in pregnancy and rarely in puerperium as a consequence of uncontrolled preeclampsia. Autoregulatory mechanisms of posterior hemisphere are comparitively weaker than anterior. Patient develops vasogenic edema involving posterior part of cerebral hemisphere. Clinically patients present with headache, altered alertness or behaviour, convulsions and visual loss.

❖ Untreated PRES may lead to severe cerebral ischaemia, infarction and even death[15].

❖ Important neurological problem that mimics eclampsia is cerebral venous thrombosis. But CVT usually prefers the puerperium.

Neuro Imaging

❖ Computed Tomography shows normal study, especially when taken in the first day of the event.

❖ MRI FLAIR sequences show Hyper intensity involving the occipital, parietal and lees frequently posterior frontal lobes. Cortex and subcortical parts of the above areas show diffuse abnormal signal intensities. This is due to the white matter oedema occurring in the posterior part of cerebral hemisphere[42].Bevan H *et al* reported that partial or asymmetric hyper intensities in MRI were more common with eclampsia comparing to other causes of PRES.

❖ Cytotoxic edema occurring due to ischaemic infarction may get confusedwith reversible vasogenic oedema.Diffusion-weighted MRI is useful to differentiate these two conditions.

Management

The only definitive treatment for pre-eclampsia and eclampsia is termination of pregnancy. Pharmacological therapy focuses on the treatment of hypertension and prophylaxis against seizures.

Magnesium sulphate is the first-line therapy in both prophylactic and therapeutic management. It interferes with the action of post synaptic NMDA receptors of brain, inhibits the presynaptic GABA release and also reduces the intracytoplasmic calcium level by inhibiting calcium entry through voltage gated calcium channels.

Figure IV- MRI posterior reversible encephalopathy

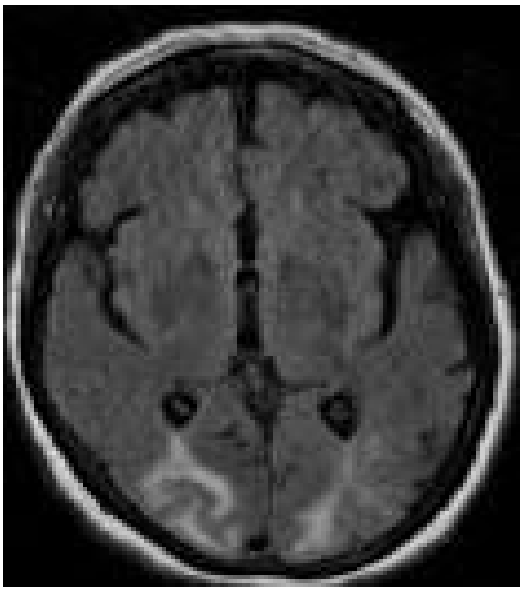
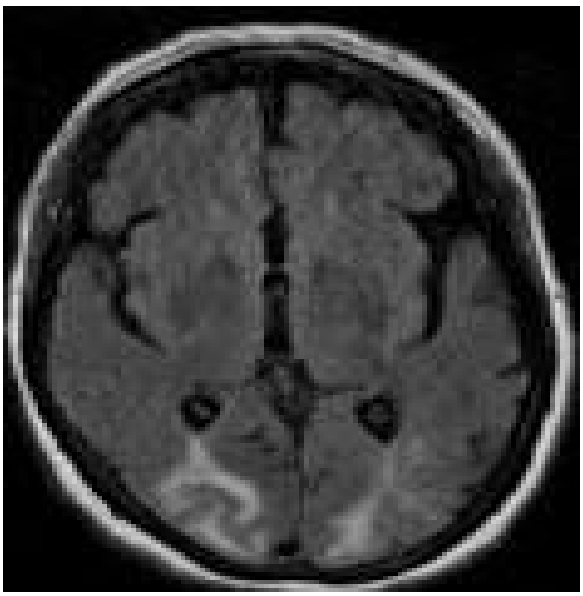


Figure V - MRI posterior reversible encephalopathy



Patients with preeclampsia may have the chance of recurrence in the subsequent pregnancy. One recent publication has shown that patients with pre- eclampsia have increased risk of non-pregnancy related ischaemic stroke in future[60].Women with history of pre eclampsia or eclampsia should be aware of the risk factors for CVT and better to have regular follow up.

I.2.POSTPARTUM CEREBRAL ANGIOPATHY(PCA)

- Postpartum cerebral angiopathy is characterized by reversible multifocal vasospasm involving the cerebral arteries.
- Predisposing factors for PCA are pregnancy and puerperium, drug abuse, migraine, physical injury and hypercalcaemia. Sometimes it occurs without obvious precipitating factor[61].
- This problem may look similar to the syndrome that has been discussed with eclampsia. But antepartum and intrapartum periods are usually uneventful in these patients. PCA commonly manifests a few days after parturition.
- Common clinical features of PCA include acute onset of headache, photosensitivity, vomiting, altered sensorium, convulsions.
- Studies have reported that cerebral vasoconstriction in postpartum cerebral angiopathy can cause a variety of neurological deficits. This is because of transient ischaemia, cerebral infarction and cerebral haemorrhage.

Pathophysiology

The pathophysiology of Postpartum Cerebral Angiopthy remains unclear. A disturbance in the control of vascular tone is likely to be the basic problem. This may look alike to the Posterior Reversible Encephalopathy Syndrome that has been described with eclampsia. Hence few authors mention that these both are same with variable presentation. But in contrast to eclampsia, the pathology of PCA is limited to the nervous system and patients have experienced pregnancy and delivery usually

without any complications. Postpartum Cerebral Angiopathy has been reported with use of drugs causing vasoconstriction such as ergonovine and bromocriptine in the antepartumperiod[29,62].

Diagnosis

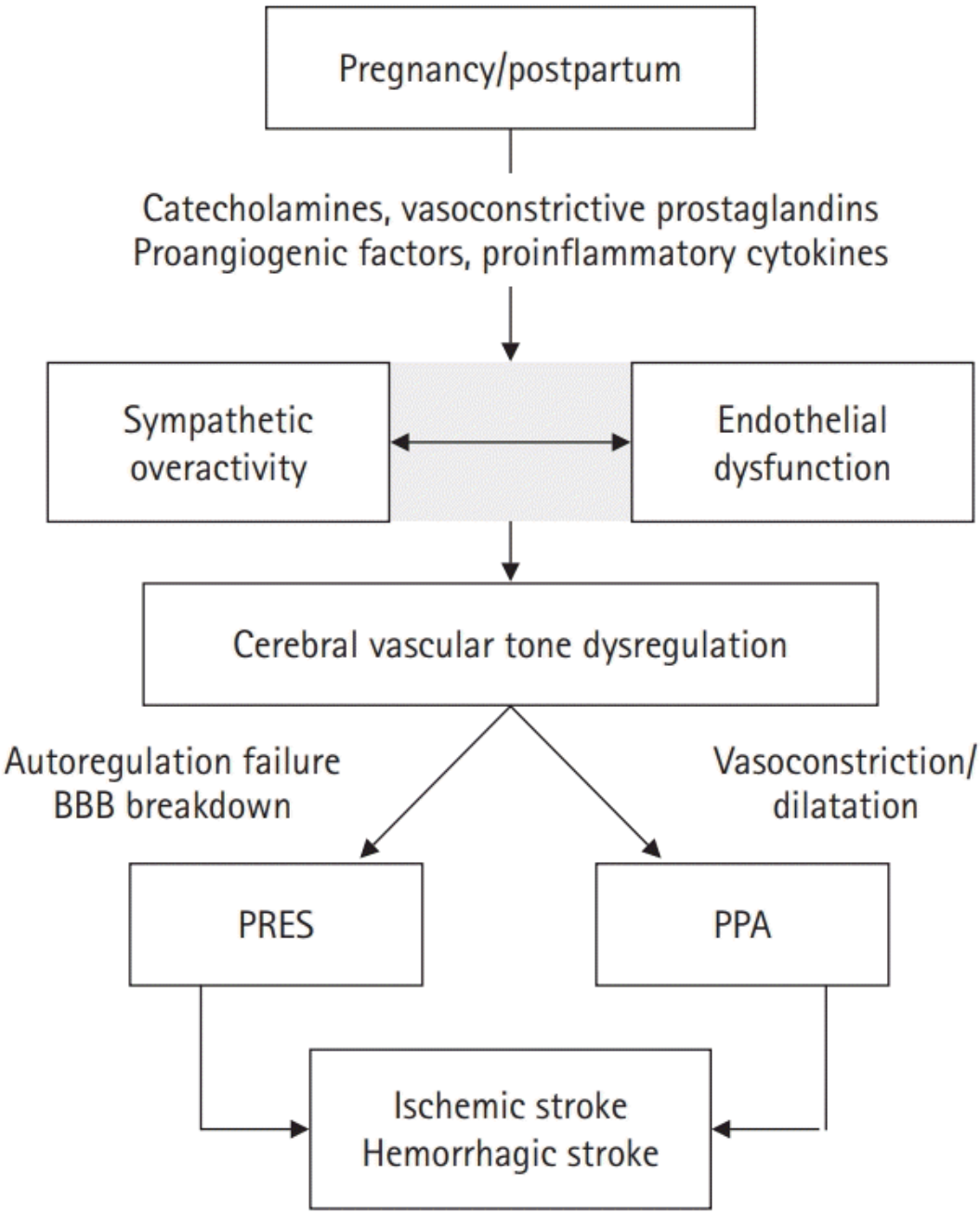
Neuroimaging PCA shows segmental narrowing of the cerebral arteries at multiple sites. But complete recovery is noted in 1-2 months.

Cerebrospinal fluid analysis is useful in differentiating PCA from SAH as CSF analysis is normal in PCA. Brain biopsy may be required sometimes to differentiate PCA and vasculitis as both have different therapeutic implications. The obstetric significance regarding PCA is that it may get confused with CVT especially in postpartum stroke. Radiological investigations help in the diagnosis[29,63].

Treatment

Treatment of PCA is based on observational data obtained through clinical studies. Vasodilators and steroids have been proposed in the management of PCA but the disease process is usually a self-limiting with a benign course. Complete recovery from the symptoms and angiographic findings are observed in 1-2 months. Rarely intracerebral haemorrhage, death and recurrence in subsequent pregnancies may happen. The following picture clearly defines the etiopathogeneis of PRES/PCA.

FIGURE VI - ETIOPATHOGENESIS OF PRES/PCA



J.DIAGNOSIS OF CVT

Objectives of investigations are,

- diagnosis of cerebral vein/sinus thrombosis
- identification of vein/ sinus involved
- identification of underlying pathogenic factors
- evaluation of extent of neural damage

J.1.NEURO IMAGING:

Imaging studies play a major role in accurate diagnosis of Cerebral Venous Thrombosis.

A. Computed tomography: There are direct and indirect signs of CVT[64]

Direct Signs of Cerebral Venous Thrombosis

- The **cord sign** which is visible on CT scans without contrast enhancement represents the thrombosed cortical vein. Since it is a rare sign few studies question its diagnostic value.

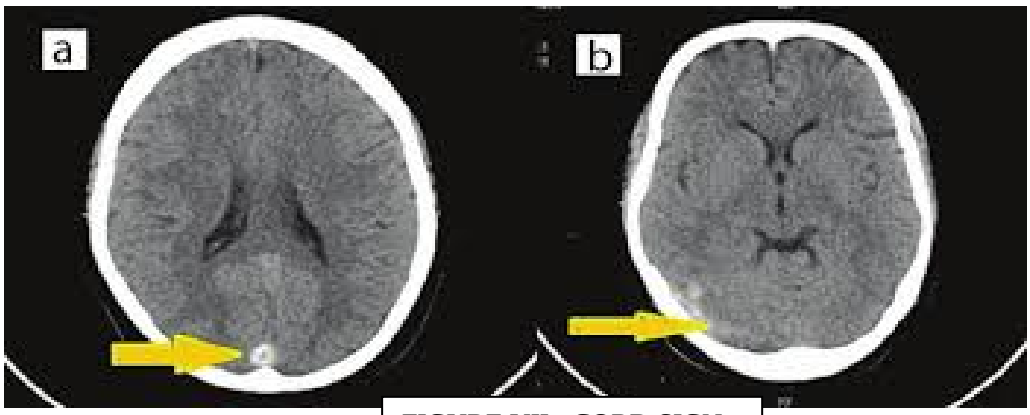


FIGURE VII - CORD SIGN

- The **dense triangle sign:** It is an early sign of SSS thrombosis. Spontaneous opaque triangle area is visualized due to Superior Sagittal Sinus occlusion by freshly coagulated blood. It presents in 2-3% of cases.

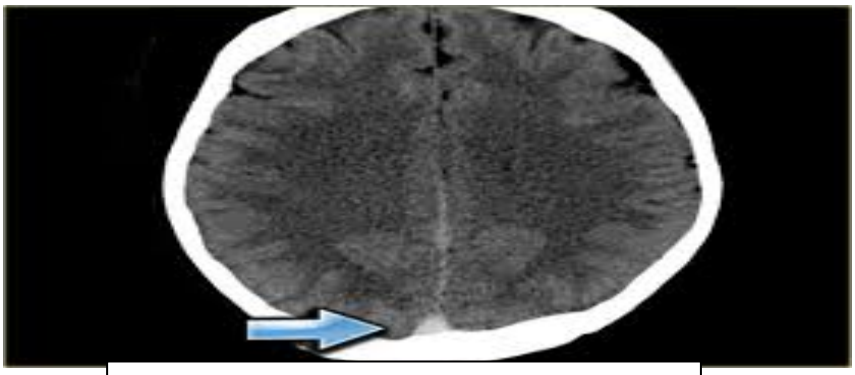


FIGURE VIII - DENSE TRIANGLE SIGN

- The **empty delta sign/Empty triangle sign:** It was described by Buonanno *et al*. It is evident in contrast CT images. It appears as a filling defect in the posterior part of SSS in case of occlusion.

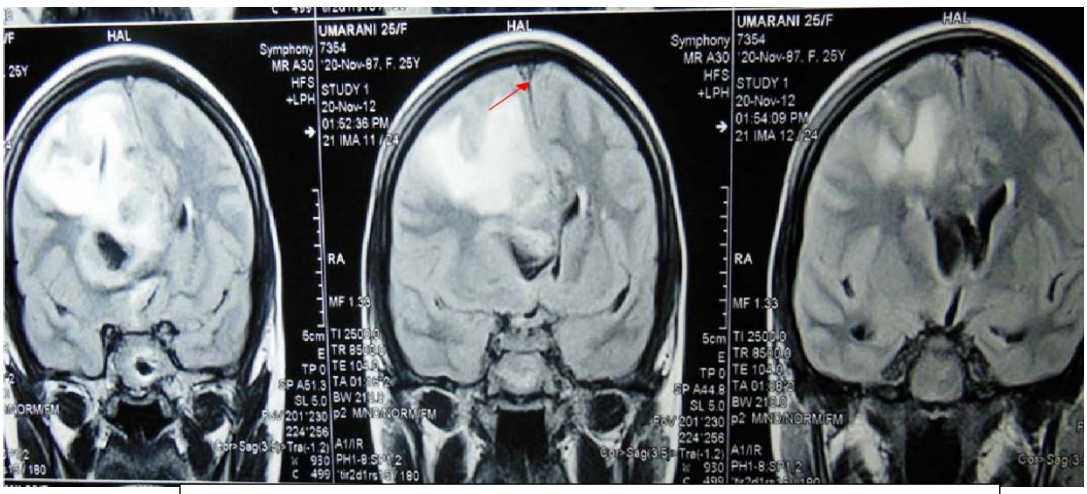


FIGURE IX - EMPTY DELTA SIGN

It is the most common direct sign of CVT. In certain following conditions the empty delta sign is not evident.

(1) Thrombosis not involving the posterior part of the Superior Sagittal Sinus

(2) CT imaging performed either in the first 3 days or after 2 months of onset of symptoms.

False delta sign is noted in cases with early division of SSS.

Indirect Signs in CT imaging:

- Dense contrast enhancement of the falx and tentorium is evident in 20% of patients. It is due to venous stasis or congestion of the dural layer.
- Visualization of small ventricles with swelling and diffuse hypodensity suggests cerebral edema.
- Hypodensity of white matter without contrast enhancement is indicative of cerebral edema. It is present in about 75% of patients. It may be diffuse or localized and is sometimes associated with mass effect.
- Venous infarcts are haemorrhagic infarcts and they appear in CT scan as spontaneous hyperdense lesions in 10% to 50%cases.
- In rare cases, there can be a subarachnoid hemorrhage or subdural hematoma along with other features. Sometimes it may be the only sign of CVT.
- Non - hemorrhagic venous infarcts appear as focal hypodense lesions with or without gyral enhancement.

In 10% to 20% of clinically proven cases, CT imaging may be normal. It is more common with patients presenting with isolated intracranial hypertension than those

with focal neurological features. CT may be normal where the venous infarct has not established completely[65].

Only few patients show direct pathognomonic signs of CVT. But the indirect signs are evident in most of the patients. However, MRI or angiographic confirmation is suggested for appropriate management.

B.Magnetic Resonance Imaging and Venography[66]

The gold standard for the diagnosis of CVT is the combination of **MRI** which localizes the thrombus with **MRV** which shows non visualization of the same. In day 1-5 the lesions appear as isointense area in T1 weighted MRI sequences and Hyperintense area in T2 weighted sequences. In day 5 -15, it becomes hyperintense in both sequences. Brain imaging can be normal 10-20% of patients.

J.2.Trans cranial venous Doppler:

It might be useful in identification of tortuous, distended basal vein which occurs in Superior Sagittal Sinus thrombosis.

J.3.D Dimer ASSAY:

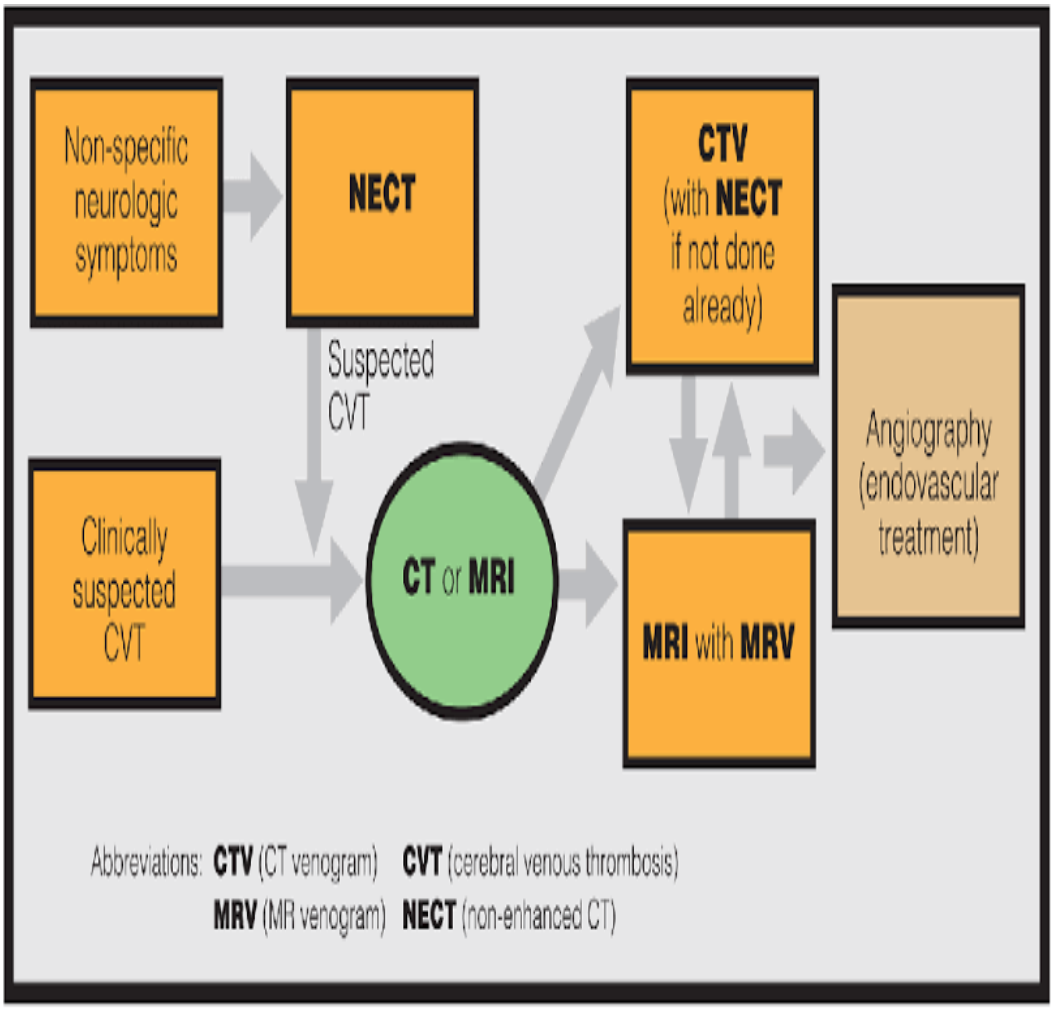
In most patients with recent CVT there is elevation of D Dimer concentration. A low value will rule out the diagnosis.

J.4.LUMBAR PUNCTURE:

It wont helpful in establishing the diagnosis of CVT. It may be used to rule out other causes like meningitis.

J.5.MISCELLANEIOUS: Investigations to diagnose pro thrombotic conditions like APLA syndrome, Protein C or S deficiency, Factor V Leiden mutation, Homocysteinemia.

Algorithum for imaging in CVT cases



K.TREATMENT OF CEREBRAL VEIN THROMBOSIS

Heparin anticoagulation is a time honoured treatment and it is used in all cases of CVT irrespective of the etiology.[69]

Aims of anticoagulation are,

- prevent extention of the thrombus
- recanalise the occluded vein or sinus
- treat the underlying prothrombotic state
- prevent formation of thrombus in other parts of body
- prevent recurrence

Dose:

The dose is variable and adjusted to maintain APTT at 1.5 to 2 times normal. Started as 5000 units iv bolus followed by infusion of 1000 units per hour. Subcutaneous route has unreliable bioavailability and delayed onset.

Instead of Unfractionated heparin, Low molecular weight heparins like Enoxaparin, Dalteparin are used because of their reliable pharmacokinetics and poor side effects. Regular monitoring with coagulation profile is not required.

Heparin reduces both mortality and morbidity in CVT. Haemorrhagic infarct is not a contraindication for heparin therapy[41].Thrombolytic drugs like streptokinase and urokinase are also been tried in some patients and found to be effective.

Symptomatic management:

- Patient with raised ICT will improve with osmotic diuretics like mannitol, head up position, hyperventilation
- Anti epileptic drugs to control seizures

- Steroids have no role even in patients with parenchymal lesions
- Physiotherapy
- Hydration
- Adequate care must be taken to prevent aspiration and bedsores.

Long term management

After the acute stage heparin is replaced by Warfarin for 6-12 months aiming an INR of 2-3. Patients with Thrombo embolic diseases like APLA syndrome are advised to have lifelong prophylaxis[43].

CRMD RECOMMENDATIONS:

Confidential Review of Maternal Deaths[70], Kerala 2004-05 recommends the following in view of prevention of CVT.

- Early ambulation
- Early and adequate fluid intake
- Use of elastic compression stockings
- Change in the concept about BEDREST

Thrombo prophylaxis if more than 3 of following risk factors exist (moderate risk)

- Obesity (BMI >30%)
- Age >35
- Multiple pregnancy
- Extensive varicose veins

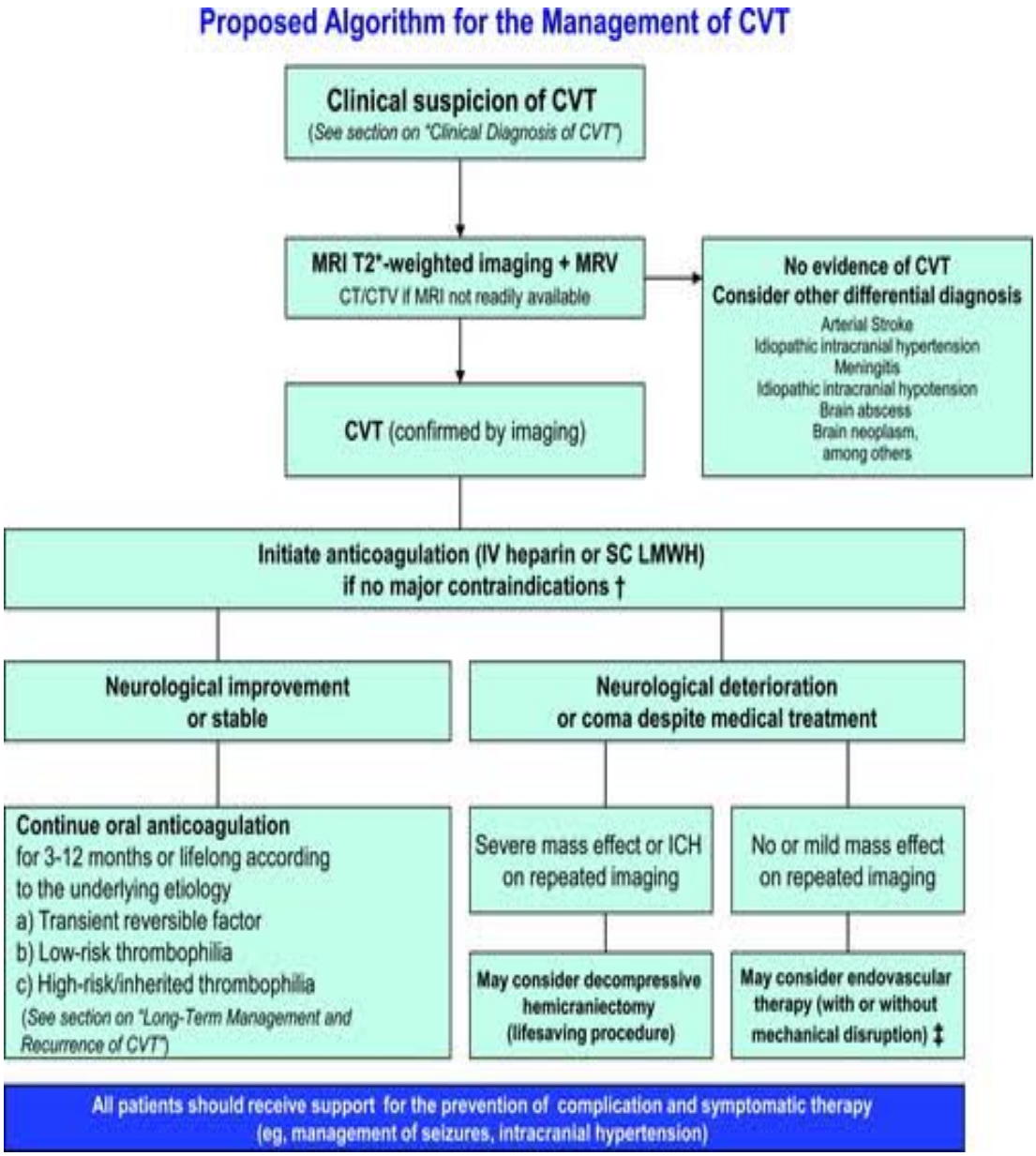
- Air travel
- Cesarean or cesarean hysterectomy
- Sickle cell anemia
- Enforced bed rest > 4days
- APLA syndrome
- Thrombophilia

Prophylactic heparin

• LMWH 5000 units or UFH 5000 units twice daily to be started 4 - 6 hrs after Vaginal delivery and 8 hrs after Cesarean section

Continue for 3 – 5 days or till the patient becomes fully ambulant.

THE AMERICAN STORKE ASSOCIATION 2019 GUIDELINES[71] recommends the following for management in CVT.



L.OUTCOME IN CVT:

Functional Recovery

In patients with venous thrombosis, the chance for recovery of function is much better when compared to arterial thrombosis. Among all underlying conditions, the puerperal state is a favourable one with 80-90% survival. Cantu et al has mentioned that favourable outcome in puerperal CVT may be due to limited and transient occlusion with rapid recanalization or formation of collaterals.

- Residual seizures has been reported in 10-30% of patients who had seizures during the acute stage of CVT.
- Recurrence of venous thrombosis at another site can occur in patients with prothrombotic states, but is generally uncommon with long term anticoagulation.
- One hundred of 138 cases of cerebral venous thrombosis related to pregnancy and the puerperium recovered completely in the series of Bansal et al[33].
- Srinivasan K in India observed 135 patients with CVT related to pregnancy (129 venous thrombosis and 6 arterial thrombosis).Among them 80 recovered without significant neurological deficit. Fifty of these cases followed up for 2 years were doing well. 10 patients had residual focal neurologic deficit without disability and 10 had recurrent seizures[34].

Mortality:

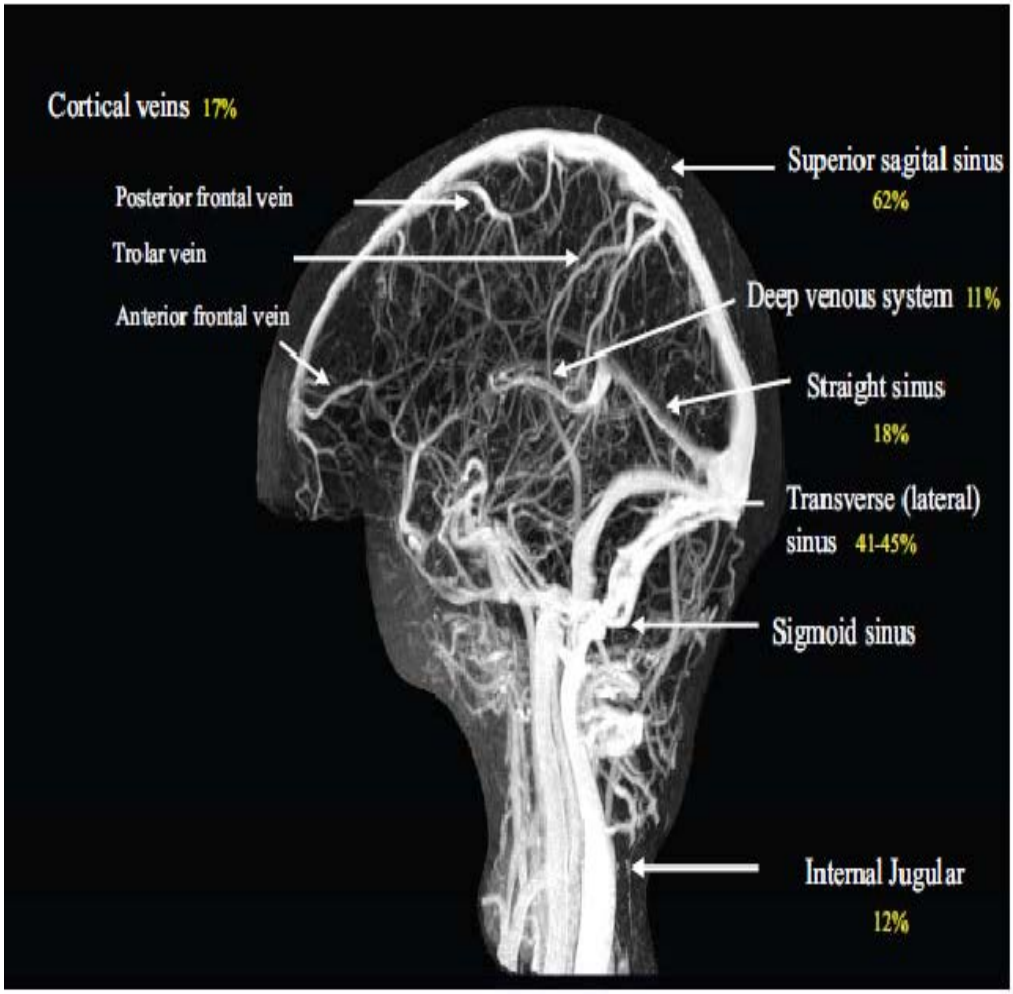
The main causes of mortality in Cerebral Vein Thrombosis are,

- ❖ Brain lesion itself, particularly large hemorrhagic infarcts
- ❖ Associated complications like sepsis, uncontrolled seizures and pulmonary embolism.
- ❖ Underlying conditions like carcinoma, septicemia, leukemia andParoxysmal Nocturnal Haemoglobinuria.

Factors suggestive of bad prognosis are as follows[15,35]:

- Rate of evolution of thrombus
- Age of the patient
- Infection as a cause for CVT
- Severe focal symptoms and coma
- Presence of hemorrhagic infarct
- Empty delta sign on CT scan
- Mass effect with midline shift

Figure X - Cantu et al[7] 1993- Diagramatic reprenstation of Radiological findings



Radiological findings in a series of 113 patients by Cantu et al7 1993

Findings	**Computed Tomographic Scan**		**Magnetic Resonance Imaging**	
	Puerperal	**Non-puerperal**	**Puerperal**	**Non-puerperal**
Normal	5 (8.4%)	4(11.1%)	0 (0%)	0 (0%)
Signs of CVT*	19 (32.2%)	13 (36.1%)	17 (89.4%)	19 (95%)
Non-haemorrhagic venous infarct	16 (27.1%)	7(19.4%)	3 (15.7%)	2 (10%)
Haemorrhagic venous infarct	21(35.5%)	12(33.3%)	10 (52.6%)	11 (55%)
Intracerebral haemorrhage	6 (10.1%)	5 (13.8%)	2 (10.5%)	4 (20%)
Unilateral lesions	25 (42.3%)	15 (41.6%)	8 (42.1%)	12 (60%)
Bilateral lesions	18(30.5%)	9 (25%)	7 (36.8%)	5 (25%)

* delta sign, dense triangle or empty delta sign

METHODOLOGY

Study design : Open labelled pospective observational non interventional study

Study centre : Department of Obstetrics and Gynaecology, Govt. Mohan Kumaramangalam Medical College Hospital.

Study Population : The study was conducted on 75 radiologically confirmed cases of cerebral venous thrombosis admitted in Obstetrics and Gynaecology department of Govt. Mohan Kumaramangalam Medical College Hospital

Study Period : February 2018 to July 2019.(18 months)

Inclusion criteria:

- Postnatal mothers with confirmed clinical and radiological diagnosis of cerebral venous thrombosis within 6 weeks of delivery.
- Exclusion criteria:
- Patients whose clinical presentation could be explained by any other neurological disease.
- Women without radiological evidence of CVT.
- Women who delivered 6 weeks ago.
- Women who do not want to part of study.

Data Collection:

All patients admitted during the study period are subjected to analysis based on a preformed proforma.

- Age of the mother, socio economic status, parity, prescence of predisposing factors, mode of delivery, time of presentation, treatment given, hospital course, outcome were noted.

- Detailed analysis of the collected data was done at the end to find predisposing factors and the spectrum of postpartum CVT.

- Assessment of consciousness level, Glascow coma scale score at the time of admission were also recorded in all patients. .

- Neuroimaging in the form of CT brain and MRI brain with MRA and MRV was done in all patients.

- Details like presence of cerebral edema, haemorrhagic infarct, nonhaemorrhagic infarct, presence of direct signs like cord sign, dense delta sign etc., occurrence of focal or diffuse subarachnoid haemorrhage in CT brain were recorded.

- In MRI with MRV, type and number of sinuses involved, involvement of cortical veins, internal jugular vein extension, and laterality of the sinuses involved were noted.

- Investigations like complete blood count, erythrocyte sedimentation rate (ESR), blood urea, blood sugar, serum creatinine, serum electrolytes, lipid profile, X-ray chest, Electrocardiogram, ELISA for Human Immunodeficiency Virus (HIV),VDRL, coagulation profile including bleeding time, clotting time, prothrombin time, activated partial thromboplastin time were done in all patients and investigations like antinuclear antibody (ANA), antiphospholipid

antibodies, tests for procoagulant states like protein C, protein S, antithrombin III (AT III) and serum homocysteine with an aim to detect the underlying etiology were done in certain patients as needed.

- Outcome at the end of the hospital stay was recorded in all patients. The modified Rankin score was used for outcome assessment. The modified Rankin Scale (mRS) is commonly used for expressing the degree of disability of post stroke patients as well as those with other neurological disability. Hence it is widely used in the analysis of outcome in stroke clinical trials. mRS was actually introduced by Dr. John Rankin of Stobhill Hospital Glasgow, Scotland in 1957 and was first modified by Prof. C. Warlow's group at Western General Hospital in Edinburgh . The currently used modified Rankin Scale was given by van Swieten, et al., in 1988.The scoring is done from 0 to 6 as follows,

0 - No symptoms.

1 - No significant disability. Able to carry out all usual activities, despite some symptoms.

2 - Slight disability. Able to look after own affairs without assistance, but unable to carry out all previous activities.

3 - Moderate disability. Requires some help, but able to walk unassisted.

4 - Moderately severe disability. Unable to attend to own bodily needs without assistance and unable to walk unassisted.

5 - Severe disability. Requires constant nursing care and attention,bedridden, incontinent.

6 -Dead.

In our study, good outcome was defined as modified Rankin Scale score of 0 to 2 and poor outcome was defined as modified Rankin Scale score of >2 . Factors associated with good and poor outcome were also analysed in our study. These demographic, clinical, laboratory, neuroimaging, outcome data were recorded and analysed using a standard proforma. Statistical analysis was done using SPSS software.

The study was approved by the ethical committee of our hospital.

RESULTS AND ANALYSIS

Our study was conducted GOVT MOHAN KUMARAMANGALAM MEDICAL COLLEGE AND HOSPITAL, Salem during a period of 18 months and the results were studied.

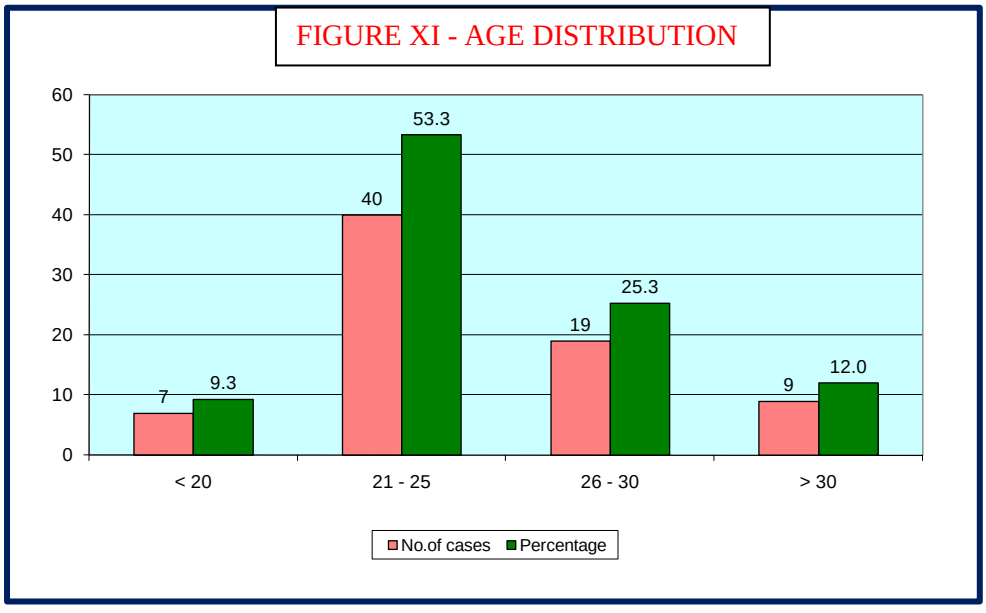
The results of the study are documented below.

AGE DISTRIBUTION

Table II

Age in years	No.of cases	Percentage
≤ 20	7	9.3
21 - 25	40	53.3
26 - 30	19	25.3
> 30	9	12.0
Total	75	100

Maximum occurrence of CVT was noted in the age group of 21-25 years contributing to 53.3%. Similarly the incidence of 25.3% was noted in 26-30yr. The youngest age of stroke is 16 yr old girl with moderate anemia and severe preeclampsia.



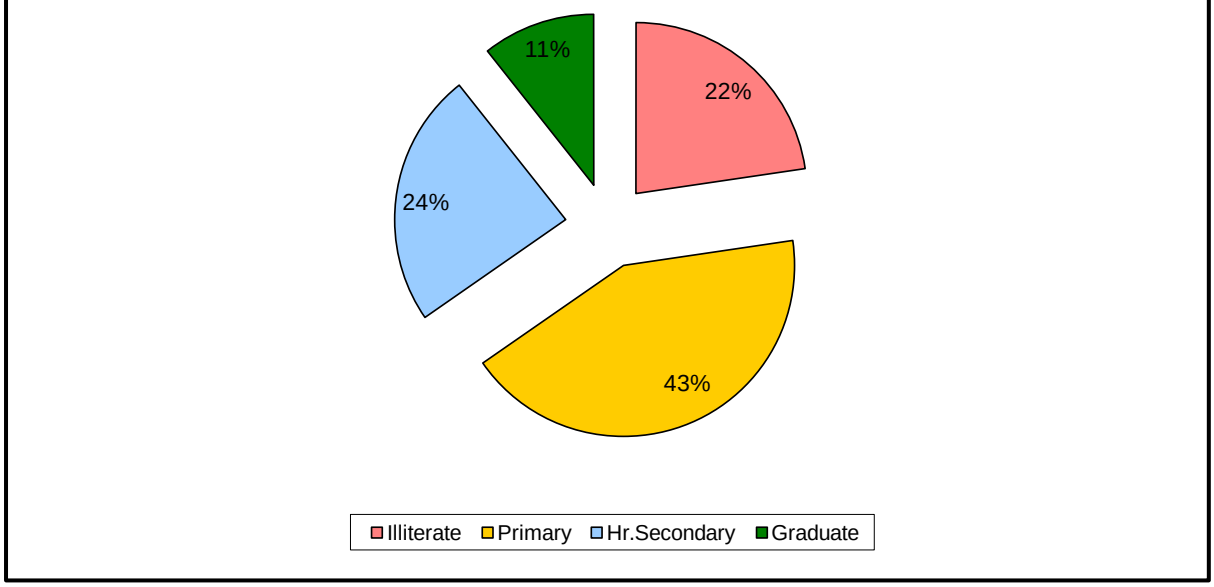
EDUCATION :

TABLE III

Education	No.of cases	Percentage
Illiterate	17	22.7
Primary	32	42.7
Hr. Secondary	18	24.0
Graduate	8	10.7
Total	75	100.0

Maximum incidence of CVT is seen in women who studied primary class 42.7%. This was followed by women who studied higher classes & illiterates as 24% & 22.7% respectively.

FIGURE XII - EDUCATION LEVEL OF PATIENTS

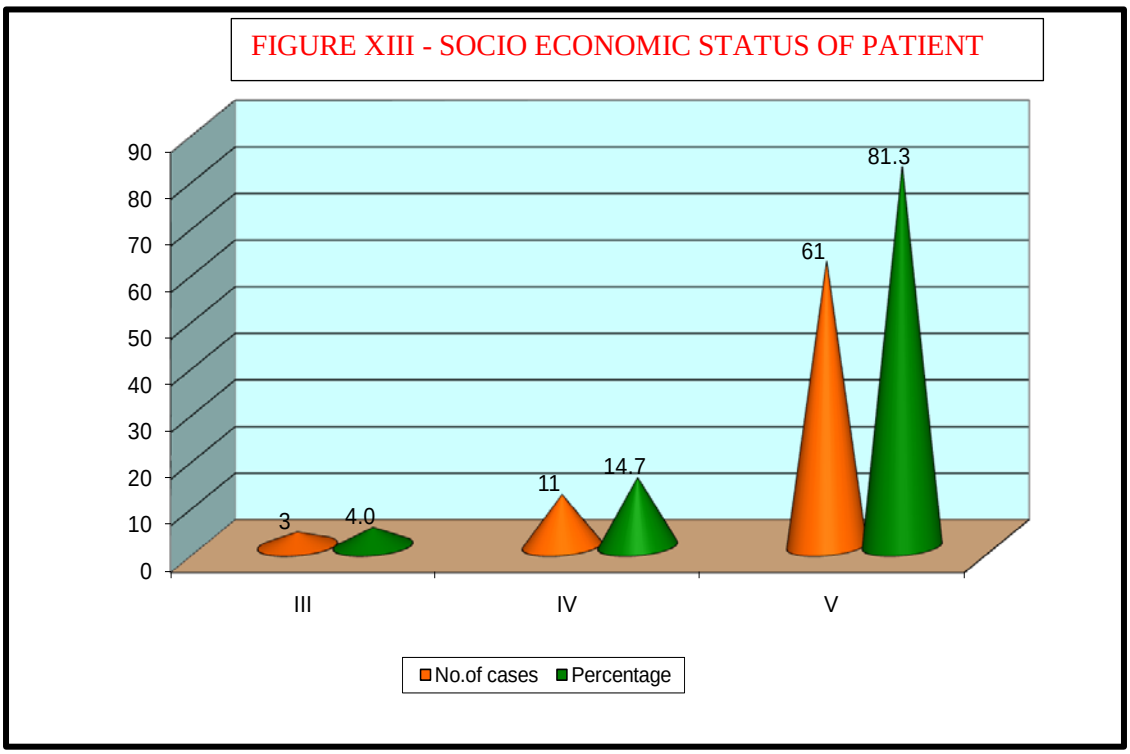


SOCIO ECONOMIC STATUS :

TABLE IV

Socio economic status	No.of cases	Percentage
III	3	4.0
IV	11	14.7
V	61	81.3
Total	75	100.0

Patients were classified using Modified Kuppusamy Scale. 81.3% of patients were from class V socioeconomic class followed by class IV with 14.7%.



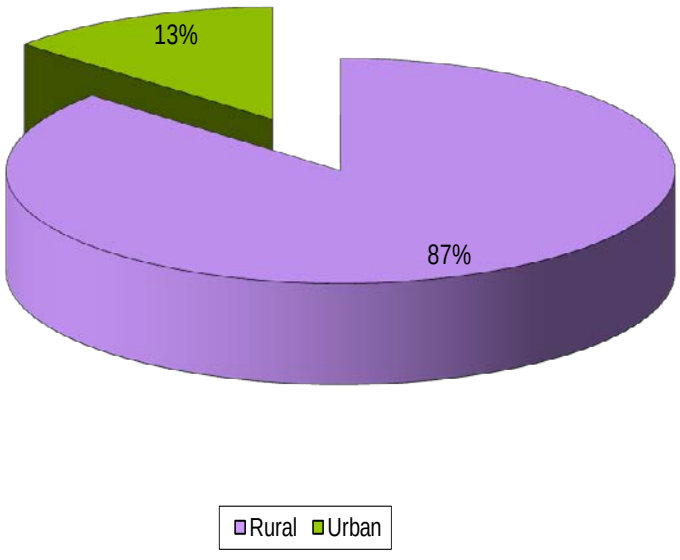
AREA DISTRIBUTION :

TABLE V

Area	No.of cases	Percentage
Rural	65	86.7
Urban	10	13.3
Total	75	100.0

Maximum occurrence was noted in patients from rural area contributing to 86.7%.

FIGURE XIV - AREA WISE DISTRIBUTION OF PATIENTS



PARITY DISTRIBUTION

TABLE VI

Parity	No.of cases	Percentage
P1	45	60.0
P2	18	24.0
P3	9	12.0
P4	3	4.0
Total	75	100.0

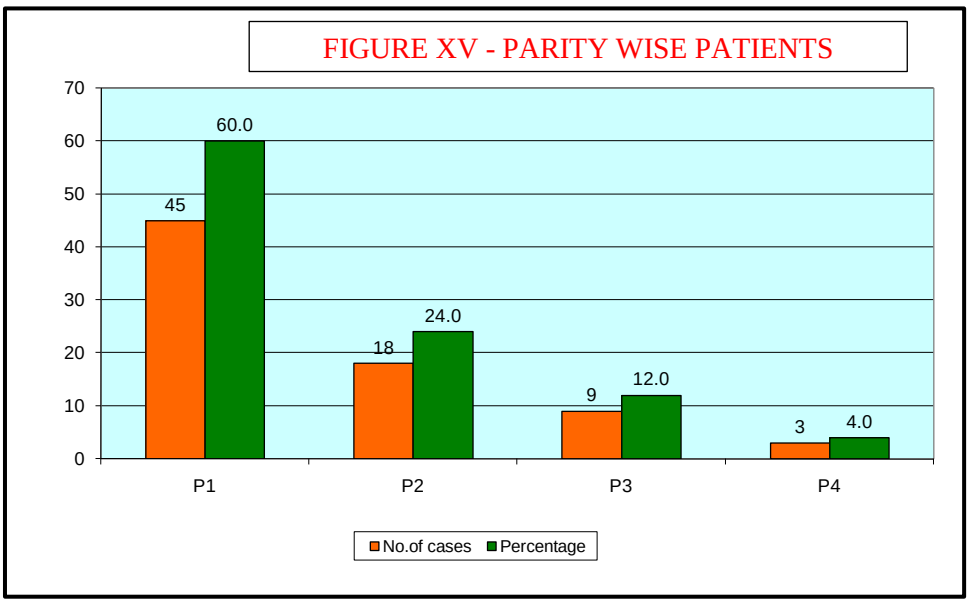
P1 - Postnatal patients after first delivery(primi para) irrespective of the outcome of delivery.

P2 - Postnatal patients after second delivery irrespective of outcome of previous and present pregnancy

P3 - Postnatal patient after third delivery irrespective of outcome of pregnancies

P4 - Postnatal patient after fourth delivery irrespective of outcome of pregnancies

60% of patients were primipara showing the highest incidence, followed by P2 group with 24%.

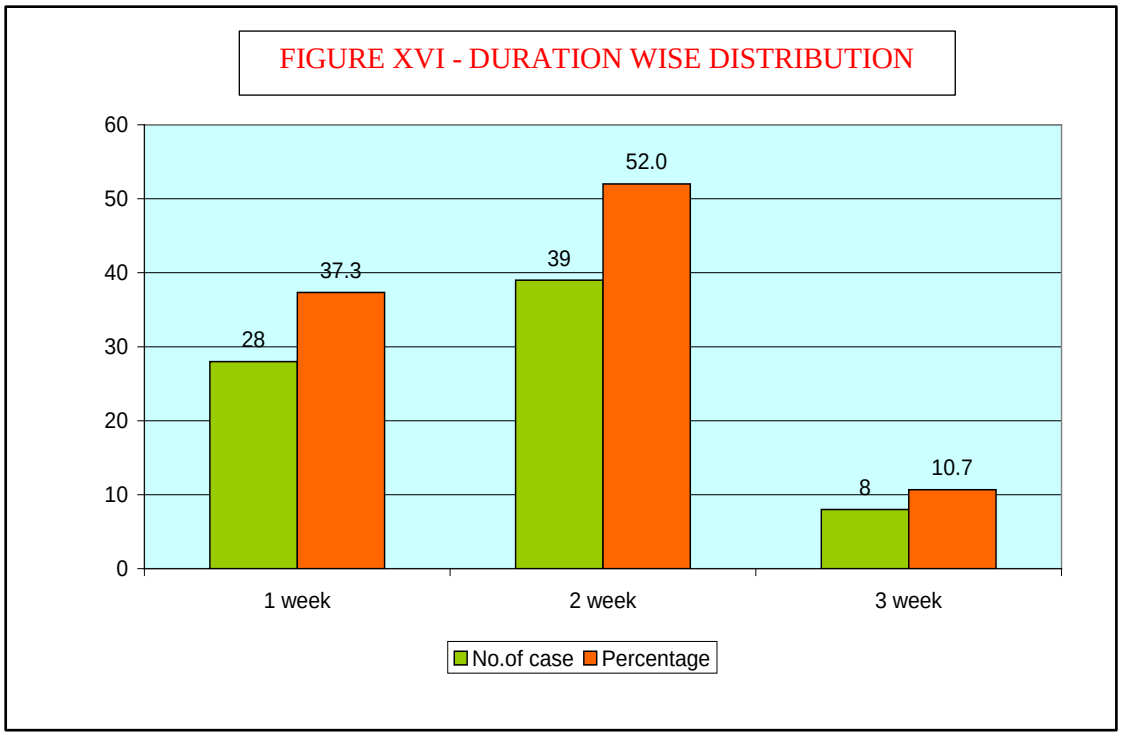


TIME OF PRESENTATION :

TABLE VII

Duration	No.of cases	Percentage
1 week	28	37.3
2 week	39	52.0
3 week	8	10.7
Total	75	100.0

52% of patients had CVT in the 2nd week of postpartum period, followed by 28% of postnatal women in the first week of postpartum.

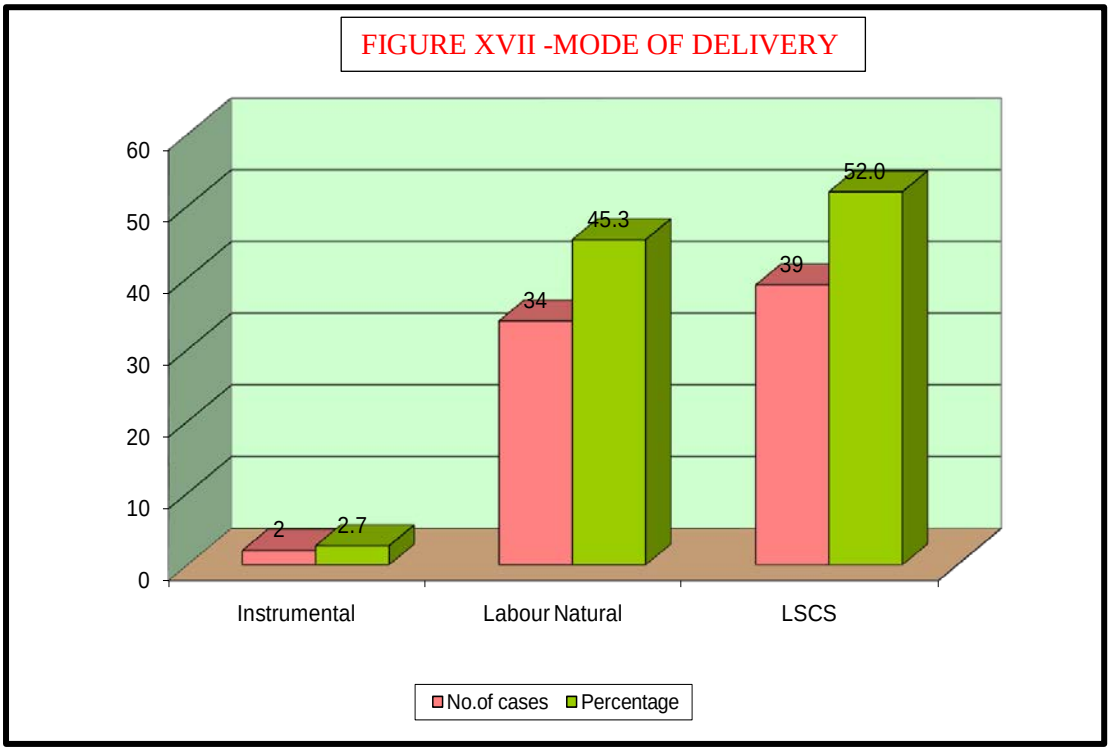


MODE OF DELIVERY

TABLE VIII

Mode of delivery	No.of cases	Percentage
Instrumental	2	2.7
Labour Natural	34	45.3
LSCS	39	52.0
Total	75	100.0

Of all the patients with postpartum CVA 52% had undergone caeserean section due to various reasons. 48% had undergone vaginal deliveries either labour natural or instrumental delivery.



CO-MORBIDITY

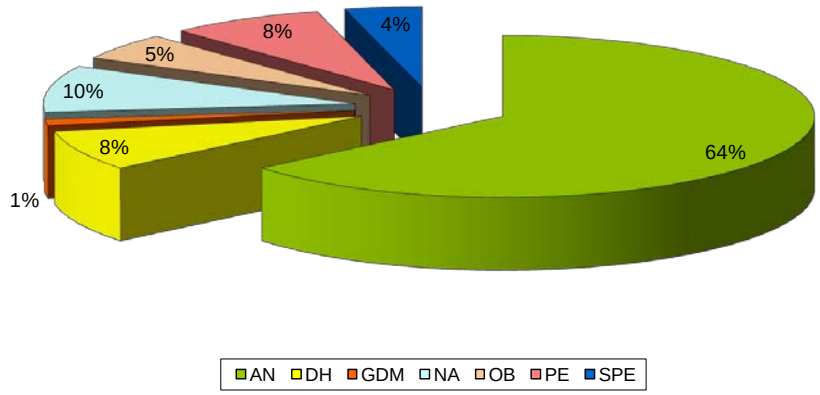
TABLE IX

CO MORBIDITY	No.of cases	Percentage
ANEMIA	48	64.0
DEHYDRATION	26	34.67
GDM/DM	5	6.67
HYPERTENSIVE DISORDERS	36	48
OBESITY	12	16
MULTIPLE PREGNANCY	6	8.0
BLOOD TRANSFUSION	32	42.67
LSCS	39	52.0

In our study we found Anemia (64%), LSCS(52%), Hypertensive disorders (48%), Blood transfusion (42.2%), Dehydration (34.67%) are the most important predisposing factor for Cerebral venous throbosis..

The following figure shows the comorbidities associated with cerebral venous thrombosis.

FIGURE XVIII - COMORBIDITIES

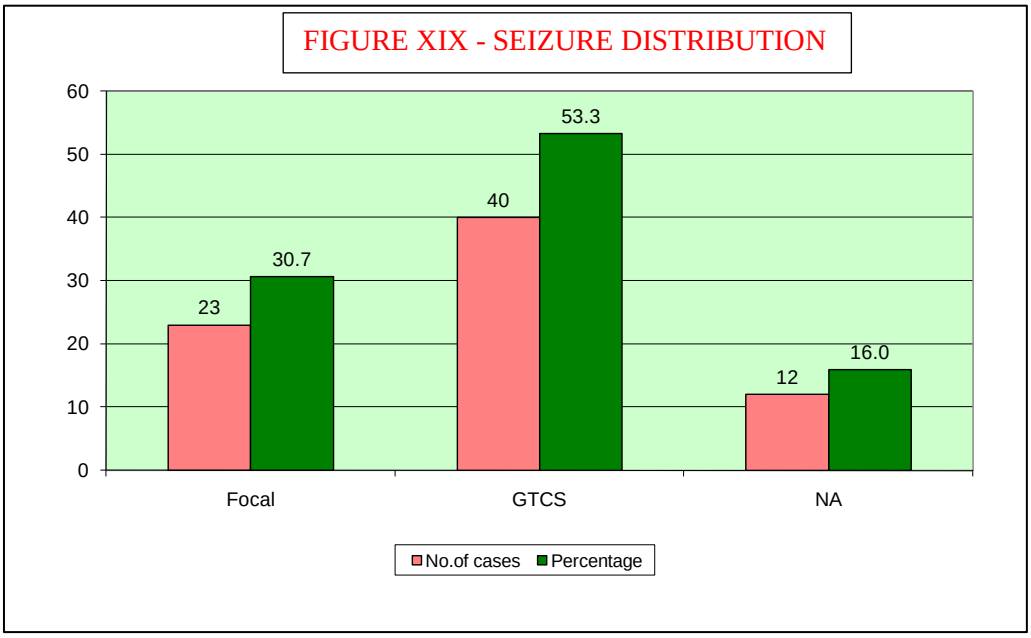


SEIZURE DISTRIBUTION :

TABLE X

Seizure	No.of cases	Percentage
Focal	23	30.7
GTCS	40	53.3
NA	12	16.0
Total	75	100.0

84% of patients had seizure which is either focal or generalized seizures. Early appearence of seizure might be hallmark of bad prognosis. 53.3% had GTCS and 30.7% had focal seizures.



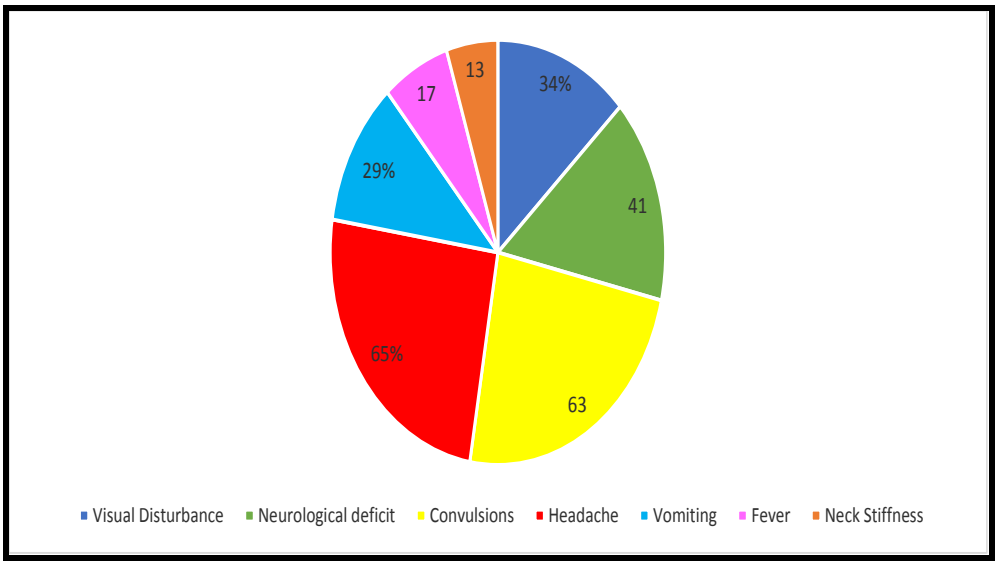
SYMPTOMATOLOGY :

TABLE XI

Symptoms	No.of cases	Percentage
Altered sensorium	52	69.33
Visual distrubanne	34	45.33
Neurological deficit	41	54.67
Convulsions	63	84.00
Headache	65	86.67
Vomitting	29	38.67
Fever	17	22.67
Neck stiffness	13	17.30

In our study the most common symptom noted was Headache which was present in 86.67% of patients. It was followed by convulsions 84% and altered sensorium 69.33%.

FIGURE XX - SYMPTOMOLOGY

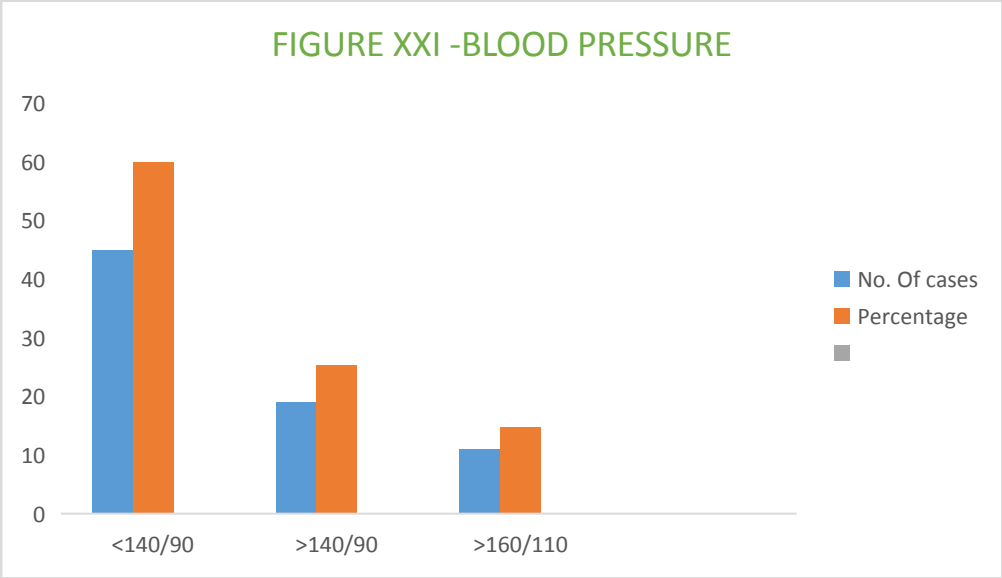


BLOOD PRESSURE :

TABLE XII

BLOOD PRESSURE	No.of cases	Percentage
<140 systolic and or 90mmHg diastolic	45	60.0
≥140systoic and or 90mmHg diastolic	19	25.3
≥160 systolic and or 110mmHg diastolic	11	14.7
Total	75	100.0

In the present study 25.30% of patients blood pressure between 140/90 mmHg and 160/110 mmHg and 14.70% patients had blood pressure of more than 160/110 mmHg. 60% of patients had blood pressure below 140/90 mmHg .



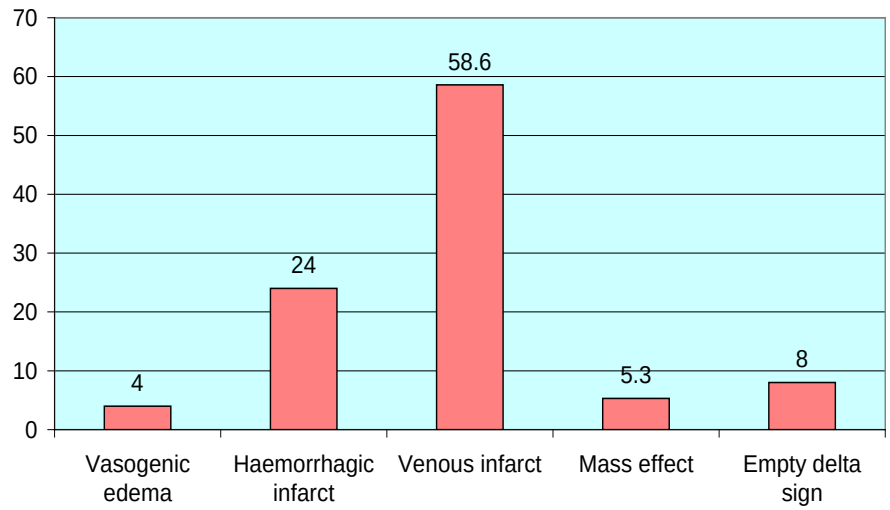
RADIOLOGICAL FINDINGS

TABLE XIII

RADIOLOGICAL FINDINGS	No.of cases	Percentage
Vasogenic edema	15	20.0
Haemorrhagic infarct	18	24.0
Venous infarct	44	58.6
Mass effect	14	18.67
Empty delta sign	9	12.0

Most common feature was Venous infarct with 58.60% followed by haemorrhagic infarct with 24%. Empty delta sign was present 12% patients. Mass effect with midline shift was present in 18.67% of patients which indicates bad prognosis.

FIGURE XXII - RADIOLOGICAL FINDINGS

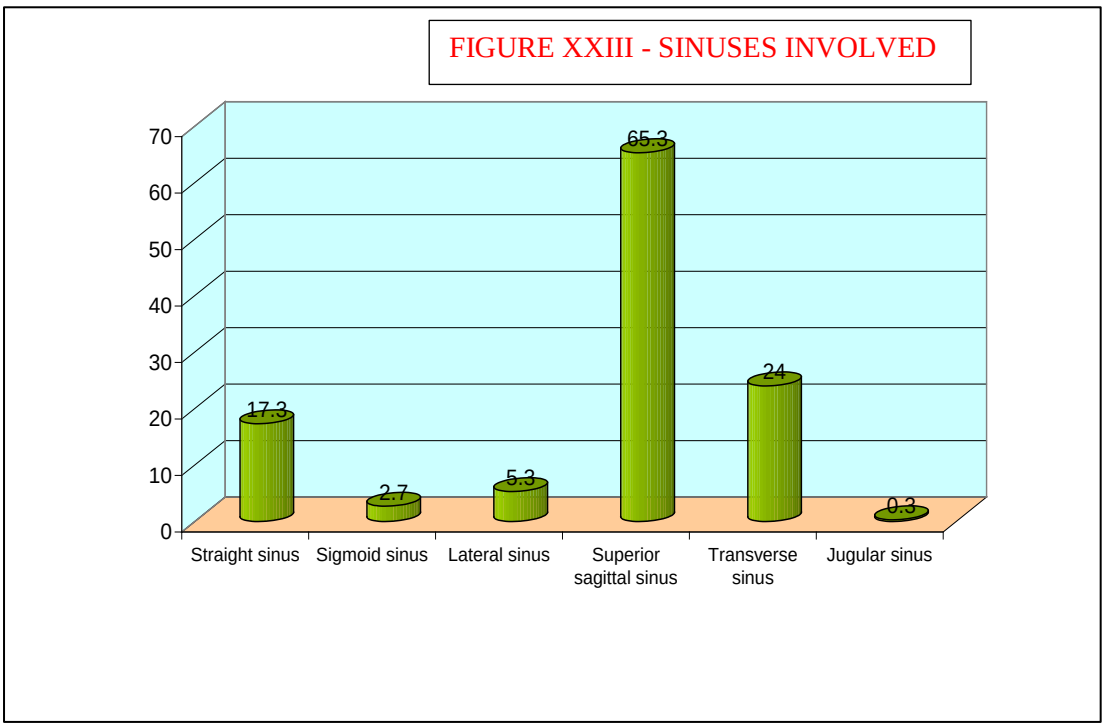


SINUS INVOLVED

TABLE XIV

SINUS INVOLVED	No.of cases	Percentage
Straight sinus	13	17.3
Sigmoid sinus	8	2.7
Lateral sinus	11	5.3
Superior sagittal sinus	49	65.3
Transverse sinus	18	24.0
Jugular sinus	2	0.3

The most common sinus involved was Superior Sagittal Sinus - 65.30% , followed by the transverse sinus with 24%.

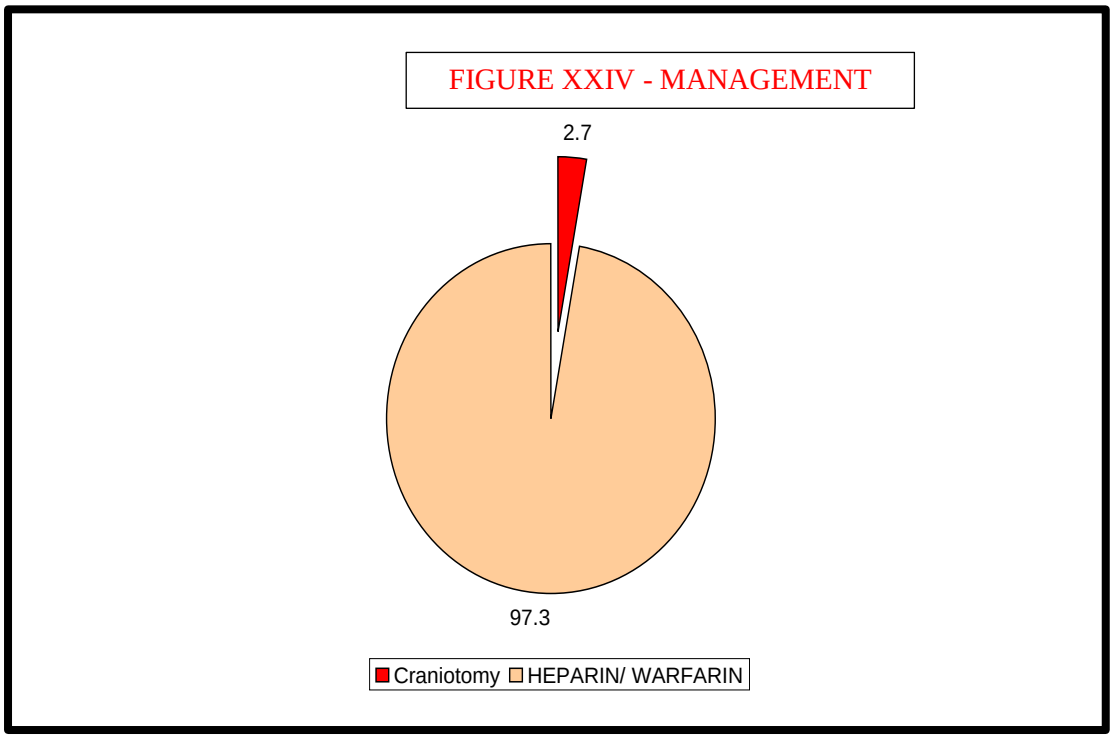


MANAGEMENT :

TABLE XV

Management	No.of cases	Percentage
Decompressive Craniotomy	3	4
HEPARIN/ WARFARIN	72	96
Total	75	100.0

Decompressive craniotomy was done in 3 patients with midline shift, achieved good prognosis in teo of these. Rest others managed with Inj.Heparin and lateral changed to Oral anticoagulant.



The following picture shows the intra operative and post operative picture after left fronto tempero- parietal decompressive craniotomy.

FIGURE XXV - INTRA – OPERATIVE PICTURE

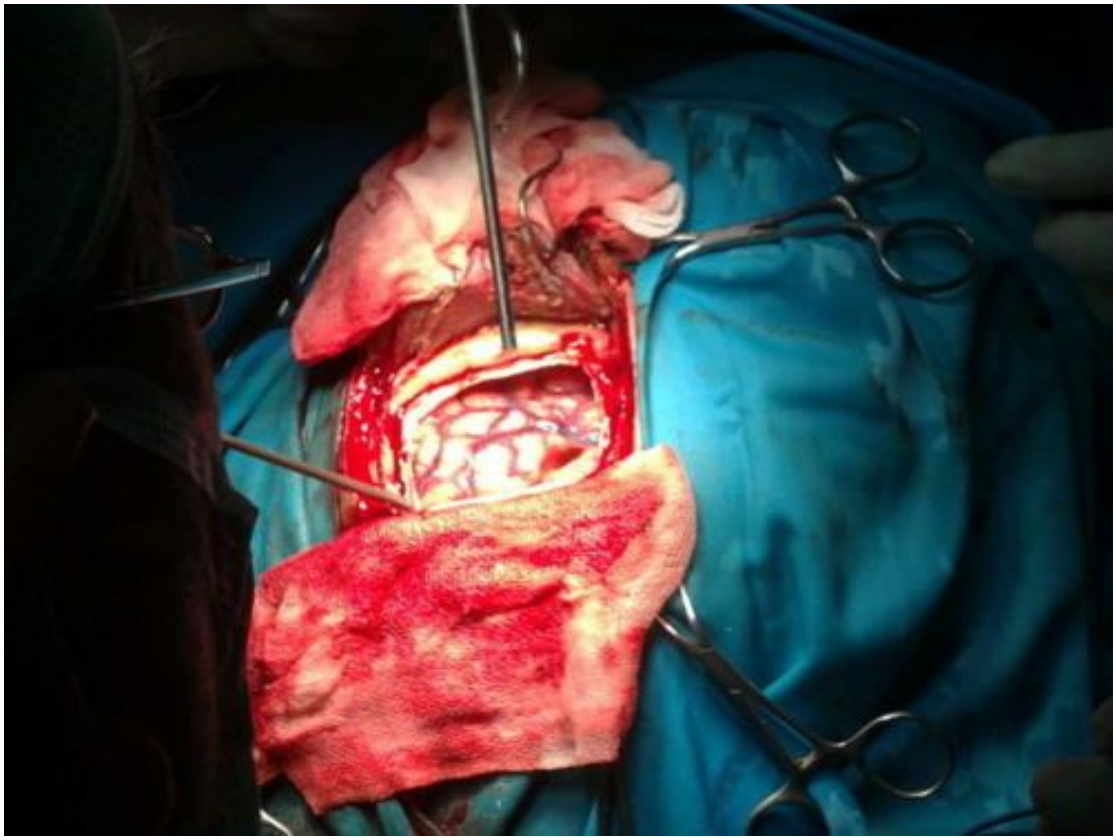


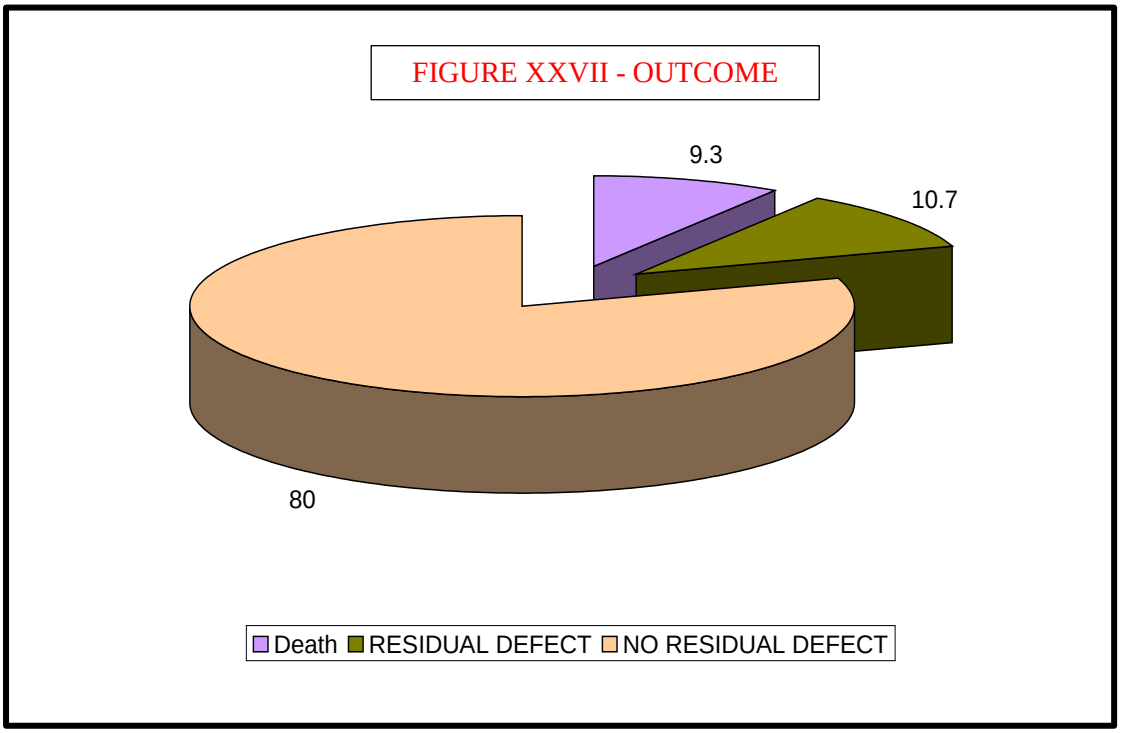
FIGURE XXVI - POST – OPERATIVE PICTURE

OUTCOME :

Table XVI

Outcome	No.of cases	Percentage
Death	7	9.3
RESIDUAL DEFECT	8	10.7
NO RESIDUAL DEFECT	60	80.0
Total	75	100.0

We observed that 80% of patients showed good prognosis in the form of recovery without residual problem. Prognosis was good in patients with Venous infarct. We found that 10.7% of patients had recovered with residual deficit. The mortality was 9.30%.



DISCUSSION

The event of Cerebro Vascular accident in puerperal period is challenging both in diagnosis as well as in management too. They are at increased risk of thrombo embolic disease since pregnancy and puerperium are hypercoagulable state[46]. Nearly 50% of young stroke in women is related to pregnancy and puerperium[47].

Cerebral Venous thrombosis leads the cause for stroke in pregnancy and puerperium as first which has its maximum incidence in puerperium. In India Bansal et al reported puerperal CVT as 4.5/1000 obstetric admission[33].

The chance of Cerebro Vascular Thrombosis increases with immunological and haematological abnormalities, infections, pre eclampsia, dehydration and already existing hypercoagulable state. The mortality rate was 5-30% which varies with time of detection and treatment initiated[36]. As early as possible neuroimaging should be done either as CT or MRI. This study was done to find the risk factors, clinical presentation, outcome and management of CVT in puerperal period.

We had 75 patients, all diagnosed to have Cerebral venous thrombosis in a period from February 2018 to July 2019. Highest incidence of CVT was seen in 21-25 years contributing to 53.3% of total cases. This is similar to Lanska et al, a study by him mentioned 15-25 year is most vulnerable to obstetric stroke[37].25.3% of cases belonged to 26-30 year group. Youngest age patient was 16 year old, a case of anemia with severe pre eclampsia.

Among 75 patients maximum incidence was noted in patients who did their primary school. Maximum occurrence of CVT was noted in Class V socio-economic class which was 81.3%, followed by class IV 14.7%.This might be because most of

patients admitted to our hospital belong to class V apart from risk factors like anemia, gestational hypertension, poor nutrition ,lack of awareness. Prakash BC and Bansal C said that it has to be evaluated further for their higher incidence in lower socio economic class[33].

Among 75 patients 86.7% of patients are from rural area which may be due to late identification of risk factors, poor awareness and non accessibility of heath care system. Wrong beliefs like prolonged bed rest, post partum water restriction, poor nutrition may be the cause for highest incidence in rural areas. Among 75 mothers the maximum incidence was noted in primipara which is 60.0%, followed by second gravida contributing to 24%.

In this study 52% of CVT occurred in 2ndweek of postpartum. Prakash BC and Bansal C reported that postpartum CVT usually occurred 7-10 days after delivery[33] which almost similar to our study.

Post partum CVT was most common among LSCS patients contributing to 52%. The sole responsible factor for obstetric CVT was caeserean section, increasing the risk to 3 fold[48]. Also caesarean section will increase the risk due to post surgical decline of protein C level due to surgically induced tissue damage[15]. There may be also prolonged immobilisation causing venous stasis.

CVT is a multifactorial disorder proving that when two or more risk factors exists, the chance of CVT is high. In our study we found Anemia (64%), LSCS(52%), Hypertensive disorders (48%), Blood transfusion (42.2%), Dehydration (34.67%) are the most important predisposing factor for CVT. Brown et al showed women with pre eclampsia were 60% more likely to have ischemic stroke than women without it[49].

Independent risk factors for CVT are postpartum period, caesarean delivery, anemia, maternal hypertension[48.] Headache was the most commonest symptom preceding this neurological event. In our study 86.67% of patients are preceded by headache which is similar to study by Kumar et al with 66%, Nagaraj et al showing 71%[76]. Convulsions, altered sensorium are the other two common symptoms with 84% , 69.3%.

This is to conclude that patients with severe, constant headache in postpartum period should be evaluated even if there is no neurological deficit.

84% of patients had seizure which is either focal or generalized seizures which is similar to study by Kumar S et al. Early appearance of seizure might be hallmark of bad prognosis. 53.3% had GTCS and 30.7% had focal seizures. In our study 22.67% of patients had fever at the onset of CVT. Fever and infection may predispose to CVT as the fever develops in patients in those with deep cerebral veins as their pons is involved[36].One patient of brainstem infarct had persistent hyperpyrexia.

Among 75 patients studied 45.33% had visual disturbance like blurring of vision, diplopia, floaters or transient loss . Some cases of diplopia is due to raised ICT compressing 6th cranial nerve. Posterior reversible encephalopathy syndrome causes transient loss of vision. In our study 48% of postpartum CVT patients observed to have hypertensive disorders. Among them, 25.3% had BP $\geq$ 140/90mmHg and $\leq$160/110mmHg. Study by James et al found only 20% of patients of CVT related to pre eclamptic toxemia exhibit sustained diastolic pressure of >105mmHg before the stroke develops[14].

Anemia is the important predisposing factor for postpartum CVT. In our study 64% of patients were anemic. Most of them are severely anemic, almost all of them

had Iron sucrose infusion either in antenatal period or postpartum. These patients are more prone to infection explaining its increased incidence.

Radiological investigation was done in all patients. Study by Srinivasan K showed 50 cases of CVT among 10,000 deliveries. Developing countries have increased prevalence, 10 times more than in developed countries. 58.67 % had venous infarct, 24% had haemorrhagic infarct, 8% had empty delta sign and mass effect was observed in 5.33% of patients. In Cerebral Venous Thrombosis we observed superior sagittal sinus (65.33%) was most commonly involved. This was followed by Transverse sinus with 24%.It was reported similarly in Ameri et al(72%) and Strolz et al (72.2%).

All patients of suspected CVT was received in Intensive care unit or High dependency unit and a multidisciplinary treatment was initiated involving Obstetrician, Neurophysician, Neurosurgeon, Radiologist and Physiotherapist. Unfractioned Heparin 5000 units was given intravenously, thrice daily for patients with CVT. Anticonvulsants, anti hypertensives and anti edema measures was given in appropriate manner. Coagulation profile was done periodically.

Decompressive Craniotomy was done in 3 patients and 2 recovered well. Patients on ventilator or bed ridden are monitored carefully with measures to prevent aspiration, bed sores, exposure keratitis and infections. The risk factors are corrected accordingly like anaemia with blood transfusion, dehydrated patients with Intravenous fluids. After the acute phase patients are stepped down, shifted to ward and their anti coagulation was maintained with T.Warfarin of INR 1.5 to 2.

In our study 80% of patients recovered well with no neurological deficit & 10.7% of patients had neurological deficit like hemiparesis. Patients with cerebral

venous thrombosis have good outcome & the mortality rate was 9.33%.

There was 48 maternal death in Government Mohan Kumaramangalam Medical College during the study period out of which 7 (9.33%) have occurred due to cerebral venous thrombosis. All these patients had haemorrhagic infarct. Treadwell SD et al and Dash et al[59] observed haemorrhagic infarct and empty delta sign as the poor prognosis. In this present study we found that higher maternal age, Hypertensive disorders, Haemorrhagic infarct and Intra cranial haemorrhage as poor prognostic factors. When deaths occur, they usually result from Trans tentorial herniation or intracranial haemorrhage.

CONCLUSION

Cerebral venous thrombosis is one of the most serious complication of puerperium and it is a recognized rising cause for maternal mortality. It has a wide spectrum of clinical presentation.

CVT is the commonest cause for post partum stroke. Though hypercoagulable state of pregnancy creates the risk, it is possible to prevent CVA by timely identification and correction of risk factors. Important risk factors are anemia, preeclampsia, infection, operative procedures and dehydration.

According to the study

- Highest incidence of Cerebral Venous Thrombosis was observed in age group 20- 25years.
- Maximum number of CVT occured in Primiparas.
- 95% of Cerebral Venous Thrombosis occured in lower socio economic group (class V and class IV).
- 86.7% of patients belong to rural area.
- Incidence of postpartum Cerebral venous thrombosis was highest in 2nd week of postpartum with 52%.
- 52% of patients with Cerebro vascular accident had undergone Lower segment caesarean section.
- The commonest risk factor anaemia was noted in 64% of study population and the second commonest hypertensive disorders of pregancy was noted as 48%.
- Headache was the most commonest symptom followed by convulsions.

- Altered sensorium was noted in 62.2% of study population.
- Generalized tonic clonic seizure were more common.
- In postpartum cerebral venous thrombosis Superior sagital sinus was most commonly involved.

Hence people must be made aware of the risk factors and early symptoms of cerebral venous thrombosis. Risk factors like anemia, pre eclampsia, infection and dehydration should be identified at the level of Primary health care systems itself.

Identification of risk factors and recommendations for thrombo prophylaxis should be considered. Postpartum headache deserves prompt and focused evaluation. Early diagnosis and early initiation of treatment reduces the mortality of CVT in young female.

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ANNEXURES

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Communication of Decision of the Institutional Ethics Committee(IEC)

Ref. No. GMKMC&H/4341/IEC/01/2017-21 **Date: 25.01.2018**

Protocol title	**"CLINICAL PROFILE OF POSTPARTUM CEREBRAL VENOUS THROMBOSIS AND ITS MANAGEMENT"**
Guide/Principal Investigator	DR.B. JEYAMANI, MD.,DGO, Associate Professor and HOD, Department of O&G, GMKMC, Salem-30.
Student	Dr.U. KALAIYARASI, 1ST Year, Post Graduate Student of MS O&G, GMKMC, Salem
Name & Address of Institution	Govt. Mohan Kumaramangalam Medical College & Hospital, Salem, Tamil Nadu.
Type of Review	☑ New review ☐ Revised review ☐ Expedited review
Date of review (D/M/Y)	20.01.2018
Date of previous review, if revised application:	Nil
Decision of the IEC	☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions/ Reasons/ Remarks:	Nil
Recommended for a period of :	Feb 2018 to July 2019

Please note *

- Inform IEC immediately in case of any Adverse events and Serious adverse events.
- Inform IEC in case of any change of study procedure, site and investigator
- This permission is only for period mentioned above. Annual report to be submitted to IEC.
- Members of IEC have right to monitor the trial with prior intimation.

R. Vidhyalaxmi
for Signature of Member Secretary

DEAN
Govt. Mohan Kumaramangalam
Medical College, SALEM-636 030.

URKUND

Urkund Analysis Result

Analysed Document:	Dr. Kalaiyarasi for plagiarism.doc (D57220795)
Submitted:	10/18/2019 10:18:00 AM
Submitted By:	kalainehaa22@gmail.com
Significance:	16 %

Sources included in the report:

thesis.pdf (D42231904)
Thesis ajey jha.docx (D55726516)
https://www.bhj.org.in/journal/special_issue_bhims_2011/download/414-419.pdf

Instances where selected sources appear:

14

PROFORMA

POSTPARTUM CEREBRAL VENOUS THROMBOSIS

HISTORY :

Name : Age :

IP No. : Address :

Contact number:

Socio economic status :

Time of presentation :

Postnatal day:

Mode of delivery :

Co- morbidity:

Symptoms:

Headache -

Nausea / Vomiting Altered sensorium-

Fever -

Convulsions - Neurological deficit - Visual disturbance-

Past History :

HT / PIH / DM / Heart disease / Seizure disorder / TIA / Bleeding

diathesis / Migraine / Repeated abortions / OCP intake/ Auto immune disorders

Personal History :

Smoking / Alcohol and other substance abuse

EXAMINATION :

General Examination :

Pallor / Jaundice / Pedal edema / Cyanosis / Clubbing /

Lymphadenopathy Evidence of DVT

Blood pressure -

Pulse Rate - Temperature - Respiratory Rate and Type - CVS -

RS –

P/A –

P/V -

Neurological Examination : Level of consciousness - Orientation -

Cranial Nerves - Motor System – Sensory System -

Cerebellar Function - Fundus Examination - Neck stiffness-

INVESTIGATIONS:

Complete blood count BT and CT

Urinalysis - Blood Sugar - Blood Urea -

Serum Creatinine – Liver function test- Serum Electrolytes - Blood group and Type- Chest X-ray -

ECG -

HIV and VDR L -

Radiological investigations: CT Brain /MRI/ MRA & MRV Other Investigations –

DIAGNOSIS:

TREATMENT:

OUTCOME:

LIST OFABREVIATIONS FOR MASTER CHART

AI-Arterial infarct AF-Afebrile

AN-Anemia AP-Antepartum

AS-Altered sensorium BT-Blood transfusion C-Convulsion

CHT-Chronic hypertension CS-Cord sign

CVT-Cerebral venous thrombosis D-Death

DH-Dehydration E-Edema

Ed-Empty Delta sign F-Febrile

FOCAL-Focal seizures

GTCS-Generalized Tonic Clonic Seizures H-Headache

HI-Heamorrhagic infarct

ICH-Intra cranial haemorrhage JS-Jugular sinus

LN-Labour naturalis ME-Mass effect

N-Normal

NA-Not applicable

ND-Neurological deficit NS-Neck stiffness

P-Papilloedema PE- Pre eclampsia PP-Postpartum

PPH-Postpartum haemorrhage

PRES-Posterior reversible encephalopathy R-Rural

RF-Renal failure

RND-Recovered, No deficit RD-Recovered with deficit SG-Sigmoid sinus

SLE-Systemic lupus erythematosus SS-Superior sagittal sinus

ST-Straight sinus

TA-Takayasu arteritis U-Urban

V-Vomiting

VD-Visual defect VI-Venous infarct

PATIENT CONSENT FORM

STUDY TITLE: "CLINICAL PROFILE OF POSTPARTUM CEREBRAL VENOUS THROMBOSIS AND ITS MANAGEMENT"

DEPARTMENT OF OBSTETRICS AND GYNECOLOGY, GMKMCH SALEM

PARTICIPANT NAME: AGE: SEX:

I.P. NO:

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I have been explained about the possible complications that may occur during the period of study. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study.

Time: Patient name;

Date: Signature / Thumb Impression of Patient:

Place Name and signature of the Investigator

ஆராய்ச்சி ஒப்புதல் படிவம்

பெயர் : தேதி :

வயது : உள்நோயாளி எண் :

பாலினம்: ஆய்வு சேர்க்கை எண் :

இந்த ஆய்வின் நோக்கம் மற்றும் விவரங்கள் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது. இவ்வாய்வில் இருந்து நான் எந்த நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எனக்கு எந்த பாதிப்பும் இல்லை என்பதையும் தெளிவாக புரிந்து கொண்டேன்.

முடிவுகளை அல்லது கருத்துகளை வெளியிடும் போதோ அல்லது ஆய்வின் போதோ என்னுடைய பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டார்கள் என்பதையும் அறிந்து கொண்டேன்.

இந்த ஆய்வில் எவ்வித நிர்பந்தமும் இன்றி எனது சொந்த விருப்பத்தின் பேரில் நான் பங்கு பெறுகின்றேன்.

நான் சுயநினைவுடனும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் சேர்த்துக்கொள்ள சம்மதிக்கின்றேன்.

ஆராய்சியாளர் ஒப்பம் பங்கேற்பாளர் ஒப்பம்

இடது

பெருவிரல் ரேகை

MASTER CHART

S.NO	NAME	AGE	IP NO	EDUCATION	SOCIAL ECONOMIC STATUS	AREA	PARITY	DURATION	MODE OF DELIVERY	CO MORBIDITY	SEIZURES	SYMPTOMS	BP	RADIOLOGICAL FINDING	SINUS INVOLVED	MANAGEMENT	OUT COME
1	Sangeetha	29	17252	GR	IV	R	P2L2	1 WK	LSCS	AN/HY	Focal	H/C/AS	130/90	VI/CVT	SSS/TS/SS	HE/AC	RD
2	Usha	22	30451	HS	IV	R	P1L1	2 WK	LN	AN/OB	Focal	C/AS/ND	130/90	VI/CVT	SS	HI/E	NRD
3	Ambika	25	29875	PR	V	R	P1L1	1 WK	LN	AN	GTCS	H/C/BV	110/70	VI/CVT	SS/TS	HE/AC	NRD
4	Rekha	23	26183	IL	V	R	P1L1	2 WK	LN	NA	GTCS	H/C/ND	130/80	VI/CVT	SS/SG	HE/AC	RD
5	Muniammal	22	25612	IL	V	R	P1L1	2 WK	LN	DH	NA	H/V/AS	120/90	HI	TS	HE/AC	NRD
6	Reshmidass	24	43906	GR	IV	U	P1L1	1 WK	LN	AN/SPE/F	GTCS	H/AS/C/ND	110/80	HI/ME/E	SS/TS/ST/TS/DCV	AE/HE	DEATH
7	Kamali	22	35431	HS	V	R	P2L2	3 WK	LSCS	NA	Focal	C/AS	110/70	ED/E	SS/SG	HE/AC	NRD
8	Yogapriya	19	9759	PR	V	R	P1L1	2 WK	LN	DH/GHTn	GTCS	H/V/C	160/100	HI	TS/LS	HE/AC	NRD
9	Gayathri	19	15741	PR	V	RR	P2L2	1 WK	LN	AN/DH	GTCS	H/C/AS/V	110/70	VI/CVT	SS	HE/AC	NRD
10	Chitra	24	45868	PR	V	R	P2L2	2 WK	LSCS	AN/OB	Focal	H/C/ND	130/80	VI/CVT	SS,JS	HE/AC	NRD
11	Indumathi	27	46437	PR	V	R	P2L2	1 WK	LN	AN/HY	NA	H/C/FD	110/780	VI/CVT	TS	HE/AC	NRD
12	Kayalvizhi	29	69342	GR	III	U	P1L1	2 WK	LSCS	AN/F	GTCS	H/C/V/AS	120/80	HI/CVT	HI/ME/MS	CRANIOTOMY DONE	RD
13	Thulasimari	20	68137	IL	V	R	P2L2	3 WK	LSCS	AN/DH	GTCS	V/AS	120/70	VI/CVT	SS/TS	HE/AC	NRD
14	Manjula	21	61808	IL	V	R	P2L2	1 WK	LSCS	GDM	Focal	H/C/FD/V	120/80	HI	SS/SG	HE/AC	NRD
15	Madhammal	23	82541	IL	V	R	P1L1	2 WK	LN	AN/GHTn	GTCS	C/AS/ND	140/100	VI/CVT	TS	HE/AC	NRD
16	Archanamary	22	73708	HS	IV	U	P1L1	1 WK	LSCS	NA	Focal	C/ND	110/80	HI	HI/ME	HE/AC	NRD
17	Dhanalakshmi	21	80224	PR	V	R	P1L1	1 WK	LSCS	AN/BT	GTCS	H/AS	120/70	VI/CVT	SS/SG	HE/AC	NRD
18	Manimegalai	26	71624	HS	V	R	P1L1	2 WK	LSCS	AN/GDM	Focal	V/H/BV	120/780	VI/CVT	SS	HE/AC	NRD
19	Vennila	21	86455	PR	V	R	P1L1	2 WK	LSCS	AN/BT	NA	H/AS	120/80	VI/CVT	LS	HE/AC	NRD
20	Anurani	25	92416	IL	V	R	P2L1	2 WK	INST	OB/HY	GTCS	H/C/F/AS	150/90	VI/CVT	SS/SG/LS	HE/AC	NRD
21	Sandhiya	23	46437	PR	V	R	P1L1	1 WK	LSCS	AN/BT	Focal	H/C/ND	120/70	VI/CVT	TS	HE/AC	NRD
22	Selvi	25	47160	PR	V	R	P1L1	2 WK	LSCS	DH	GTCS	H/AS/F	100/70	VI/CVT	SS	HE/AC	NRD
23	Vanitha	36	24929	IL	V	U	P3L3	3 WK	LSCS	AN/PPH	GTCS	C/AS/F/ND	110/780	VI/CVT	SS/JS	HE/AC	NRD
24	Chandra	28	20905	PR	V	R	P2L2	1 WK	LN	NA/DH	GTCS	H/C/V	120/80	VI/CVT	SS	HE/AC	NRD
25	Meera	32	21832	HS	IV	U	P2L3	2 WK	LN	Twin/PE	Focal	ND/NS	150/90	VI/CVT	SS	HE/AC	NRD
26	Kaliyammal	24	43442	PR	V	R	P2L2	1 WK	LSCS	AN/BT	NA	H/V/BV	120/80	VI/CVT	TS	HE/AC	NRD

27	Amudha	22	57801	HS	V	R	P1L1	3 WK	LN	AN/GHTn	NA	AS/V/F	140/90	HI	SS/ST/SG/TS/LS	HE/IVF	DEATH
28	Maheshwari	16	54312	HS	V	R	P1L1	2 WK	LN	NA	GTCS	H/C/BV	100/70	HI/ME	TS/JS/LS	HE/IVF	DEATH
29	Arulmozhi	21	64987	HS	V	U	P1L1	1 WK	LSCS	AN/OB	GTCS	ND/NS	130/80	VI/CVT	TS	HE/AC	NRD
30	Valarmathi	32	45613	IL	V	R	P2L2	2 WK	LN	AN/HY/BT	GTCS	H/C/AS	120/70	VI/CVT	SS	HE/AC	NRD
31	Arumugakani	35	24609	IL	V	R	PILI	2 WK	LSCS	AN/BT	GTCS	H/C/NS/AS	120/80	HI/CVT	TS/SG	HE/AC	NRD
32	Manonmani	32	30071	PR	V	R	P3L3	2 WK	LN	AN/SPE/BT	Focal	H/C/F/FV	150/90	ED/E	TS	HE/AC	NRD
33	kavitha	25	39897	PR	V	R	PILI	2 WK	LN	NA	GTCS	C/AS/ND	120/80	HI/E	LS/SS	HE/AC	NRD
34	Subbulakshmi	29	59771	PR	V	U	P2L2	1 WK	LSCS	DH/AN/IS	Focal	H/AS	140/90	VI/CVT	SS	HE/AC	NRD
35	Valli	28	26077	HS	V	R	P32L3	2 WK	LSCS	AN/BT	GTCS	H/C/AS	100/70	VI/CVT	SS/SG	HE/AC	NRD
36	Kannilakshmi	25	25438	HS	V	R	PILI	2 WK	LSCS	PE/GDM	Focal	H/V	130/80	HI/CVT	SS/SG/LS	HE/AC	NRD
37	Jayapriya	24	21254	PR	V	U	P2L2	3 WK	LSCS	PE/DH	GTCS	C/AS/ND/NS	140/90	HI/ME/E	SS/ST/TS	HE/AC	DEATH
38	Deviga	26	18108	GR	V	R	PILI	2 WK	LN	ANHY/OB	GTCS	H/C/F/	100/70	ED/E	SS/SG	HE/AC	NRD
39	Mumtaj	28	21254	HS	V	R	P3L3	2 WK	LN	AN/BT	Focal	H/C/BV/UC	130/80	HI/ME/E	SS/SG/ST	HE/AC	RD
40	Saalammal	21	16745	IL	V	R	PILI	3 WK	LSCS	AN	NA	ND/NS	110/70	VI/CVT	SS/TS/ST/SG/DCV	CRANIOTOMY	NRD
41	Lakshmi	22	28914	GR	IV	R	PILI	1 WK	LSCS	AN/OB	GTCS	H/C/AS	120/20	HI/CVT	SS	HE/AC	NRD
42	Mythili	24	55013	PR	V	R	P1L1A2	2 WK	LN	SPE	Focal	H/C/NS/AS	150/100	VI/CVT	TS	HE/AC	NRD
43	Kaaviya	23	27653	HS	V	U	PILI	1 WK	LN	DH	GTCS	H/C/F/BV	150/90	VI/CVT	SS/TS/SG	HE/AC	NRD
44	Jeeva	22	48220	PR	V	R	PILI	2 WK	LN	AN/BT/DH	GTCS	C/AS/ND	140/90	HI/ME/MS	SS/LS	HE/AC	DEATH
45	Rajeshwrai	25	90312	GR	III	R	P1L1	2 WK	LSCS	AN/GDM	NA	H/AS	120/80	VI/CVT	SS/SG	HE/AC	NRD
46	Palaniammal	31	48472	IL	V	R	P4L4	2 WK	LN	AN/BT	Focal	H/C/AS	120/70	VI/CVT	TS	HE/AC	NRD
47	Thangam	23	80362	PR	V	R	P1L1	2 WK	LN	AN/IS	GTCS	H/C/AS/V	140/90	VI/CVT	SG	HE/AC	NRD
48	Vijiyarani	24	13296	PR	V	R	P1L1	1 WK	LN	AN	Focal	H/AS/F	120/80	VI/CVT	TS/JS	HE/AC	RD
49	Deepa	22	15038	HS	V	R	PILI	2 WK	LSCS	AN/HY	NA	H/C/AS	110/70	VI/CVT	SS	HE/AC	NRD
50	Meenal	33	23101	IL	V	R	P3L3	1 WK	LSCS	AN/IS	GTCS	H/C/AS/ND	150/90	VI/CVT	SS/SG	HE/AC	NRD
51	Suganya	21	35871	PR	V	R	P1L1	1 WK	LN	AN/BT/GHTn	GTCS	H/C/BV	140/90	VI/CVT	SS/SG	HE/AC	NRD
52	Priya	27	34281	PR	V	R	P1L1	2 WK	LSCS	PE/DH	GTCS	H/C/ND	120/80	VI/CVT	SS	HE/AC	NRD
53	Deepa	23	59231	HS	V	R	P1L2	1 WK	LSCS	AN/HY/OB	NA	H/V	110/80	VI/CVT	SS/SG	HE/AC	NRD
54	Suganthi	24	42851	IL	IV	R	P3L2	1 WK	LN	PE/TWIN	GTCS	H/BV	140/100	HI/CVT	TS	HE/AC	NRD
55	Padma	26	3914	GR	IV	R	P2L1	2 WK	LN	AN/BT	Focal	H/BV/ML	110/80	VI/CVT	TS	HE/AC	NRD
56	Vijiya	29	43593	PR	V	R	P1L1	1 WK	LSCS	AN/DH	NA	ND	120/80	VI/CVT	LS	HE/AC	NRD
57	Santhamani	32	49041	IL	V	R	P3L3	2 WK	LN	AN/BT	GTCS	H/C/AS	120/80	VI/CVT	SS/TS	HE/AC	NRD
58	Geetha	29	40063	HS	III	R	P4L4	3 WK	LSCS	OB/HY/DH	NA	H/BV/V	140/100	HI/CVT	SS/SG	HE/AC	NRD
59	Poomathy	27	36845	IL	V	R	P2L2	2 WK	LN	AN/IS	GTCS	H/C	140/90	VI/CVT	SS/TS	HE/AC	NRD

60	Sahana	31	94657	PR	V	R	P1L1	1 WK	LSCS	AN/GHTn	Focal	ML	130/80	VI/CVT	SS	HE/AC	NRD
61	Munirathinam	26	32313	IL	V	R	P4L4	2 WK	LN	AN	GTCS	H/AS	100/70	HI/CVT	SS/TS/ST/SG/DCV/ LS	HE/IVF	DEATH
62	Tamilselvi	24	51090	PR	V	R	P1L1	1 WK	LSCS	AN/BT	Focal	H/C/AS	110/70	VI/CVT	TS	HE/AC	NRD
63	Pavithra	22	43772	PR	V	R	P1L1	1 WK	LSCS	DH	NA	H/C/AS/V	120/70	VI/CVT	LS/SS	HE/AC	NRD
64	Latha	24	3671	HS	V	R	P1L1	2 WK	LN	AN/IS	GTCS	H/AS/F	130/80	VI/CVT	SS/SG	HE/AC	RD
65	Revathi	25	31887	PR	IV	R	P1L1	1 WK	INTL	AN/SPE/BT	Focal	H/C/AS	150/100	VI/CVT	SS/TS	HE/AC	NRD
66	Ramya	26	59242	PR	V	R	P1L1	2 WK	LSCS	SPE	GTCS	H/C/AS/ND	160/110	VI/CVT	SS/TS/SG	HE/AC	NRD
67	Senbagam	27	21443	HS	IV	R	P3L3	2 WK	LN	OB/HY	GTCS	H/C/BV	140/100	VI/CVT	SS	HE/AC	RD
68	Fathima	18	91343	PR	V	R	P1L1	1 WK	LSCS	AN/DH	Focal	H/C/ND	100/70	VI/CVT	SS/SG/ST	HE/AC	NRD
69	Durga	21	17342	PR	V	R	P2L2	1 WK	LSCS	AN/GHTn	GTCS	H/V	150/90	VI/CVT	TS/LS	HE/AC	NRD
70	Manimegalai	19	9108	HS	V	R	P1L1	2 WK	LSCS	SPE/GDM	Focal	H/BV	150/90	VI/CVT	SS/LS	HE/AC	NRD
71	Ramalakshmi	19	11650	PR	V	R	P1L1	1 WK	LSCS	PE/DH	GTCS	H/BV/ML	160/100	VI/CVT	SG/SS/TS/LS/DCV	HE/AC	DEATH
72	Senbagam	23	71790	PR	V	R	P2L2	2 WK	LSCS	AN/HY/OB	Focal	V/C	120/80	VI/CVT	SS/TS/SG	HE/AC	NRD
73	Banu	21	72785	PR	V	R	P1L1	2 WK	LN	AN/BT	GTCS	BV/ML	160/90	VI/CVT	SS/LS	HE/AC	NRD
74	Santha	28	91702	IL	V	R	P3L2	3 WK	LN	NA	GTCS	H/BV	160/110	VI/CVT	TS/JS	HE/AC	RD
75	Saritha	28	87324	GR	IV	U	PILI	2 WK	LSCS	OB/SPE/GDM	GTCS	H/C/V/AS	140/100	VI/CV	SS/SG	HE/AC	NRD