DEMOGRAPHIC PROFILE AND EVALUATION OF CEREBRO VASCULAR ACCIDENTS IN PREGNANCY AND PUERPERIUM

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M.D. OBSTETRICS AND GYNAECOLOGY

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CERTIFICATE

This is to certify that the dissertation entitled, **‘‘DEMOGRAPHIC PROFILE AND EVALUATION OF CEREBROVASCULAR ACCIDENTS IN PREGNANCYAND PUERPERIUM”** by **Dr.K.Vanithasri**, Post graduate in Obstetrics and Gynaecology (2010-2013), is a bonafide record of work carried out under our supervision and guidance in Department of Obstetrics and Gynaecology and is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai, for M.D. Degree Examination in Obstetrics and Gynaecology, Branch II, to be held in March 2013.

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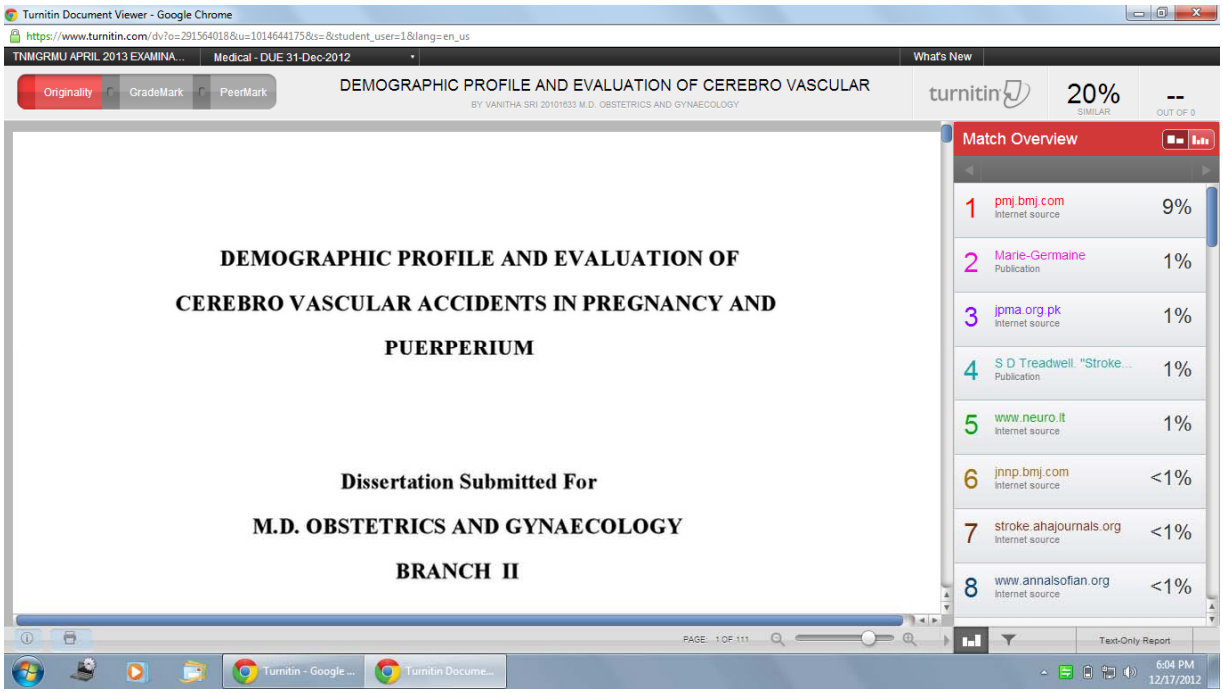
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I solemnly declare that this dissertation entitled **‘‘DEMOGRAPHIC PROFILE AND EVALUATION OF CEREBROVASCULAR ACCIDENTS IN PREGNANCYAND PUERPERIUM”** is a bonafide record of work done by me in the Department of Obstetrics and Gynaecology at Tirunelveli Medical College Hospital from 2010 to 2013 under the guidance and supervision of PROF. DR. J.SARALA M.D. D.G.O., This dissertation is submitted to Tamil Nadu Dr.M.G.R. Medical University in partial fulfillment of the University regulations for the award of M.D. (BRANCH – II) Obstetrics and Gynaecology degree examination to be held in April 2013.

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CONTENTS

DEMOGRAPHIC PROFILE AND EVALUATION OF CEREBROVASCULAR ACCIDENTS IN PREGNANCY AND PUERPERIUM

1. INTRODUCTION

Stroke in younger age groups is more common in females than males.The outcome is poor in females in terms of morbidity and mortality[1]. There have been many studies analyzing the role of female endocrine physiology in CVA. Pregnancy, hormonal contraceptives, and hormone replacement therapy are some of the female specific risk factors which have been analyzed by many authors[2]. Because of it s physiological prothrombotic state pregnancy and puerperium are known risk factors for Thrombo embolic disorders[3]. CVA during pregnancy and puerperium are recognized and feared complications as they account for around 12% of all maternal mortality. Since the direct causes for maternal mortality has been brought down, there is growing interest on the non-obstetric causes like stroke.[4]

Pregnancy related stroke has a wide spectrum of clinical presentation which makes it as a diagnostic and therapeutic challenge to the treating obstetrician. Development in the field of Neuroimaging helps us for early diagnosis and changed our approach towards this disorder. Here we discuss the relevant areas unique to pregnancy including the role of investigations, management and outcome.

AIM OF THE STUDY

To study the demographic profile, predisposing factors, spectrum of clinical presentation, management and prognosis of Cerebro Vascular Accidents, which occur during pregnancy and puerperium.

REVIEW OF LITERATURE

It has been estimated that more than 12% of maternal mortality occurs due to Cerebro Vascular Accidents. The morbidity in the survivors may be severe enough to cause permanent disability.[4] During pregnancy and puerperium women have a hypercoagulable state which explains the higher incidence of CVA 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.[5] The risk of CVA increases along with increasing maternal age particularly after 35 years.[6] In a study by James *et al*, few medical conditions were found to have strong association with CVA when the affected woman becomes pregnant. They included migraine, inherited or acquired thrombophilias, connective tissue disorders, cardiac disease, sickle cell disease, hypertension, anemia and thrombocytopenia as risk factors.[6] Conditions peculiar to pregnancy being significant risk factors were anaemia, postpartum haemorrhage, Hypertensive disorders of pregnancy, transfusion and postpartum infection.[1]

3.1.INCIDENCE

Since CVA related to pregnancy and 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 CVA due to ischaemia/infarction was 11 per100 000 deliveries with the puerperal period carrying the maximum risk. They also reported that the risk of haemorrhagic stroke was 9 per100 000 deliveries, again with the maximum occurrence in the puerperal period.[7] Large population based studies are required to establish the incidence exactly, and reported incidence rates vary considerably.

The incidence of stroke in women of reproductive age group, who were not pregnant was reported to be 10.7/100000 women-years.[8] Incidence of pregnancy-related stroke was calculated per 100 000 deliveries. It varied from 4.3–210 cases per 100 000 deliveries.[5,9] A recent population study in the USA analyzed the inpatient data from, 1000 hospitals. Through this study 2850 cases of CVA in pregnancy and puerperium were identified. This study reported the incidence of CVA related to pregnancy as 34.2 per 100 000 deliveries and women have 3 fold increased risk of CVA during pregnancy than non pregnant women.[6] Wiebers and Whisnant reported 13-fold increased risk of stroke in pregnant females during the course of pregnancy and puerperium.[10]

Previously it was thought that CVA in pregnancy and puerperium are secondary to thrombosis of cerebral veins only. A data analysis from Mexico showed that more than 50 percent of total cases of venous thrombosis were

pregnancy related.[11] Though pregnancy shows increased risk for venous thrombosis, most of the cerebral infarctions are secondary to occlusion of arteries. Many studies have reported CVA due to ischaemia and haemorrhage in more or less similar proportions.[12,13] But Jaigobin and Silver have reported the incidence of ischaemic stroke as 18 / 100 000 deliveries and haemorrhagic stroke as 8/ 100 000 deliveries.[11] In India, prevalence of cerebral vein thrombosis is 4.5/ 1000 obstetric admissions. In our country CVT in the early puerperal period is 10 times higher than the Western countries.[14]

Pregnancy associated CVA, particularly venous infarctions shows maximum incidence in the third trimester and the post partum period. Jaigobin and Silver analyzed 8 women with venous infarctions, 7 of which occurred in puerperal period.[11] A study on pregnancy associated thrombo embolic disorders was conducted in Taiwan and showed that 73% of CVT occurred in the post partum period.[15] A study from Sweden has shown that the risk of haemorrhagic and ischemic stroke is maximum during 2 days before and 1 day after delivery.[8] Data obtained through a population study from USA showed that around 90% CVA related to pregnancy occurred either during delivery or puerperium.[1] Lanska *et al* also have published a similar report.[16] The mortality due to CVA in pregnancy and puerperium has been estimated to be 10-13%.[6,17]

3.2. ETIOLOGY

3.2.1 PREDISPOSING CHANGES THAT OCCUR IN PREGNANCY

❖ **Hypercoagulability**

The following physiological changes take place during pregnancy leading to a hypercoagulable state

- Raised level of factor VIII
- Raised level of von Willebrand factor
- Raised level of fibrinogen
- Resistance to the action of Protein C
- Decreased level of protein S
- Plasminogen activator inhibitors (PAI) 1 and 2 levels increased
- Hyperprolactinaemia mediated platelet aggregation

❖ **Venous stasis**

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

- Pelvic vessels are compressed by the enlarged uterus
- Restricted mobility during pregnancy and puerperium

❖ **Endothelial injury**

- Vascular injury that occurs during parturition

❖ Virchow has proposed a triad which includes Hypercoagulability, Venous stasis and Endothelial injury predisposing to venous thromboembolism[18]

- ❖ Extensive anatomical, physiological and biochemical changes take place throughout pregnancy which involves all important organs.
- ❖ During pregnancy and puerperium coagulation and fibrinolytic systems are subjected to major changes and a lead to a prothrombotic state. It is a physiological preparation for delivery. These alterations are responsible for the pathogenesis of complications that occur during pregnancy and puerperium such as venous thromboembolism.[1]
- ❖ Most of the procoagulant factors are at increased concentrations during conception especially von Willebrand factor, factor VIII and fibrinogen. Factor V level starts to increase after 16 week pregnancy.[19]
- ❖ 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. [19]
- ❖ Concentrations of PAI 1 and 2 are increased during pregnancy which interferes with Fibrinolysis.[1]
- ❖ As a preparation for delivery and breast feeding, there will be a physiological increase in the concentration of prolactin during pregnancy this continues till puerperium. This physiological hyperprolactinaemia can cause platelet aggregation which is mediated through ADP stimulation.[20]

- Pregnancy related anatomical alterations like compression of great vessels by the enlarged 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.[21]

3.2.2 RISK FACTORS

Risk factors that have been recognized to cause CVA in pregnancy and puerperium are the following[6]

- High maternal age
- Black race
- Hypertensive disorders
- Cardiac disease
- Smoking
- Diabetes Mellitus
- SLE and other connective tissue disorders
- Haemoglobinopathies
- Vascular headache
- Substance abuse especially cocaine
- Caesarean section and Instrumental delivery
- Fluid and electrolyte disorders especially dehydration
- Inherited and acquired thrombophilias

- multiple pregnancy
- multi parity
- postpartum infections

- The blood loss during parturition may lead to infection and dehydration and worsens the pro coagulant condition.[1]
- Operative vaginal delivery and caesarean section increase the risk of CVA as surgically induced tissue damage causes reduction in protein C concentration which is an anti coagulant. Prolonged bed rest after operation reduces the blood flow in the lower limbs and contributes to venous stasis.[1]
- Normal pregnancy itself is not associated with endothelial injury. But during the course of vaginal or abdominal delivery, some degree of damage to pelvic vessels may take place. This increases the risk of developing venous and arterial thromboembolism.[1]
- Since atherosclerosis is not very common in younger age groups, other causes of CVA become increasingly important in these patients.

Any of these risk factors may affect the pregnancy and puerperium usually in combination. We may face difficulties in establishing whether the stroke is due to pregnancy or incidental.

3.3. STROKE SYNDROMES OF PREGNANCY

Here we will discuss the cerebro vascular events that are specific to pregnancy

3.3.1. ECLAMPSIA AND POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

- Studies have found that up to 5% of pregnancies are complicated by preeclampsia, otherwise known as Pre Eclamptic Toxemia (PET) and 1 in 200 of these women develops eclampsia.[3]
- PET is a hypertensive disorder peculiar to pregnancy and it complicates around 10% of all pregnancies.[22,23]Hypertension during pregnancy is defined as a sustained systolic BP of 140mmHg 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.[24]
- National Institute of Health Working Group has classified hypertension in pregnancy as[24]
    1. Chronic hypertension
    2. Gestational hypertension
    3. Pre eclampsia and eclampsia
    4. Pre eclampsia superimposed on eclampsia
- Pre eclampsia and eclampsia accounts for 25%-45% of pregnancy-associated Cerebro vascular accidents.[1] The development of PET is characterized by hypertension, albuminuria and edema. There is

generalised endothelial dysfunction and vasospasm involving all major organs of body.

- Pre eclampsia may also arise before 20 weeks of gestation. In case of thrombophilias, multiple pregnancy and molar pregnancy pre eclampsia may occur before 20 weeks. Sometimes it may arise in the postpartum period.[1]
- Eclampsia is defined as 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.[26]
- 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.[7] Studies have reported that patients with pre-eclampsia and eclampsia carry a greater risk for CVA. It has been observed that the most common cause of death in patients with eclampsia is intracerebral haemorrhage.[27,28]
- The risk of ischaemic stroke related to Pre eclamptic toxaemia persists even after the postpartum period.[29] 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.[30]

- 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.[6]

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.[31]
- 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 endothelial dysfunction.[31]
- In patients with pre eclampsia plasma rennin activity and angiotensin level are lower than normal. But there increased responsiveness to the pressor effect of angiotensin.[32]
- Altered immunological response in the mother and oxidative stress also have a role in the pathogenesis of PET.[24]
- 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. Isler CM *et al* from USA reported that in HELLP syndrome the usual cause for maternal mortality is Cerebro Vascular Accidents.[33]

- History of hypertension, heart disease or CVA in first degree relatives carries a higher risk for developing preeclampsia/eclampsia. It supports the rlole of possible genetic risk factors.[34]
- 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 barotrauma and damage to the vascular channels. [1]
- In patients with PET, increased vascular permeability causes third spacing 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 CVA.

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.[15]
- 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.[1]
- Important neurological problem that mimics eclampsia is cerebral venous thrombosis. But CVT usually prefers the puerperium.

Neuro Imaging

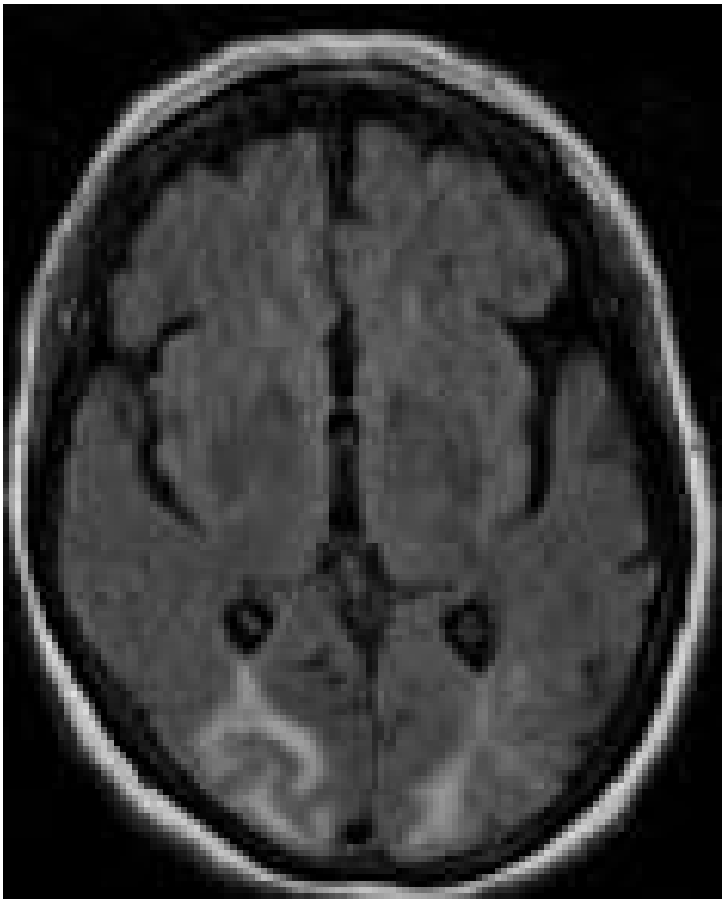
- Computed Tomography shows normal study, especially when taken in the first day of the event.
- MRI FLAIR sequences show Hyper intensity involving the occipital, parietal and lees frequently posterior frontal lobes. Cortex and subcortical parts of the above areas show diffuse abnormal signal intensities. This is due to the white matter oedema occurring in the posterior part of cerebral hemisphere. [34] Bevan H *et al* reported that partial or asymmetric hyper intensities in MRI were more common with eclampsia comparing to other causes of PRES.[35]
- Cytotoxic edema occurring due to ischaemic infarction may get confused with reversible vasogenic oedema.[23] 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.[36]

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.[29] Women with history of pre eclampsia or eclampsia should be aware of the risk factors for CVA and better to have regular follow up.

3.3.2 POSTPARTUM CEREBRAL ANGIOPATHY (PCA)

- PCA 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.[37,38,39]
- 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.[37,38]
- Common clinical features of PCA include acute onset of headache, photosensitivity, vomiting, altered sensorium, convulsions.
- Studies have reported that cerebral vasoconstriction in PCA can cause a variety of neurological deficits. This is because of transient ischaemia, cerebral infarction and cerebral haemorrhage.[40]
- Subarachnoid haemorrhage due to ruptured aneurysm, carotid or vertebral artery dissection, vasculitis of cerebral vessels, CVT, intracranial infection

and postpartum Sheehan syndrome are the important problems to be considered as differential diagnoses.[1]

Pathophysiology

The pathophysiology of PCA 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 antepartum period.[1,37]

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.[1,40]

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. [40,41]

3.3.3 ANEURYSM RUPTURE & SUB ARACHNOID HAEMORRHAGE

The third common non obstetric cause for maternal mortality is haemorrhage due to aneurysm rupture. The most common cause for Sub Arachnoid Haemorrhage is rupture of intracerebral aneurysms. The classical symptoms are thunderclap headache, vomiting, convulsions and altered sensorium.[42] When it occurs during pregnancy, it is difficult to differentiate from eclampsia. Radiological investigations and cerebrospinal fluid analysis are important to arrive at the diagnosis.

The reported incidence of cerebral haemorrhage from aneurysmal rupture during pregnancy is around 5-10/100 000 pregnancies. In a woman with cerebral aneurysm the risk of haemorrhage during pregnancy and puerperium has been reported to be 5.6 times higher.[5,7]

Aneurysms are most likely to rupture in the second half of pregnancy, and during the puerperal period. It has a greater mortality compared to general

population. Hormonal changes that occur during conception may increase the risk of aneurismal rupture.[43]

Pathophysiology

- Increased risk of aneurysmal rupture in pregnancy is attributable to the haemodynamic changes that occur during pregnancy. These changes are likely to cause aneurysm instability.[1]
- It has been reported that during pregnancy there is hyperplasia involving the smooth muscles of arteries and disruption of elastic fibre alignment. Because of these changes the vessel wall becomes weak.[1]
- Metabolic and hormonal factors related to pregnancy are also proposed as a reason for increased risk of SAH in pregnancy.
- SAH without pre existing aneurysm may occur in eclampsia

Management

Surgical management after aneurismal SAH during pregnancy improves the outcome of both mother and fetus. Usually the management is similar to that of nonpregnant patients.[1] Meyers PM *et al* have reported successful Endovascular techniques during pregnancy in the management of SAH. After successful treatment of the aneurysm, patient can continue the pregnancy till term.[44]

Studies have shown that the route of delivery has no effect on the rate of maternal complications. Most clinicians prefer vaginal delivery. Caesarean section is done when the aneurysm is diagnosed at term, neurosurgical

intervention within one week before delivery, or other maternal or fetal indications for LSCS.

3.3.4 CEREBRAL VENOUS THROMBOSIS

CVT is any thrombosis occurring in intracerebral veins and sinuses and this is characterized by a wide range of clinical features depending on the sinus involved. Women suffer with this disorder more common than men with a ratio of 3 to 1.[45] Pregnancy and puerperium are the most significant procoagulant states leading to thrombosis in the cerebral venous system. Indian studies have reported that prevalence of CVT in Indian subcontinent is 4.5 per 1000 obstetric admissions.[46] Panagariya *et al* from India has documented that CVT accounts for 50% of all young strokes and for 40% of strokes in females.[47]

A. HISTORY OF CVT

The puerperal CVT was first described in 1828 by John Abercrombie of Scotland.[1] Since then the association between CVT and pregnancy has been explained by many authors.[14] The risk of CVA due to venous thrombosis is higher during pregnancy than non-pregnant status. It was believed for a long time that almost all pregnancy related CVA were due to Cerebral Venous Thrombosis. But these studies were based on the observations made before the introduction of newer diagnostic procedures. In 1968, this concept was changed by Cross *et al*. He analyzed 31 patients with Cerebro Vascular Accidents that occurred during pregnancy and puerperium. He reported that most of the cases were due to arterial thrombosis and only one was due to venous occlusion.[13]

However, further studies have reported a greater incidence of venous infarcts in pregnancy associated stroke. Authors of those studies reported that the risk of pregnancy associated venous infarction is significantly greater during postpartum period.[1] Srinivasan K from India published a report in 1984 mentioning that CVT is responsible for around 20% of young stroke in India. He has observed around 50 cases of CVT per 10000 deliveries[48].

B. INCIDENCE

During pre antibiotic era post infective CVT was more common where as pregnancy associated aseptic CVT has become popular after post antibiotic era. Lanska DJ *et al* analyzed data from the Healthcare Cost and Utilization Project and estimated a risk of 11.6 cases of pregnancy associated CVT per 100 000 deliveries.[16] Jaigobin and Silver reported after analyzing 21 cases of cerebral infarction which occurred in pregnancy and puerperium over a 17-year period. Of these twenty one, 13 cases were arterial infarcts and eight cases were of venous origin. Of eight cases of venous origin seven cases reported in puerperium.[11]

A recent study in Taiwan has reported 11 cases of pregnancy associated CVT, 73% of which were encountered in the postpartum period.[15] Of 113 cases of CVT reported from Mexico City, 73 were related to pregnancy and 61 cases of these occurred in the puerperium.[11] In India the incidence has been calculated to be 4.5/1000 obstetric admissions.[46]

C. ANATOMY OF CEREBRAL VENOUS SYSTEM

Cerebral venous system can be classified into two major groups:

1. Superficial system: It drains superficial surfaces of both cerebral hemispheres and it is formed by sagittal sinuses and cortical veins
2. Deep system: It drains deep cortical veins of cerebral parenchyma and it is comprised by lateral sinuses, straight sinus and sigmoid sinus.

- Venous blood from the cerebral hemisphere flows through the superficial cortical veins and deep veins in to the venous sinuses of dura. There are numerous connections between the cortical veins and the dural venous sinuses .This anatomical setup facilitates the spread of the thrombus between these vessels. In the event of any occlusion it also permits formation of collaterals.
- The venous sinuses are placed between the two rigid layers of duramater. These layers protect the sinuses from compression.
- Central nervous system has no valves or muscular layer in the venous channels. Since the valves are absent blood can flow in either direction. The absence of muscular coat permits veins to remain dilated.[52]
- The cortical veins of superior surface drain into the superior sagittal sinus against the blood stream of the same. It leads to turbulence in the blood stream. Fibrous septa which present at the inferior angle of the SSS further aggravate the turbulence. These factors are responsible for the high prevalence of Superior sagittal sinus thrombosis. [52]

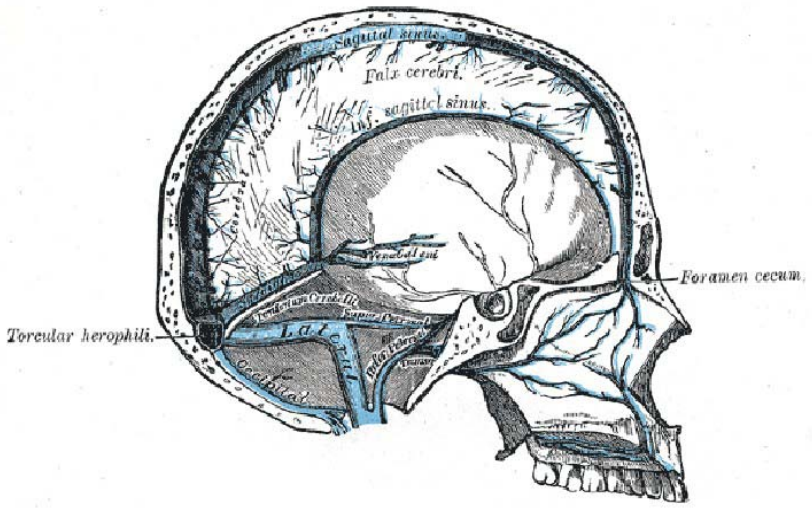


Figure 1: Sagittal section of the skull, showing the sinuses of the dura

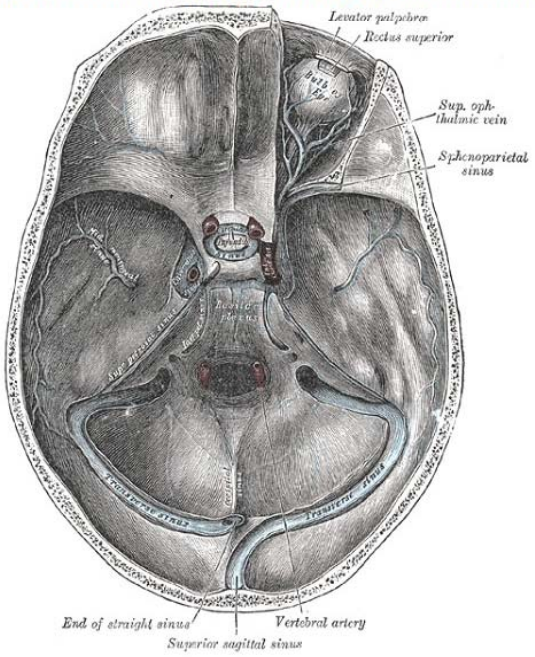


Figure 2: Sinuses at the base of the skull

D. ETIOPATHOGENESIS

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.[1] But Lanska et al has reported that more vulnerable age for peripartum CVT is 15-25 year.[49,50] 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.[47] Cantu et al has reported that cases of puerperal CVT show significantly higher incidence of anemia and raised ESR.[11]

Obstetric CVT has an acute onset and better prognosis than thrombosis due to other causes.[11] 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.[11] When maternal deaths occur, they usually result secondary to intracerebral haemorrhage. Canhao P *et al* has observed that transtentorial herniation to be the most common cause of death.[51]

E. CAUSES OF CEREBRAL VENOUS THROMBOSIS

a) Hypercoagulable Conditions.

-Pregnancy and Puerperium

-Hormonal Contraceptives

-Anti-thrombin III deficiency

-APLA Syndrome

-Protein C & S abnormalities

-Factor V Leiden and factor II gene mutations

b) Changes in vessel wall.

-Neoplasms

-Infections : local or systemic

c) Changes in blood flow/viscosity.

-Malnutrition

-Dehydration

-Cardiac failure

-Hyperviscosity syndrome

-Metabolic syndrome

F. PATHOLOGICAL CHANGES RELATED TO SYMPTOMS AND SIGNS:

- Thrombus formation and obstruction of flow in the cortical veins can eventually lead to venous infarction. Due to infarction neurological symptoms and signs take place.
- As a consequence of obstruction of flow in major venous sinuses, the venous pressure increases and there is impaired drainage of cerebrospinal fluid. This leads to increased intracranial tension casing severe headache and vomiting. Retinal examination shows papilloedema. [53]
- Obstruction of cerebral venous drainage leads to localized cerebral edema and venous infarction. The resulting ischemia causes neuronal damage and haemorrhagic spots. The distended veins sometimes lead to large haemorrhages.[53]
- Local ischemia following venous occlusion causes release of cytotoxic substances. This eventually leads to damage of the energy dependant membrane pumps of cell wall and causes cellular edema.[54]
- There is disruption of the blood brain barrier and leakage of plasma into the interstitial space. This leads to vasogenic edema. If the venous occlusion is promptly managed it reverses gradually.[1]
- Disruption of arachnoid villi located in the walls of major sinuses leads to poor drainage of CSF. This eventually results in increased intracranial tension.[1]

Because there is no pressure gradient, ventricular dilatation and hydrocephalus rarely occurs.

G. CLINICAL FEATURES

Studies have found that peripartum 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.[11]

Clinical features depend on which vein is occluded and how fast the clot propagates in the 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.[55]

- Focal Neurological Deficit:

  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.

Neurological signs and symptoms in CVT and their frequency (%)[56]

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%

H. 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

1. NEURO IMAGING:

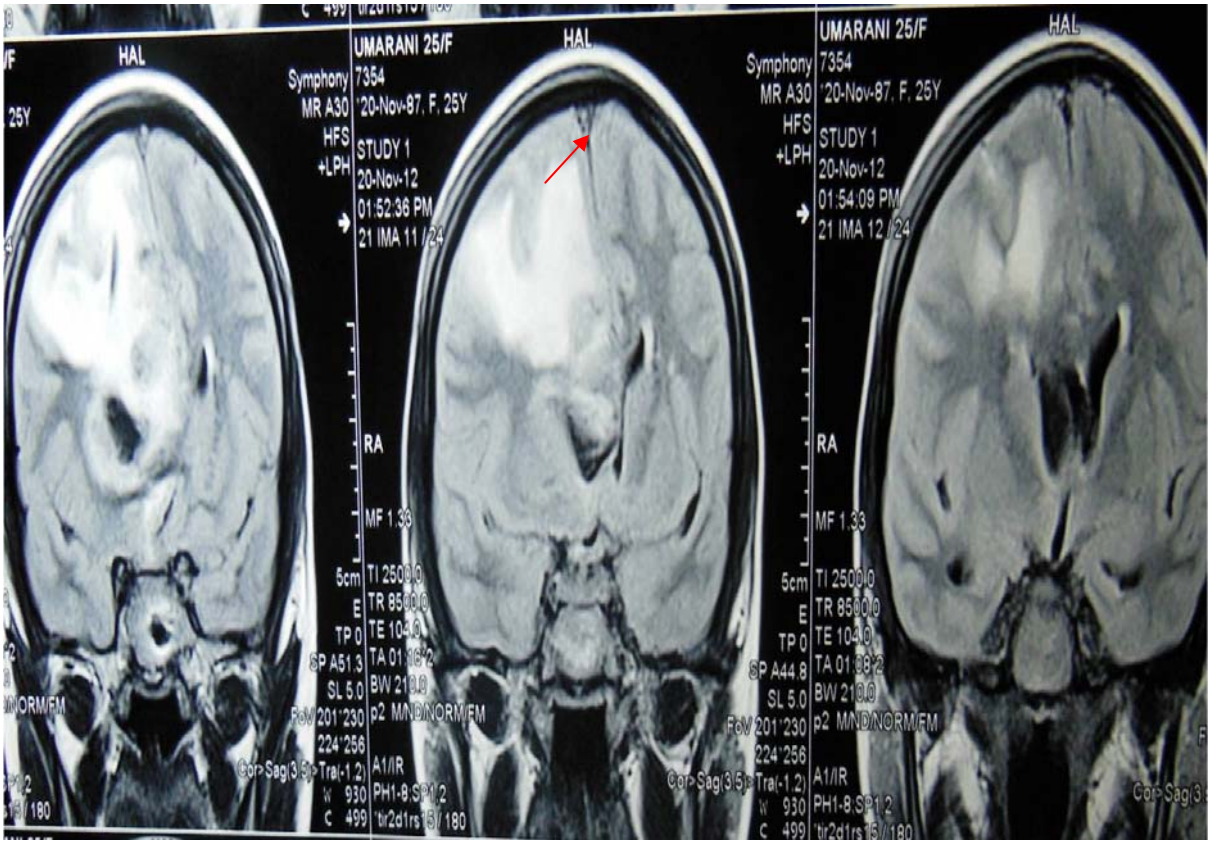
Imaging studies play a key role in timely diagnosis of Cerebral Venous Thrombosis.

A. Computed tomography: There are direct and indirect signs of CVT[57,58]

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.
- 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.
- 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.

EMPTY DELTA SIGN



It is the most common direct sign of CVT. In certain 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 signs 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. [58]

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[58,59]

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 to day5 the lesions appear as Isointense area in T1weighted 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.

C. Trans cranial venous Doppler:

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

2. D Dimer ASSAY:

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

3. LUMBAR PUNCTURE:

It is not helpful in establishing the diagnosis of CVT. It may be used to rule out other causes like meningitis.

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

I.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.[61,62,63]

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.[63] 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 bed sores.

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.[64]

Recent guidelines from American College of Chest Physicians recommend that low-dose aspirin appears to be safe in the first trimester. Warfarin may be safe for the fetus after 12 weeks, it is not usually recommended during pregnancy because of uncertainty surrounding the risks. American Heart Association / American Stroke Association has suggested few

options for pregnant women with ischaemic stroke and high risk thromboembolic conditions:[64]

- Dose adjusted high molecular weight heparin (UFH) throughout pregnancy with aPTT monitoring
- Dose adjusted low-molecular-weight heparin (LMWH) with factor Xa monitoring;

UFH or LMWH until thirteen weeks of pregnancy followed by warfarin up to 34 weeks, after that Heparin injection till delivery.

J. 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.[53]
- 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.[46]

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 and Paroxysmal Nocturnal Haemoglobinuria.

Factors suggestive of bad prognosis are as follows:[1,76]

- 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

K. CRMD RECOMMENDATIONS:

Confidential Review of Maternal Deaths, Kerala 2004-05 recommends the following in view of prevention of CVT[75]

• Early ambulation

• Early and adequate fluid intake

• Use of elastic compression stockings

• Change in the concept about BED REST

Thromboprophylaxis 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 > 4 days

– 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

3.3.5 PARADOXICAL EMBOLISM

Around 25-30% of adults have Patent foramen ovale (PFO) which is an inter atrial communication. The association between cryptogenic stroke in the young and this inter atrial communication is well known as 50% of these patients have PFO.[66]

Pathophysiology:

The presence of communication between the two atria permits shunting of emboli from right venous circulation into the left arterial circulation. This represents the most important pathophysiology behind the Paradoxical embolism.[1] Clot formation due to stagnant blood flow may also occur within the atrial chambers. When there is susceptibility to atrial arrhythmias it further potentiates the risk of paradoxical embolism.[66]

Many studies have described the association of PFO in pregnancy related CVA. As mentioned previously, pregnancy and puerperium are well known pro thrombotic states with an increased risk of venous thromboembolism. Hence venous thrombi, formed in the peripheral venous circulation, can reach the arterial circulation through the inter atrial communication. Straining during

second stage of labour may alter the pressure gradient across the atrial chambers, facilitating shunting of emboli from to left. The presence of PFO in a pregnant woman therefore provides an additional risk for CVA due to arterial occlusion. The CVA associated with PFO is an ischaemic stroke usually involving smaller areas. Patients with artificial cardiac valves may develop stroke due to cardio embolism. Rarely paradoxical amniotic emboli can cause venous or arterial events. Rare cases of air embolism resulting in ischaemic stroke during caesarean section have also been reported .

Treatment:

Treatment for patients having CVA due to paradoxical embolism includes drug therapy, in the form of warfarin or antiplatelet agents, and percutaneous transcatheter closure. ASA guidelines recommend use of aspirin for secondary prevention, while warfarin reserved for high-risk patients. Closure of atrial defect may be considered in patients with recurrent stroke in spite of adequate drug therapy. Transcatheter closure has been widely used nowadays[67]. Prophylactic anti thrombotic therapy is recommended for pregnant women with prosthetic valves and for those with history of stroke caused by paradoxical embolism.[66]

3.3.6 PERIPARTUM CARDIOMYOPATHY (PPCM)

Dilated cardiomyopathy is a well known risk factor for thrombo embolic stroke. PPCM is a disorder of unknown etiology which manifests in the peripartum period. It is characterized by symptoms of left ventricular failure in

women without pre-existing heart disease. Peripartum cardiomyopathy was first described in 1930s. A diagnostic criteria was established in 1971 which has been revised in 2000.[68]

Peripartum cardiomyopathy is a diagnosis of exclusion. It is defined as cardiac failure that manifests anytime between last month of pregnancy and 5 months postpartum in a woman without any underlying heart disease and without obvious cause for cardiac failure. Diagnostic criteria on echocardiography includes,

- Ejection fraction of less than 45%
- End diastolic shortening of less than 2.72cm/m^2

The incidence of PPCM has a wide geographical variation, ranging from 1 in 2400 to 1 in 15000 pregnancies.[1] The incidence is higher in Africans, elderly gravida, multiparous women and in whom pregnancy is complicated by multifetal gestation, hypertensive disorders or prolonged tocolytic therapy. Reported mortality associated with PPCM is about 18-50%[1]

Pathophysiology

The etiopathogenesis of peripartum cardiomyopathy is still unclear. Proposed causative factors are myocarditis, nutritional deficiency, altered immunology, stress induced cytokines and viral antigens. Stasis and thrombus formation in the dilated left ventricle is common in PPCM which can cause peripheral embolisation. The triggered emboli results in tissue ischaemia and

infarction where it gets settled. When the emboli reach the cerebral circulation it leads to arterial infarction and stroke. [69]

Treatment

Treatment of PPCM is similar to that of cardiac failure in non pregnant population. Drug therapy includes diuretics, digoxin and ACE inhibitors. Prophylactic antithrombotic therapy is advised to prevent thrombo embolic complications. Prognosis depends on normalization of left ventricular size and function within 6 months of delivery. However the risk of recurrence in subsequent pregnancies is 50-100%.[1,69]

3.3.7 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. Ovarian hyper stimulation syndrome causing Middle cerebral artery thrombosis[70]
4. stroke related to the use of anaesthetic drugs in pregnancy**.**

3.4. INVESTIGATIONS FOR STROKE IN PREGNANCY

We should proceed with the investigations as in non pregnant state but with special considerations regarding pregnancy specific causes and teratogenic issues of the diagnostic procedures. Neuro imaging findings have been discussed already under concerned disorders.[1] Here we shall discuss the issues regarding the adverse effects on the fetus.

Computed Tomography

CT Brain involves exposure to ionizing radiation. People have considerable anxiety in view of teratogenicity. The teratogenic effects of ionizing radiation on the fetus can lead to intra uterine death, congenital malformation, fetal growth restriction, damage to the central nervous system and cancer susceptibility. Risk is proportionate to the dose of radiation and duration of exposure. Absorbed dose is measured in rad or Gray.

The maximum estimated fetal absorbed dose of ionizing radiation is[71]

- 50 mrad for a CT brain

-10 mrad for cerebral angiography and

- 1.0 mrad for a chest xray

However, natural background exposure existing at sea level is about 300 mrad per year. When a fetus is exposed to radiation during maternal CT Brain study, the lifetime risk of developing cancer increases by 0.1%. Studies have reported that pregnant women exposed to < 5000 mrad have no additional risk to the fetus compared with women receiving background radiation alone.[71,72]

Magnetic Resonance imaging

MRI procedures do not cause ionizing radiation, and there are no documented adverse effects on the fetus. However any long-term effects are yet to be studied. Recent guidelines from the American College of Radiology suggest that pregnant patients can undergo MRI scans, provided that the potential risk/benefit ratio favours the procedure, unable to obtain the information through another non-ionizing technique, the information is required immediately and cannot wait until the patient completes her pregnancy.[1]

The contrast agents used in MRI studies can cross placental barrier easily. Guidelines suggest that administration of gadolinium-based MR contrast agents must be avoided unless potential benefit to the patient or fetus outweighs the theoretical risks. MRV has the advantages of being easily repeatable and noninvasive.[72]

Other investigations are Tests for haemotological abnormalities, Anti nuclear antibodies, Echocardiography, Tests for Lupus anticoagulant and Anti cardiolipin antibody, Coagulation profile, Lumbar puncture and D Dimer assay.

3.5. MANAGEMENT OF CVA IN PREGNANCY AND PUERPERIUM

Specific treatment modalities related to individual causes of stroke have been discussed under the relevant sections. We shall discuss general issues regarding the role of thrombolytic therapy during pregnancy.

The benefit of thrombolysis in acute venous and arterial occlusion is described by many authors. Historically use of thrombolytic agents during pregnancy is not being recommended because of concerns about fetal and maternal complications. Possible risks are preterm labour, abruption placenta, intra uterine death and PPH. There are no randomized controlled trials in pregnant patients available at present. Only clinical case series have been reported.

Murugappan *et al* reported 8 patients who had received thrombolytic therapy for acute ischaemic stroke during pregnancy. Seven patients had recovered well. 3 mothers had induced abortions, two spontaneous abortions, and 2 babies born healthy. [73]

The benefits of thrombolytic therapy in reducing maternal mortality and morbidity should be outweighed against the potential risks.

3.6 RECURRENCE:

Lamy *et al* has observed 489 patients with pregnancy related CVA. 13 women had recurrence but only 2 of those encountered during pregnancy. He has mentioned that occurrence of stroke during pregnancy or puerperium is not a contraindication for subsequent pregnancies.[7]

MATERIALS AND METHODS

A study of the patients with cerebrovascular accidents during pregnancy and puerperium admitted in our Institution for a period of 22 months was done.

Type of study: Prospective and Retrospective Analysis

Population under study:

Patients admitted in Labour ward, Intensive medical care unit and Neurology unit of Tirunelveli Medical College Hospital with cerebrovascular accidents during pregnancy or puerperium from January 2011 to October 2012.

Inclusion criteria:

Patients presenting with headache, altered sensorium, unifocal or multifocal seizures, neurological deficit and behavioral abnormalities during pregnancy or within six weeks of delivery.

Exclusion criteria:

Patients with history of trauma, seizure disorder, infections (meningitis and encephalitis) and intracranial space occupying lesions.

Data collection:

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

- The details noted are age of the patient, socio economic status, parity, presence of predisposing factors, time of presentation, mode of delivery and presenting symptoms.

- The general physical examination findings and vitals are recorded along with the findings on Neurological examination.
- Laboratory and Radiological investigations are done according to available facilities in our institution.
- Details about the treatment, course in hospital, outcome of treatment, and duration of hospital stay is done.

Detailed analysis of data is done at the end of the study period to find out the predisposing factors and the spectrum of presentation of CVA in pregnancy and puerperium.

Main outcome measures:

The spectrum of cerebrovascular accidents in pregnancy and puerperium, its clinical presentation, management and outcome

Secondary outcome measures:

The risk factors for CVA during pregnancy and puerperium and the prognosis based on them.

RESULTS AND ANALYSIS

87 patients with cerebrovascular accidents during pregnancy or puerperium admitted in Tirunelveli Medical College Hospital during a period of 22 months were studied.

The results of the study are documented below.

AGE DISTRIBUTION OF CASES

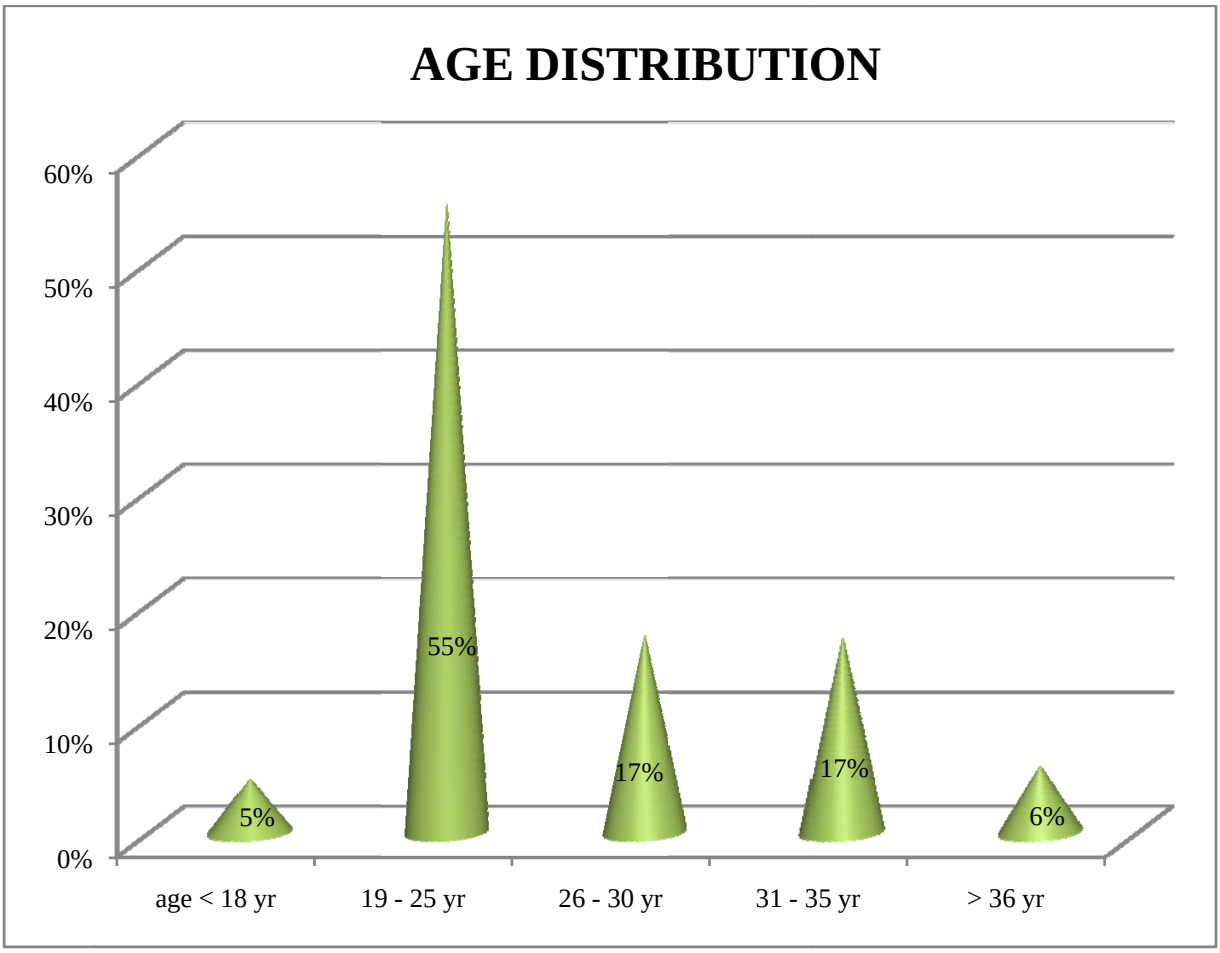
Table 1

Total number of patients: 87

AGE	NO. OF PATIENTS	PERCENTAGE
< 18 YR	**4**	**4.59%**
19 – 25 YR	**48**	**55.17%**
26 – 30 YR	**15**	**17.24%**
31 – 35 YR	**15**	**17.24%**
>36 YR	**5**	**5.74%**

Maximum occurrence of CVA was noted in the age group of 19- 25 years contributing to 55.17 %. Similar incidence was noted in 26-30 yr and 31-35 yr age groups which was 17.24%. The youngest case of stroke is a Primi of 17 year old with Takayasu arteritis.

Figure 1


AGE DISTRIBUTION
60%
50%
40%
30%
20%
10%
0%
5%
55%
17%
17%
6%
age < 18 yr
19 - 25 yr
26 - 30 yr
31 - 35 yr
> 36 yr

PARITY

Table 2

PARITY	NO. OF PATIENTS	PERCENTAGE
G_1	**38**	**43.67%**
G_2	**24**	**27.58%**
G_3	**18**	**20.68%**
G_4 and above	**7**	**8.04%**
TOTAL	**87**	

G_1- Antenatal patient with first conception (primi gravida) and postnatal patient after first delivery (primi para) irrespective of the outcome of the delivery.

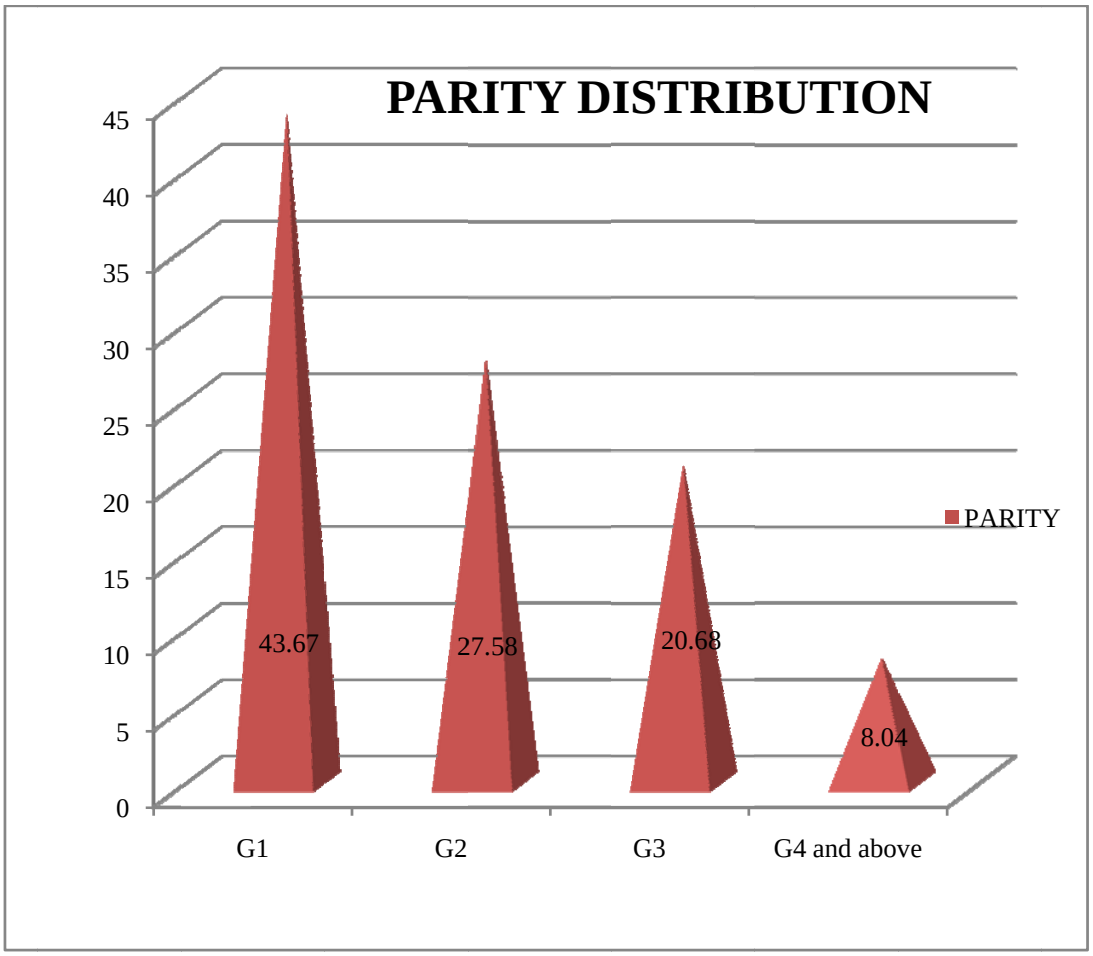
G_2- Antenatal patient with second conception (G_2A_1,$G_2P_1L_0$,$G_2P_1L_1$) and postnatal patient after second delivery(A_2, P_2L_0 ,P_2L_1 ,P_2L_2) irrespective of the outcome of previous and present pregnancies.

G_3- Antenatal patient with third conception and postnatal patient after third delivery, irrespective of the outcome of pregnancies.

G_4 – Patients with fourth conception or fourth delivery irrespective of the outcome of the delivery

43.67% of patients were primigravida or primi para (G_1) which was the highest occurrence followed by G_2 group showing 27.58% occurrence

Figure 2


PARITY DISTRIBUTION
45
40
35
30
25
20
15
10
5
0
43.67
27.58
20.68
8.04
G1
G2
G3
G4 and above
PARITY

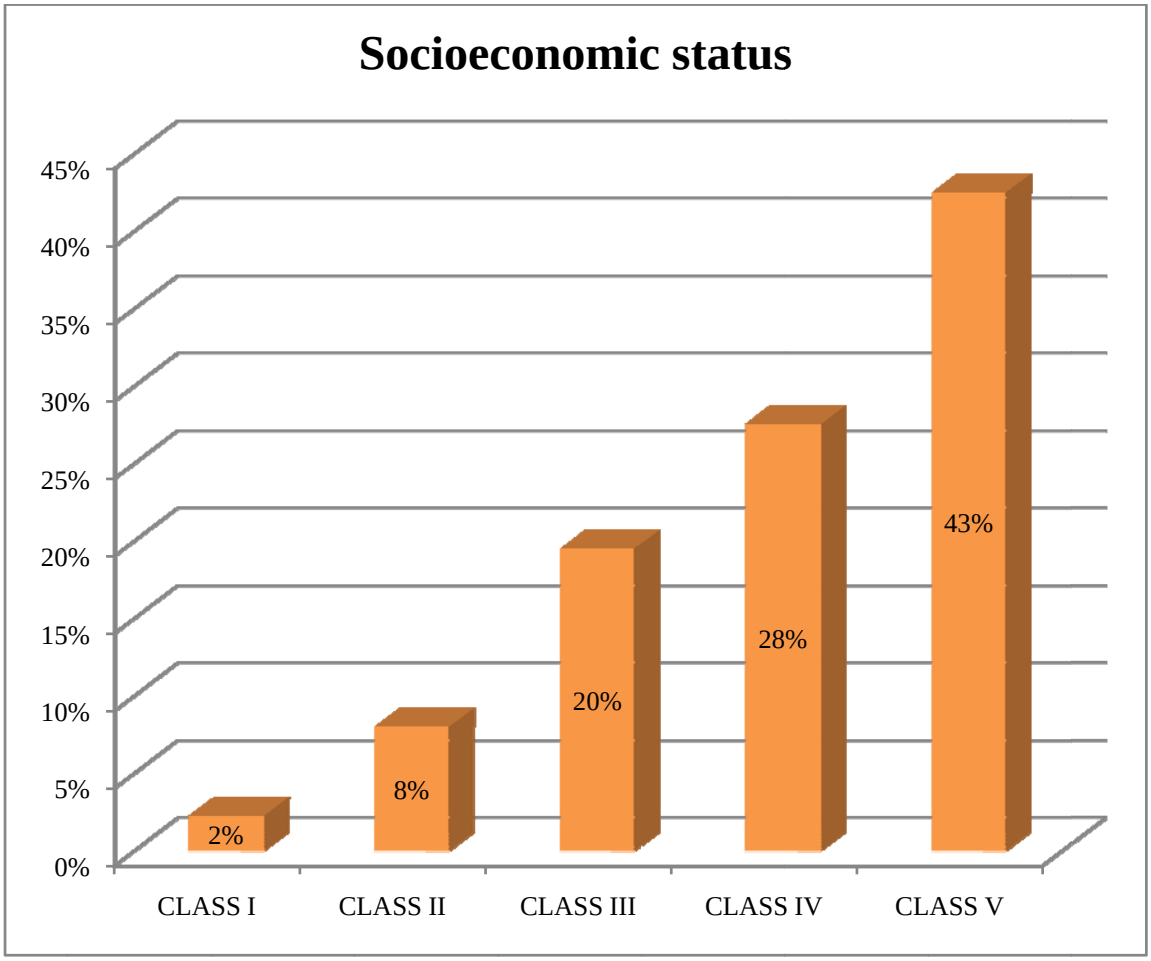
SOCIOECONOMIC STATUS

Table 3

Socioeconomic class	No. of patients	Percentage
Class I	**2**	**2.29%**
Class II	**7**	**8.04%**
Class III	**17**	**19.54%**
Class IV	**24**	**27.58%**
Class V	**37**	**42.52%**

Patients were classified using Modified Kuppusamy Scale. 42.52% of patients were from class V socio economic class followed by class IV with 27.58%.

Figure 3


Socioeconomic status
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
2%
8%
20%
28%
43%
CLASS I
CLASS II
CLASS III
CLASS IV
CLASS V

AREA OF DISRIBUTION

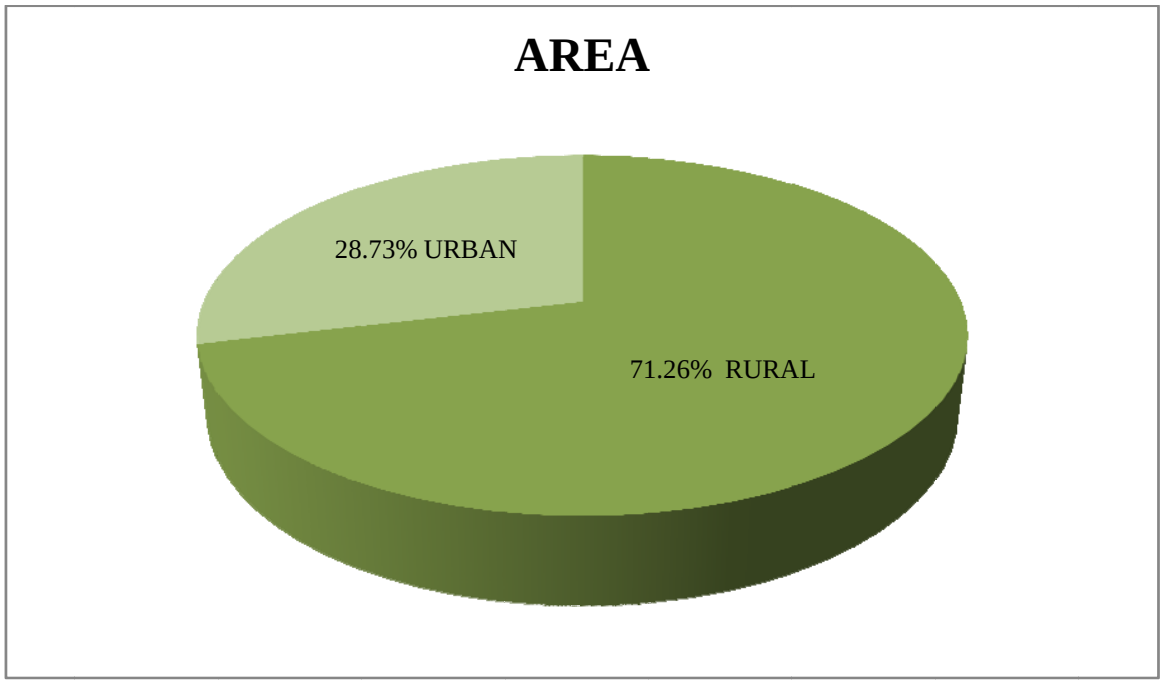
Table 4

Total number of patients: 87

AREA	NO.OF PATIENTS	PERCENTAGE
RURAL	**62**	**71.26**
URBAN	**25**	**28.73**

Maximum occurrence was noted in patients from rural area which was 71.26%. Patients belonging to urban areas contributed to 28.73%

Figure 4



TIME OF PRESENTATION

Table 5

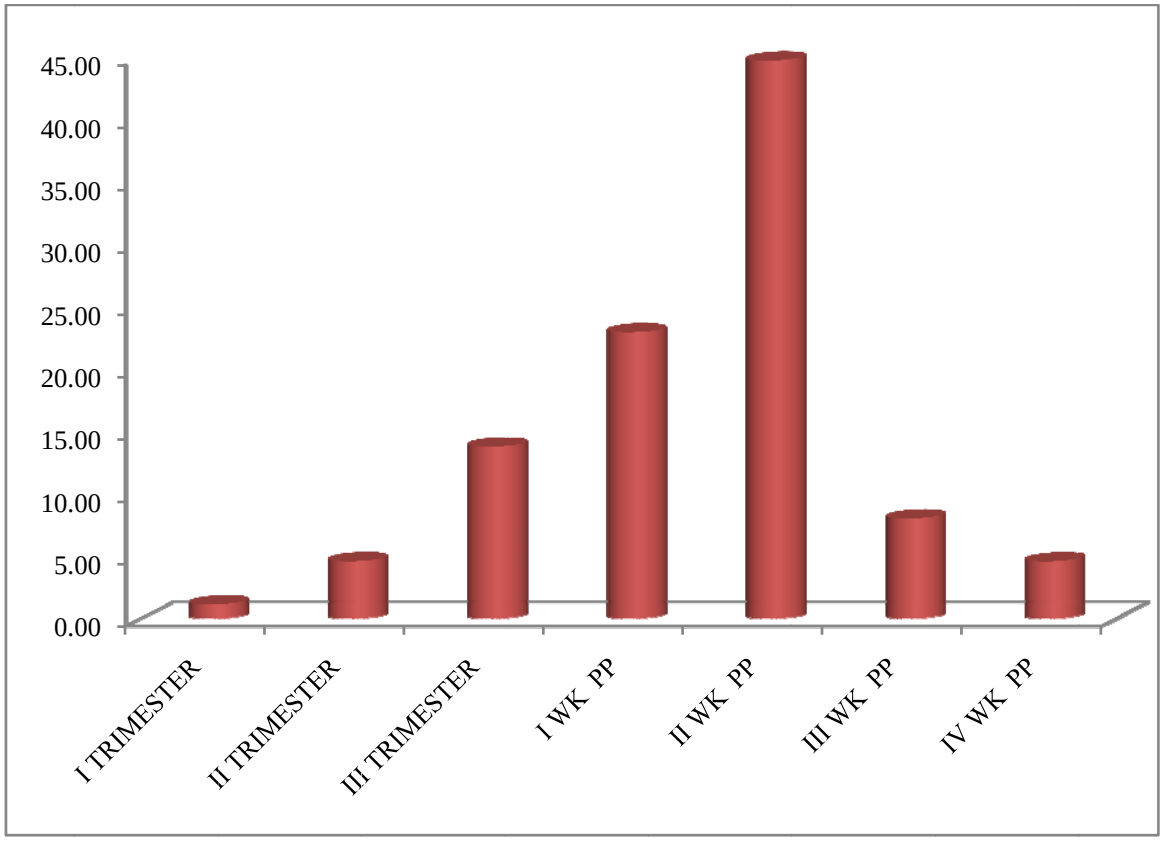
TIME OF PRESENTATION	NO. OF PATIENTS	PERCENTAGE
ANTEPARTUM	**17**	**19.54**
POSTPARTUM	**70**	**80.45**

Table 6

TIME OF PRESENTATION	**NO. OF PATIENTS**	**PERCENTAGE**
I TRIMESTER	1	1.14%
II TRIMESTER	4	4.59%
III TRIMESTER	12	13.79%
I WK POSTPARTUM	20	22.98%
II WK POSTPARTUM	39	44.82%
III WK POSTPARTUM	7	8.04%
IV WK POSTPARTUM	4	4.59%

Figure 5

TIME OF PRESENTATION



80.45% of patients had CVA in the postpartum period (Table 5) of which 44.82% was in the second week of postpartum.(Table 6). Majority of postpartum CVA are due to Cerebral Venous Thrombosis. Only one case of CVT was documented in the antepartum period.

MODE OF DELIVERY

Table 7

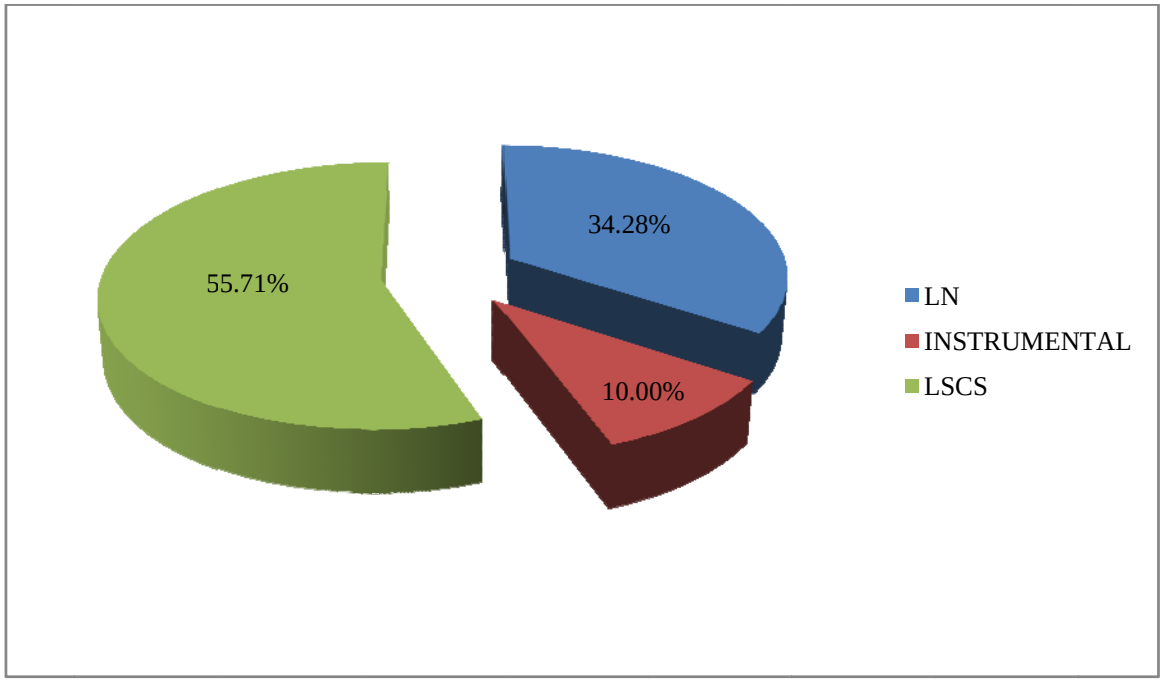
Total number of patients: 70

MODE OF DELIVERY	NO OF PATIENTS	PERCENTAGE
LABOUR NATURALIS	24	34.28%
INSTRUMENTAL	7	10%
LSCS	39	55.71%

Of all the patients with postpartum CVA 55.71% had undergone Caesarean section due to various reasons. One patient had undergone Subtotal hysterectomy due to atonic PPH. 44.28% had undergone vaginal deliveries either Labour naturalis or Instrumental delivery

Figure 6

MODE OF DELIVERY



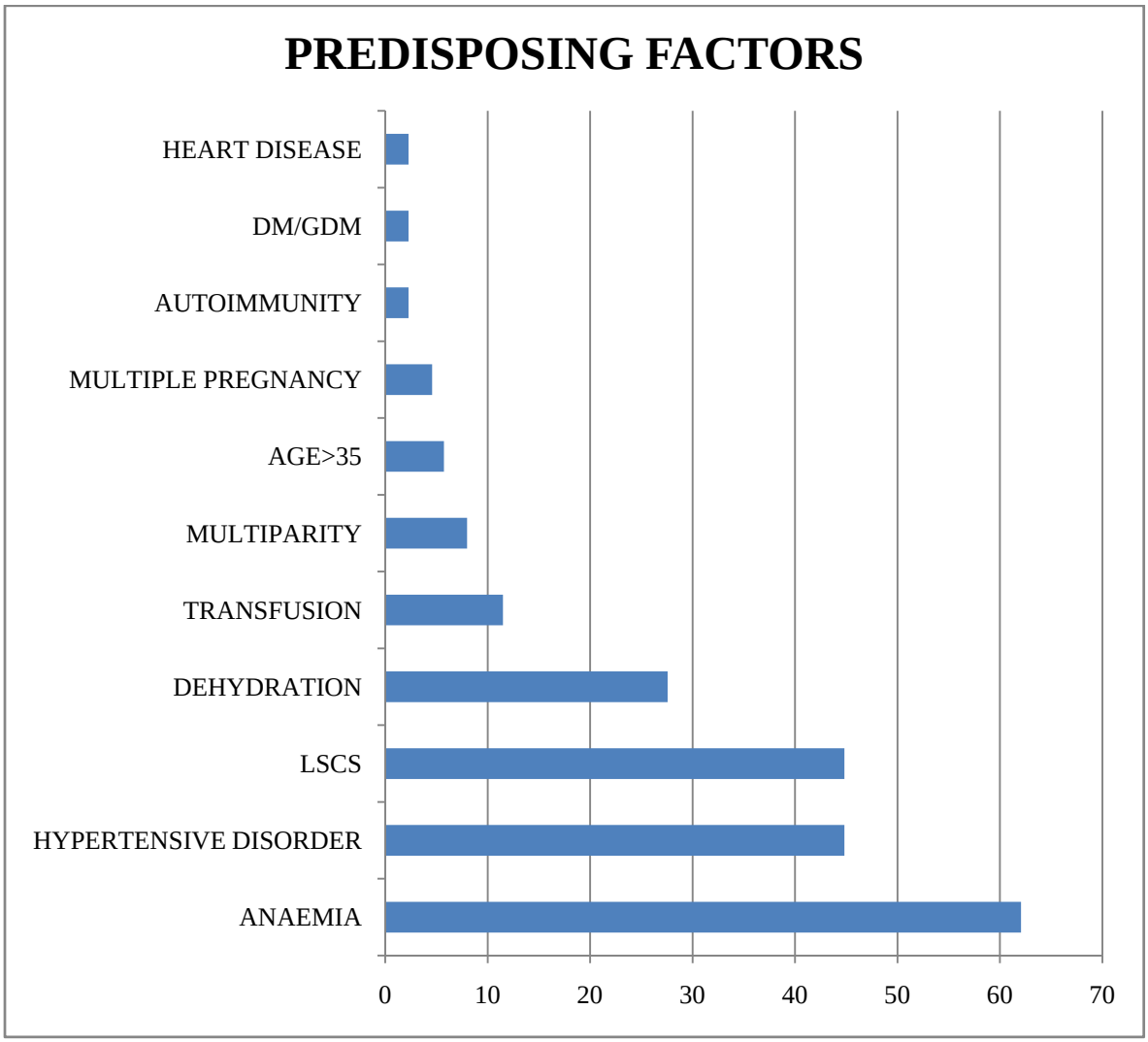
PREDISPOSING FACTORS

Table 8

RISK FACTOR	NO OF PATIENTS	PERCENTAGE
HT DISORDERS	39	44.82%
ANAEMIA	54	62.06%
LSCS	39	44.82%
DEHYDRATION	24	27.58%
BLOODTRANSFUSION	10	11.49%
AGE > 35	5	5.74%
GDM/DM	2	2.29%
MULTIPLE PREGNANCY	4	4.59%
AOTO IMMUNE DISORDER	2	2.29%
HEART DISEASE	2	2.29%

Anaemia was present in 62.06% of patients and was the most common risk factor. Hypertensive disorders and caesarean section were present in 44.82% patients. Three patients were chronic hypertensives. 11.49% of patients had undergone blood transfusion due to anemia, abruption or postpartum haemorrhage. One patient had undergone mitral valve replacement 2 year back and discontinued thrombo prophylaxis.

Figure 7


PREDISPOSING FACTORS
HEART DISEASE
DM/GDM
AUTOIMMUNITY
MULTIPLE PREGNANCY
AGE>35
MULTIPARITY
TRANSFUSION
DEHYDRATION
LSCS
HYPERTENSIVE DISORDER
ANAEMIA
0
10
20
30
40
50
60
70

SYMPTOMATOLOGY

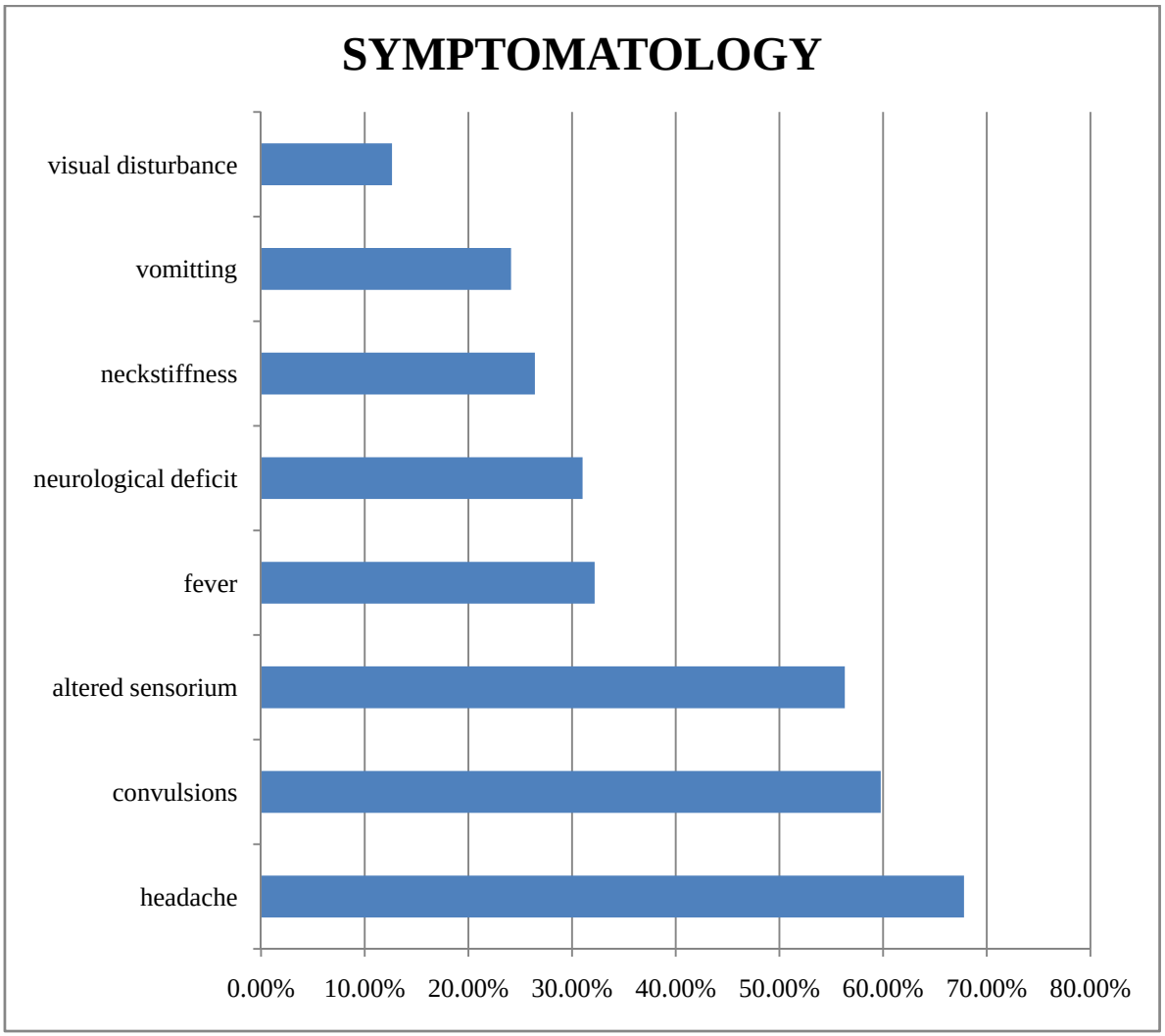
Table 9

TOTAL PATIENTS: 87

SYMPTOM	NO OF PATIENTS	ERCENTAGE
Headache	59	67.81%
Convulsions	52	59.77%
Altered sensorium	49	56.32%
Visual disturbance	11	12.64%
Neck stiffness	23	26.43%
Fever	28	32.18%
Vomiting	21	24.13%
Neurological deficit	27	31.03%

In our study the most common symptom noted was Headache which was present in 67.81% of patients. Convulsions and Altered sensorium are the next two common symptoms with 59.77% and 56.32% respectively.

Figure 8


SYMPTOMATOLOGY
visual disturbance
vomitting
neckstiffness
neurological deficit
fever
altered sensorium
convulsions
headache
0.00%
10.00%
20.00%
30.00%
40.00%
50.00%
60.00%
70.00%
80.00%

LEVEL OF CONSCIOUSNESS

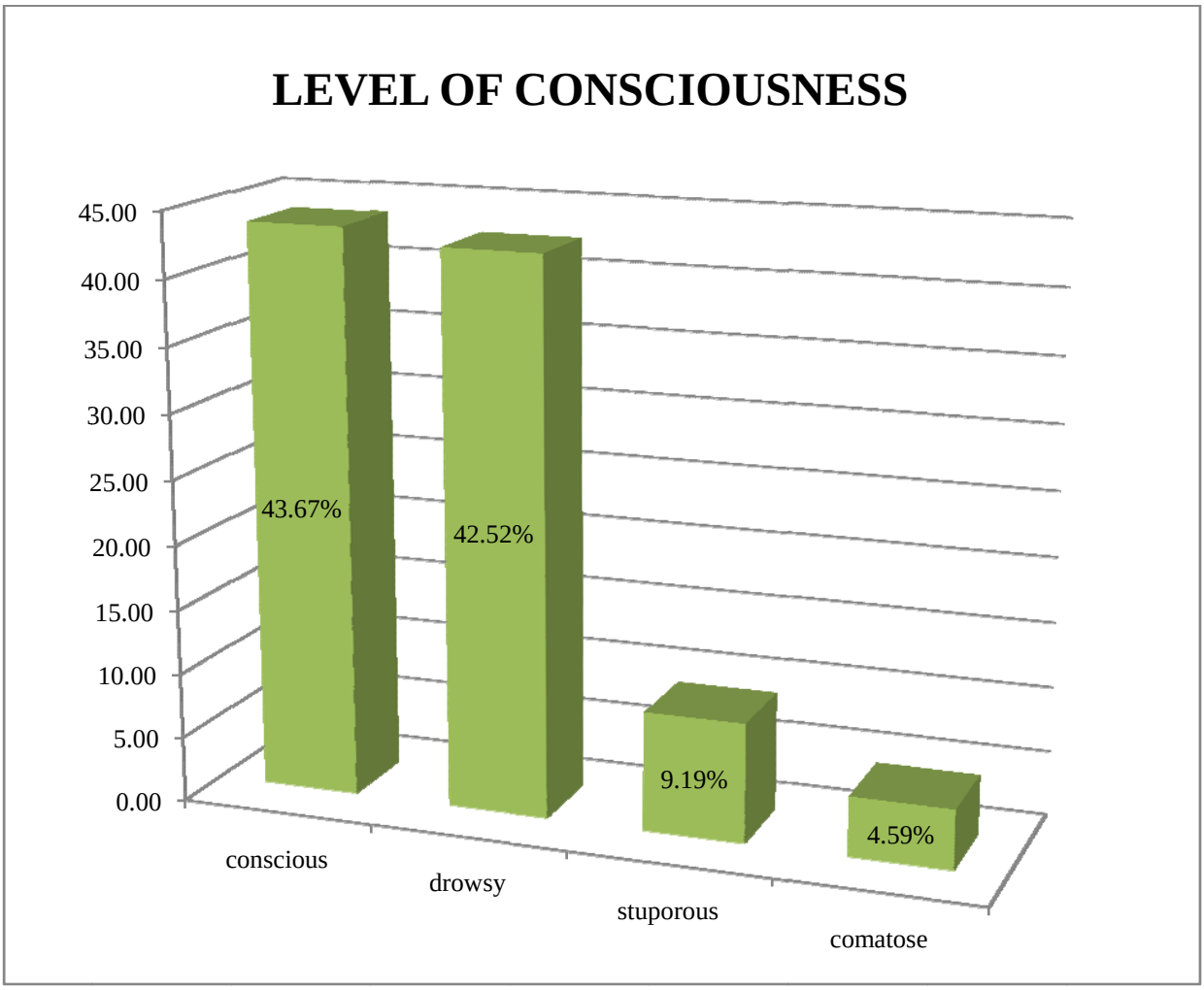
CONSCIOUS: 38 ALTERED SENSORIUM : 49

Table 10

LEVEL OF CONSCIOUSNESS	NO. OF PATIENTS	PERCENTAGE
Conscious	**38**	**43.67**
Drowsy	**37**	**42.52**
Stuporous	**8**	**9.19**
comatose	**4**	**4.59**

Of all the patients, 43.67% were conscious at the time of admission and during the hospital stay. Drowsiness was the most common feature of altered sensorium which was observed in 42.52% of patients**.**

Figure 9


LEVEL OF CONSCIOUSNESS
45.00
40.00
35.00
30.00
25.00
20.00
15.00
10.00
5.00
0.00
43.67%
42.52%
9.19%
4.59%
conscious
drowsy
stuporous
comatose

SEIZURES

TOTAL PATIENTS WITH SEIZURES: 52

Table 11

SEIZURES	NO OF PATIENTS	PERCENTAGE
GTCS	**41**	**78.84%**
FOCAL	**11**	**21.15%**

We observed that 59.77% of patients had convulsions at the time of admission or during the hospital stay. Most common type of seizures was GTCS with 78.84%. Focal seizures were present in 21.15% of patients.

Figure 10

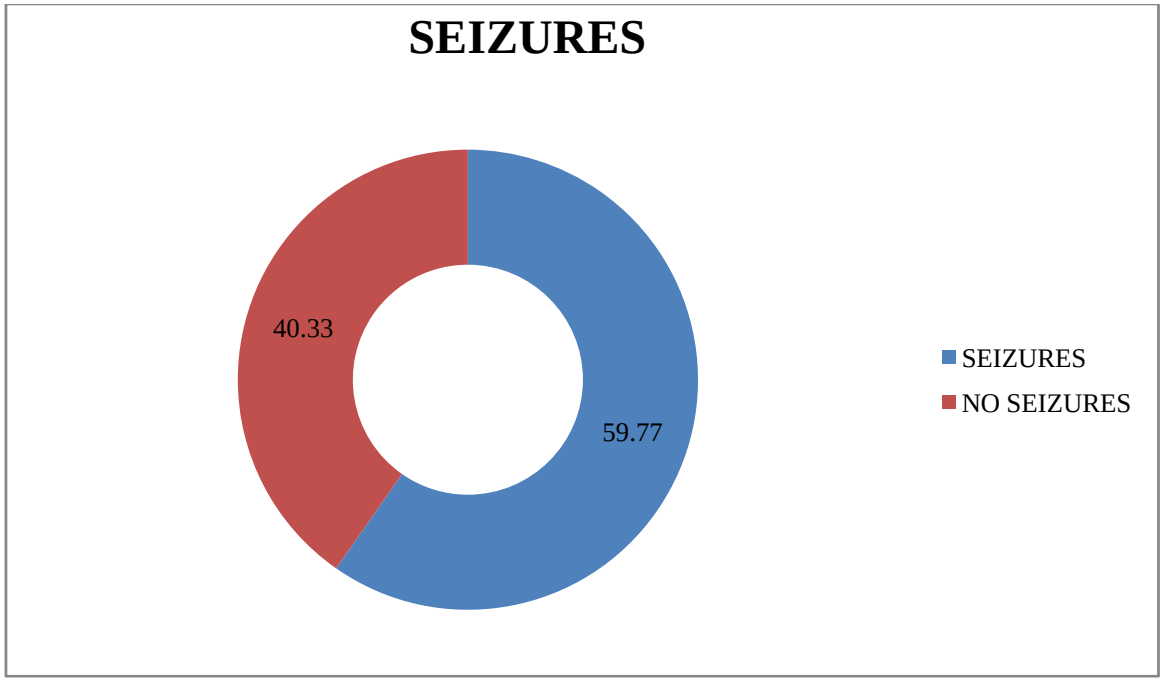
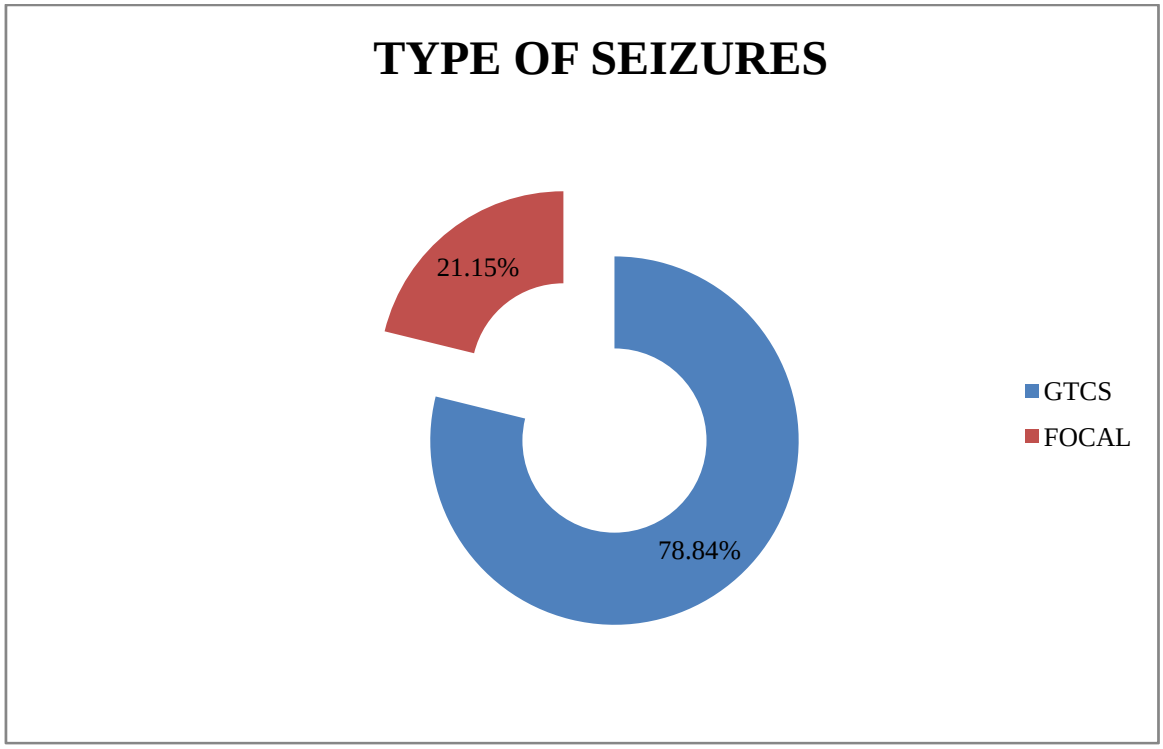


Figure 11



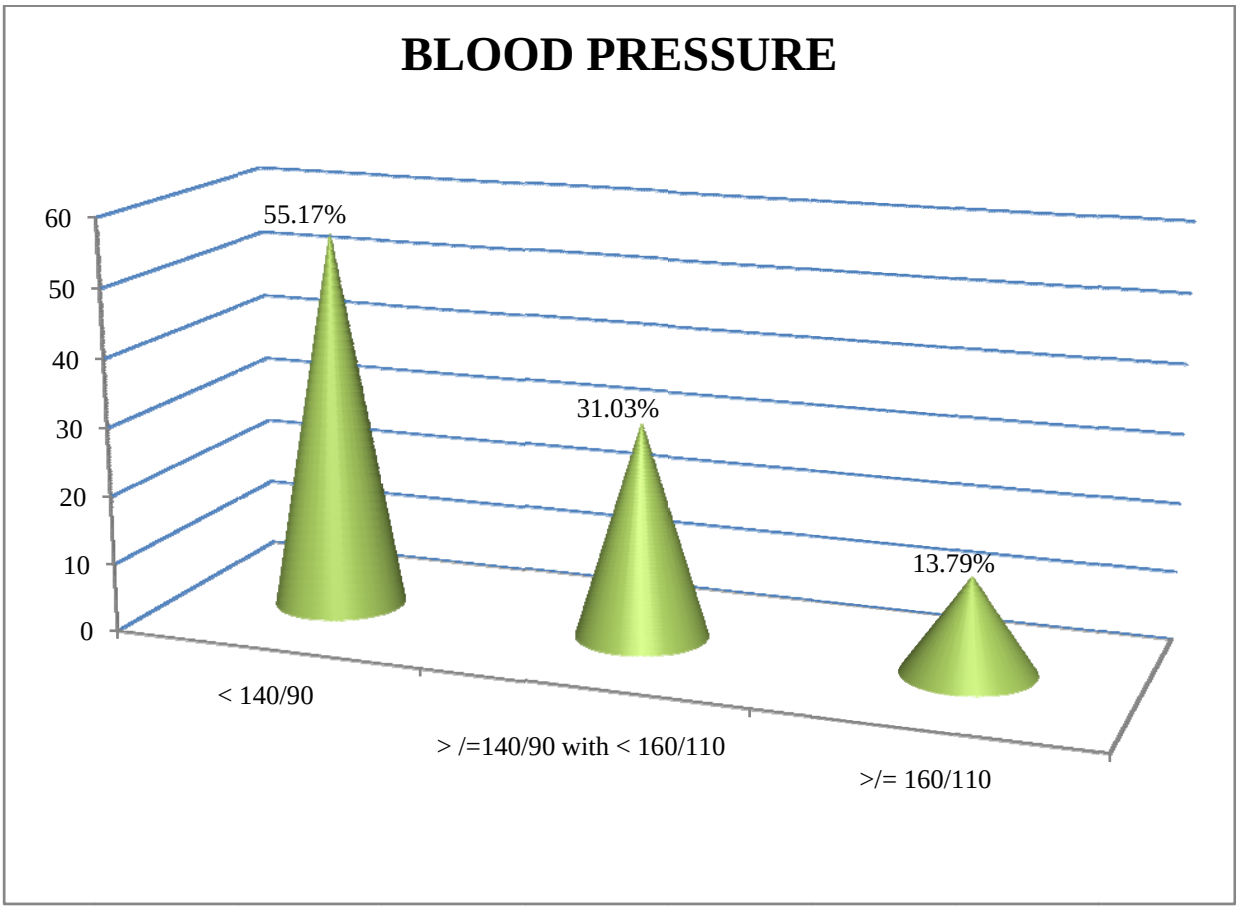
BLOOD PRESSURE

Table 12

BP	NO OF PATIENTS	PERCENTAGE
< 140 mmHg systolic and/ or 90 mmHg diastolic	**48**	**55.17**
>/= 140 systolic and/ or 90 mmHg diastolic	**27**	**31.03**
>/= 160 systolic and/ or 110mmHg diastolic	**12**	**13.79**

In the present study 31.03% had blood pressure between 140/90 mmHg and 160/110 mmHg and 13.79% patients had blood pressure of more than 160/110 mmHg. Maximum patients had blood pressure below 140/90 mmHg with 55.17%

Figure 12


BLOOD PRESSURE
60
50
40
30
20
10
0
55.17%
31.03%
13.79%
< 140/90
> /=140/90 with < 160/110
>/= 160/110

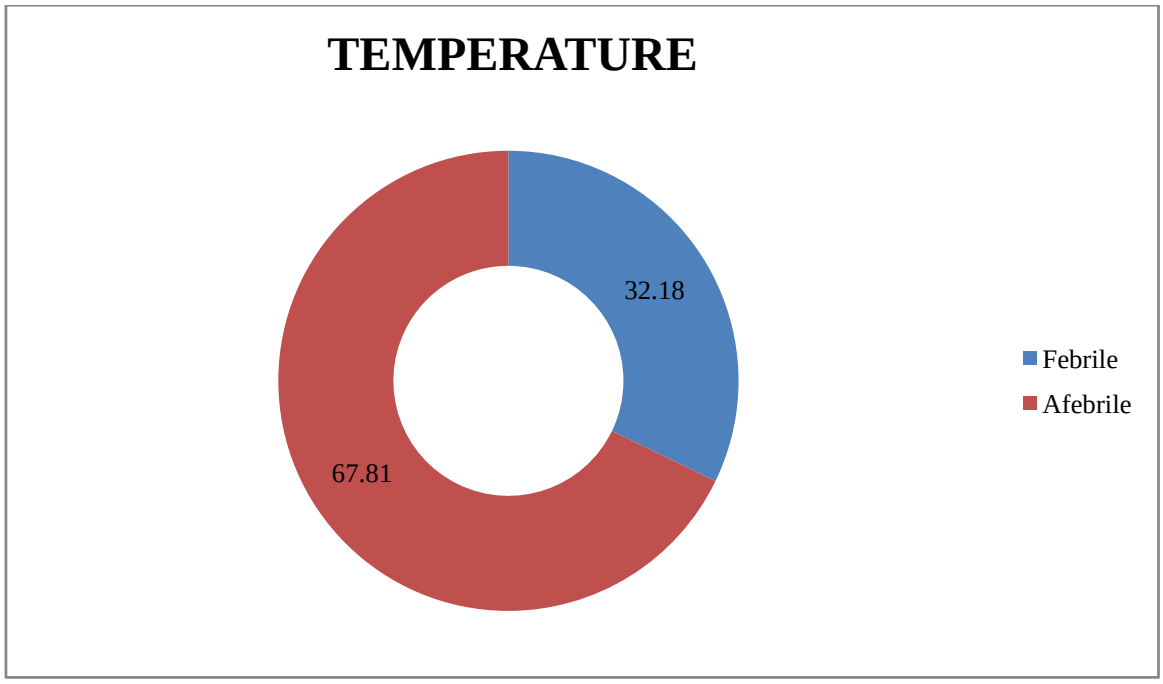
TEMPERATURE

Table 13

TEMPERATURE	NO OF PATIENTS	PERCENTAGE
Febrile	**28**	**32.18**
Afebrile	**59**	**67.81**

We observed that 32.18% of patients were febrile at the time of admission and 67.81% were afebrile. But we were not able to differentiate whether the fever had led to the occurrence of CVA or it was a result of CVA.

Figure 13



HAEMOGLOBIN LEVEL

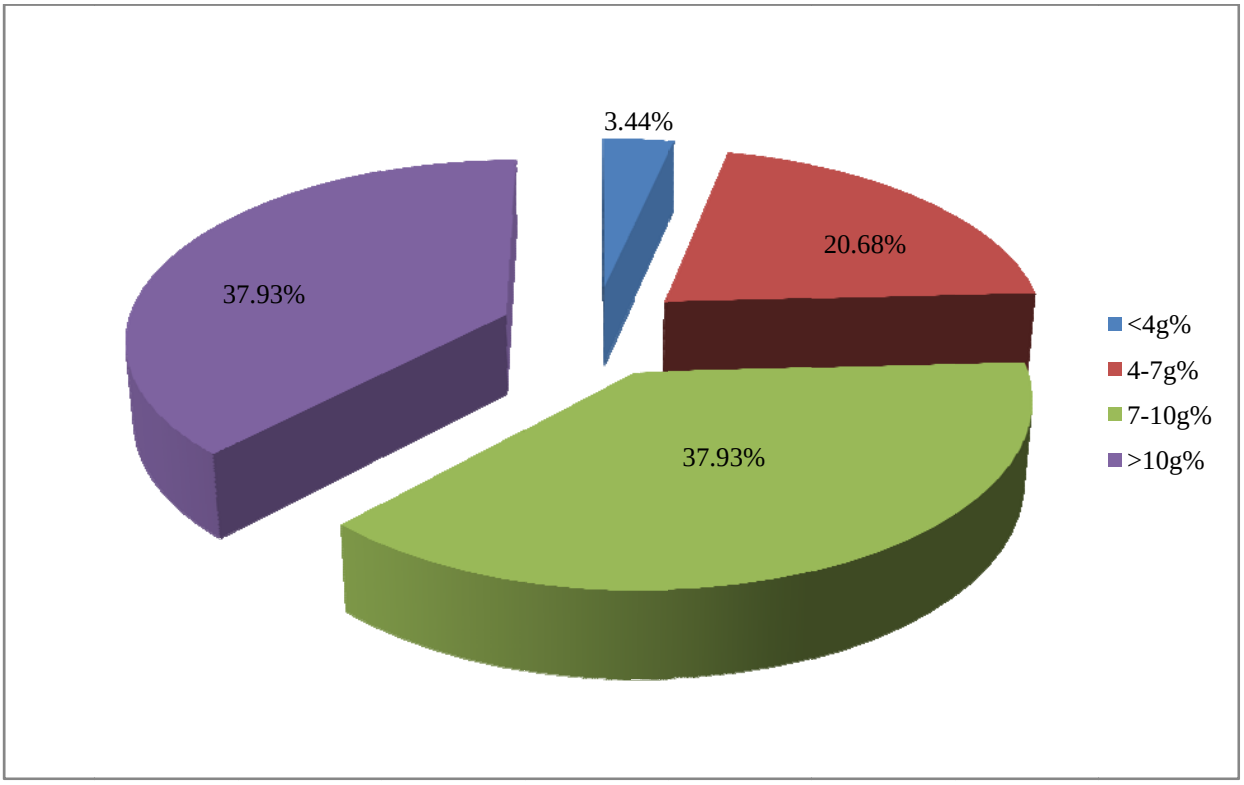
Table 14

Hb %	No. of patients	Percentage
Less than 4 g%	**3**	**3.44**
4-7 g%	**18**	**20.68**
7-10 g%	**33**	**37.93**
More than 10g%	**33**	**37.93**

We observed that 62.06% of patients were anaemic with haemoglobin level less than 10g/dl of which 37.93% had severe anemia and 3.44% had very severe anemia.

Figure 14

HAEMOGLOBIN LEVEL



FINDINGS ON NEURO IMAGING

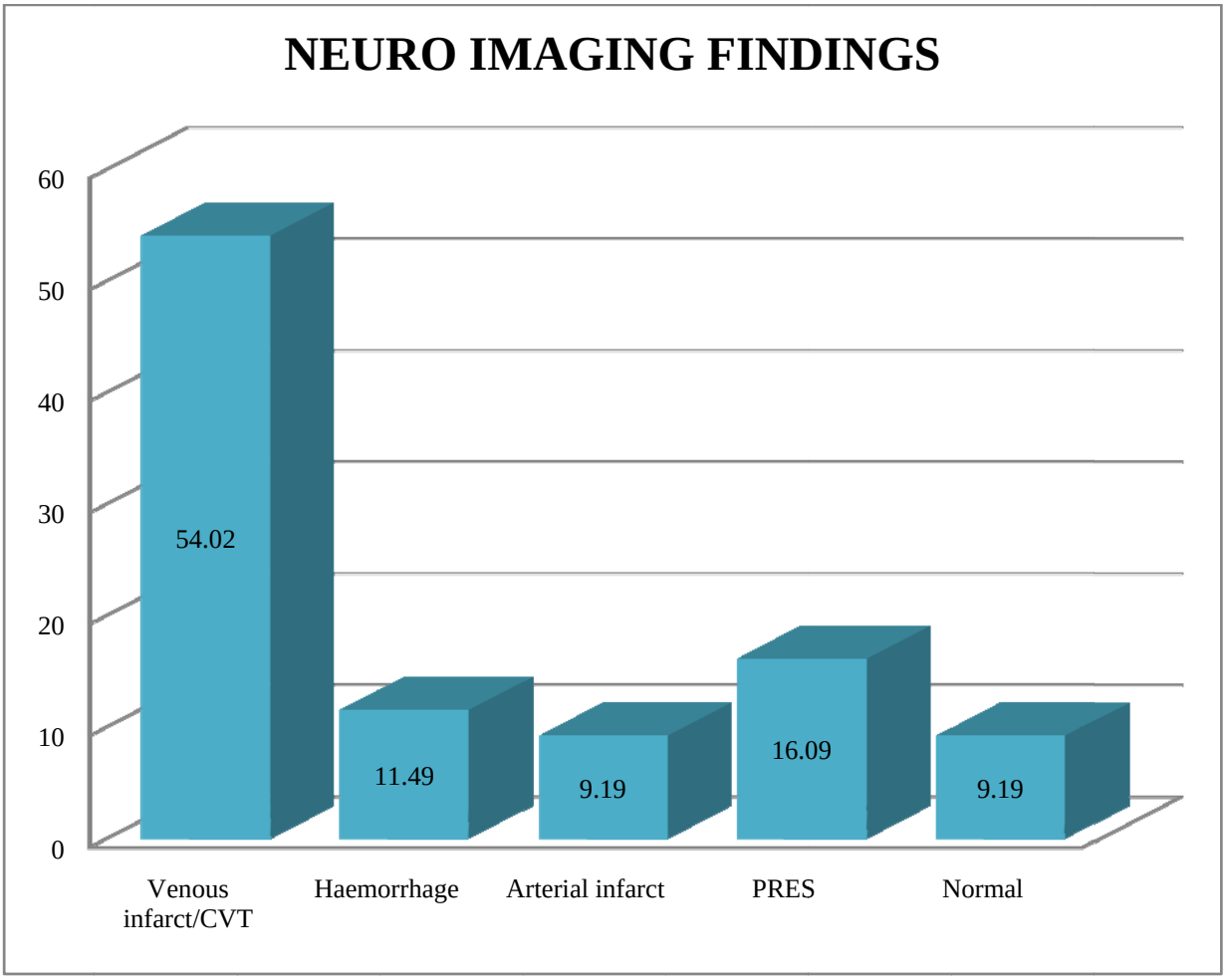
Table 15

FINDINGS	NO OF PATIENTS	PERCENTAGE
Haemorrhage	**10**	**11.49**
Arterial infarct	**8**	**9.19**
Venous infarct/ CVT	**47**	**54.02**
PRES	**14**	**16.09**
Normal	**8**	**9.19**

Patients were subjected to CT scan (plain and contrast) and MRI with /without MR Venography.

- Of 87 patients, 47 (54.02%) showed features of venous infarct/ CVT. Among them, 7 patients had normal CT findings and with the help of MRI they were diagnosed to have CVT.
- 16.09% of patients had findings suggestive of PRES.
- 8 patients with typical clinical features of CVT had normal findings in imaging studies.
- 11.49% patients had Intra Cranial Haemorrhage which is a worse prognostic factor

Figure 15


NEURO IMAGING FINDINGS
60
50
40
30
20
10
0
54.02
11.49
9.19
16.09
9.19
Venous infarct/CVT
Haemorrhage
Arterial infarct
PRES
Normal

SIGNS OF CVT IN NEURO IMAGING:

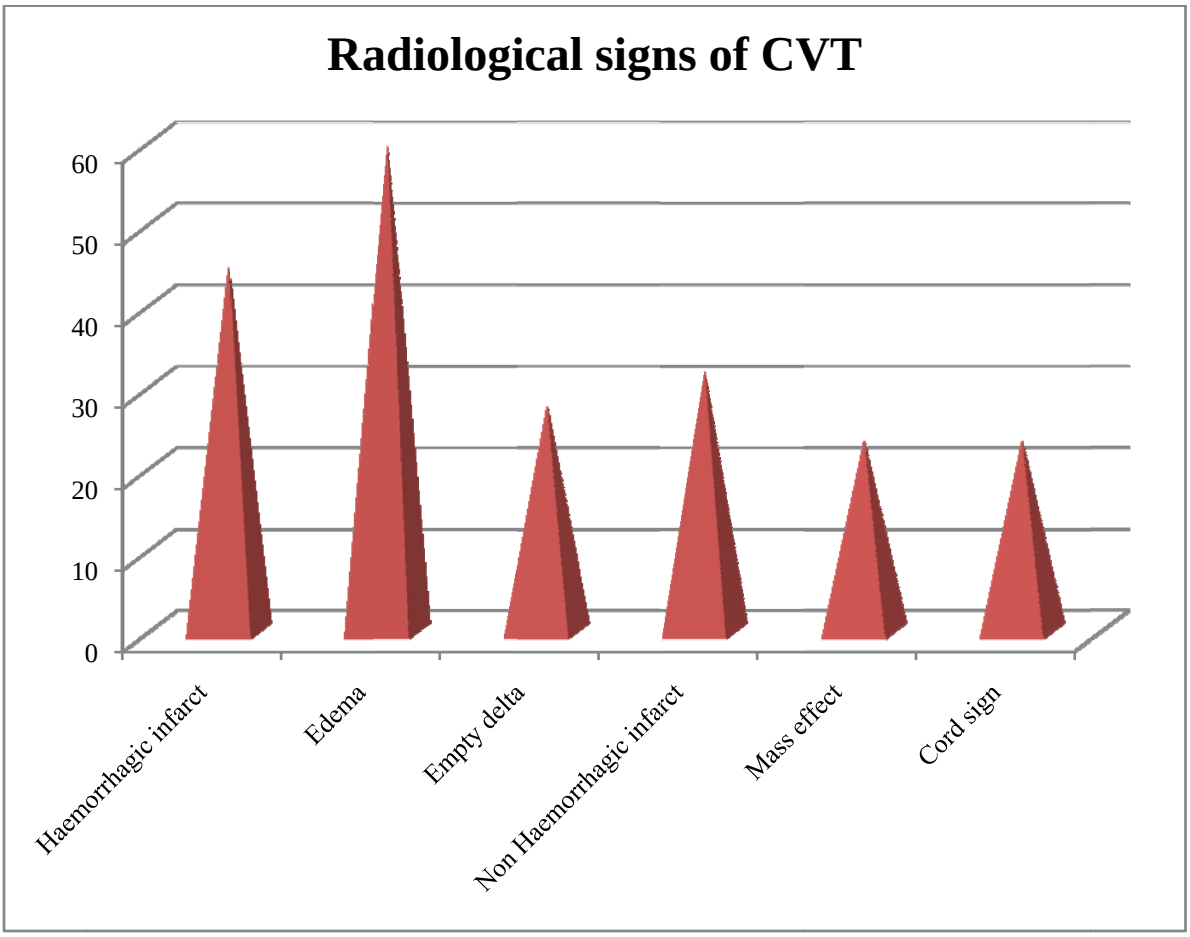
Total patient with findings suggestive of CVT: 47

Table 16

FINDINGS	NO OF PATIENTS	PERCENTAGE
Haemorrhagic infarct	**21**	**44.68**
Edema	**28**	**59.57**
Empty delta sign	**13**	**27.65**
Non haemorrhagic infarct	**15**	**31.91**
Cord sign	**11**	**23.40**
Mass effect	**11**	**23.40**

Total patients with clinical features of CVT were 55.Among them 8 patient had normal CT finding. Of the 47 patients with findings of CVT, 59.57% had cerebral edema. Considering the specific findings of CVT, most common feature was Haemorrhagic infarct with 44.68% followed by Non haemorrhagic infarct with 31.91%. Empty delta sign was present in 27.65% patients. Mass effect with midline shift was present in 23.40% of patients which indicates bad prognosis.

Figure 16


Radiological signs of CVT
60
50
40
30
20
10
0
Haemorrhagic infarct
Edema
Empty delta
Non Haemorrhagic infarct
Mass effect
Cord sign

SINUSES INVOLVED IN CVT

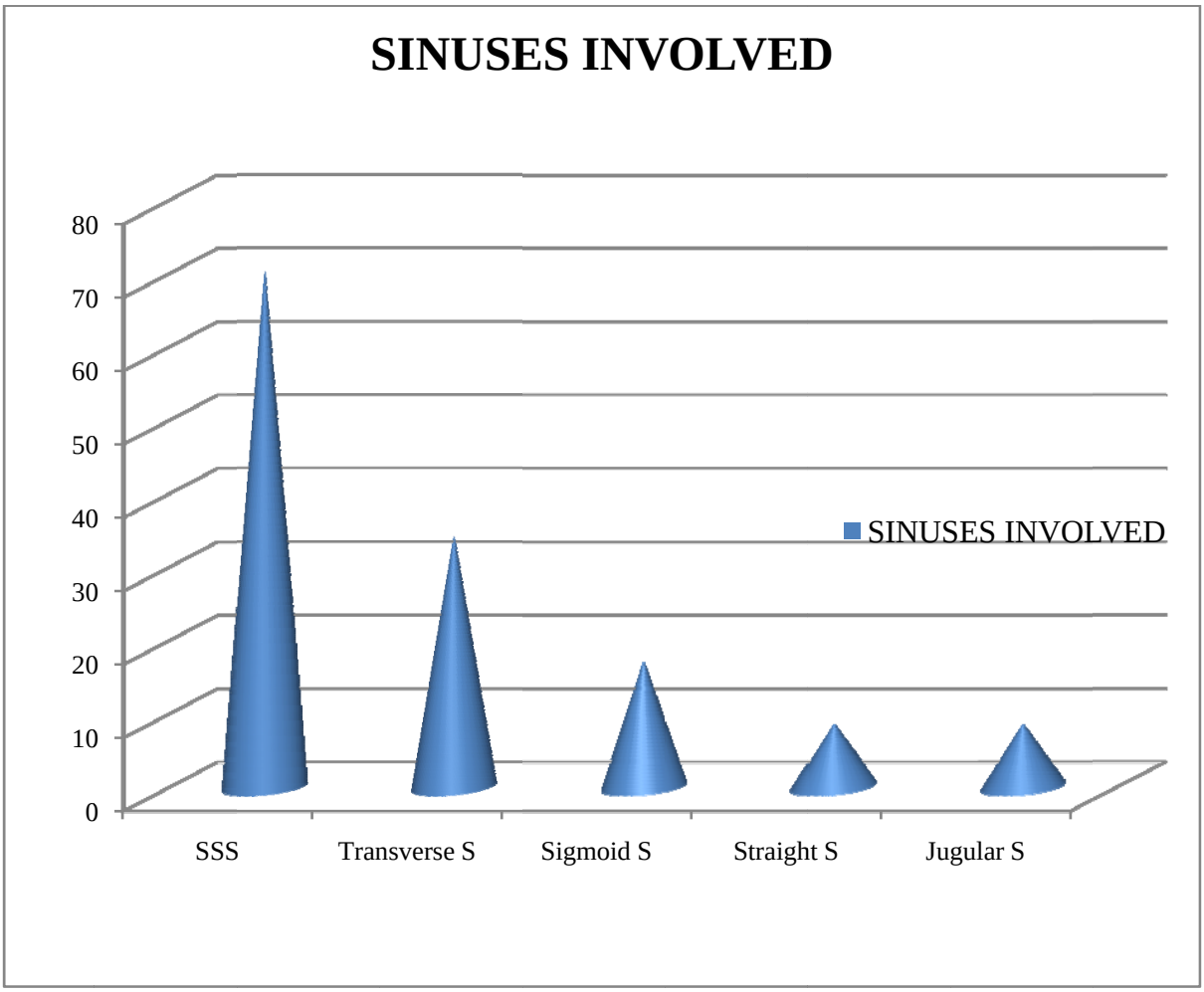
TOTAL PATIENTS: 47

Table 17

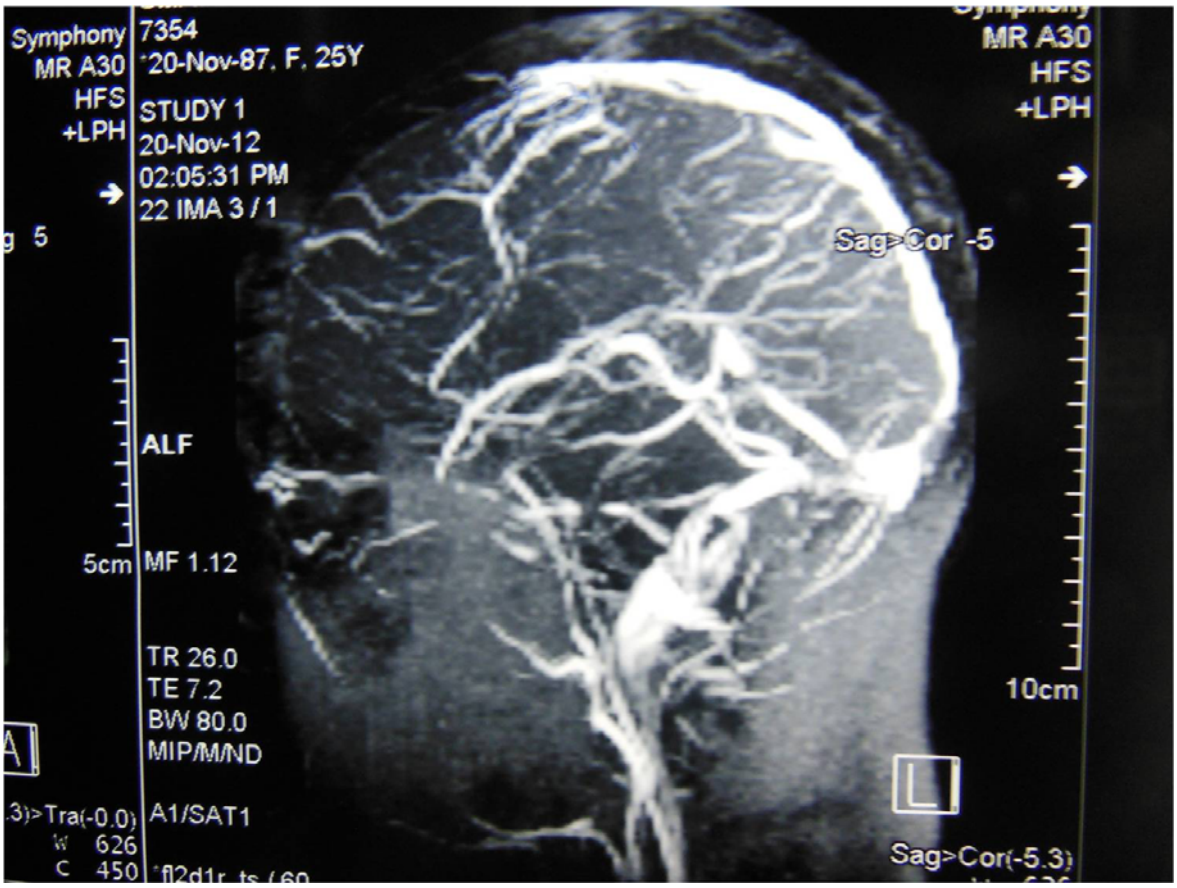
SINUS INVOLVED	NO OF PATIENTS	PERCENTAGE
Superior sagittal sinus	**33**	**70.21**
Transverse sinus	**16**	**34.04**
Sigmoid sinus	**8**	**17.02**
Jugular sinus	**4**	**8.51**
Straight sinus	**4**	**8.51**

Total patients diagnosed to have CVT were 55. Among them, 47 patients had features of CVT in CT/MRI/MRV. Of all the patients with findings of CVT, most common sinus involved was Superior Sagittal Sinus with 70.21% followed by the transverse sinus with 34.04%

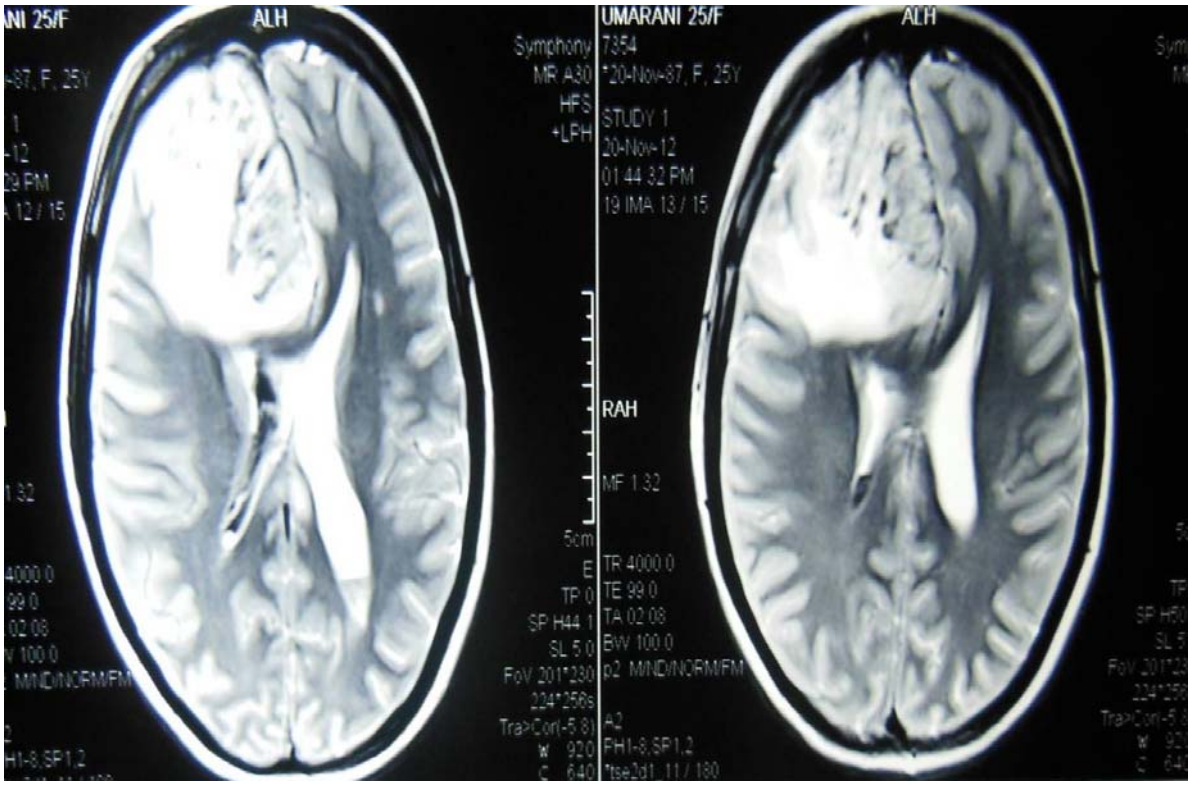
Figure 17


SINUSES INVOLVED
80
70
60
50
40
30
20
10
0
SINUSES INVOLVED
SSS
Transverse S
Sigmoid S
Straight S
Jugular S

MRV Showing filling defect in SSS



Haemorrhagic infarct with mass effect



OUT COME OF THE DISEASE

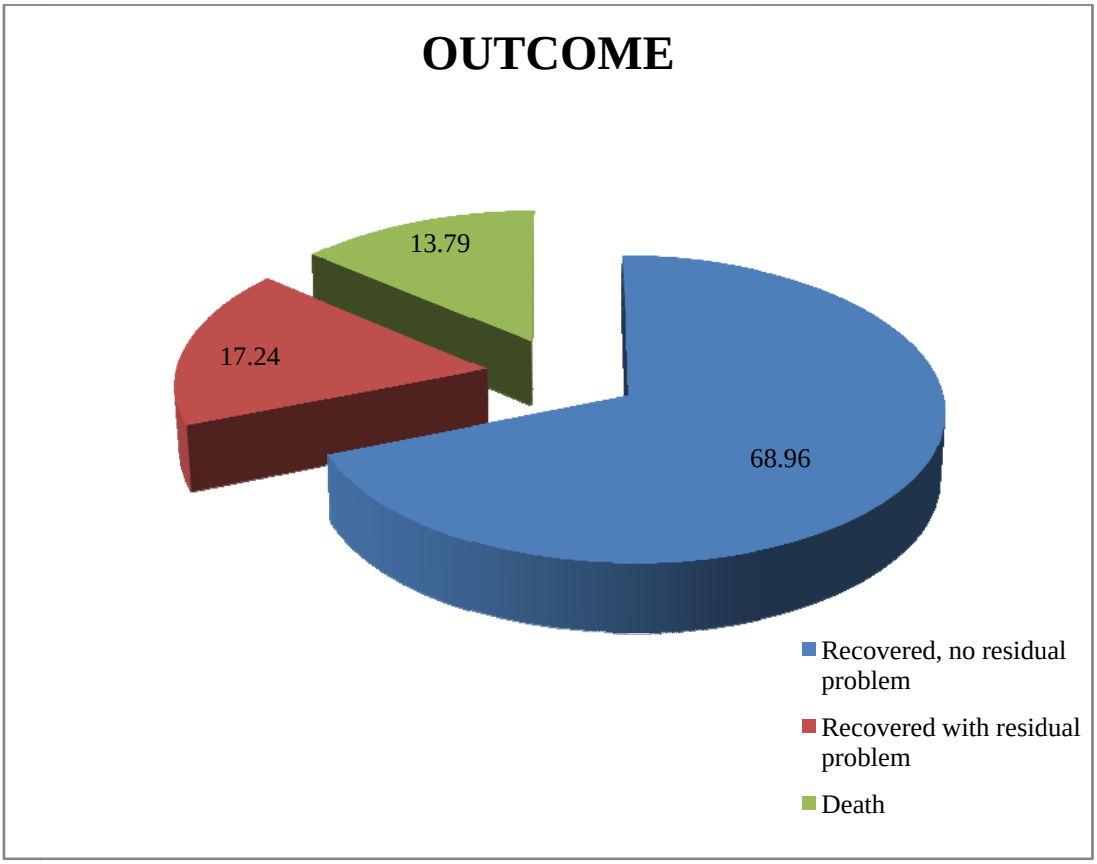
Table 18

Total patients: 87

OUTCOME	NO OF PATIENTS	PERCENTAGE
Recovered without residual problem	**60**	**68.96**
Recovered with residual problem	**15**	**17.24**
Death	**12**	**13.79**

We observed that 68 96% of patients showed good prognosis in the form of recovery without residual problem. Prognosis was good in patients who showed features of PRES and Venous infarct. We found that 17.24% of patients had recovered with residual deficit. The mortality was 13.79%.

Figure 18


OUTCOME
13.79
17.24
68.96
Recovered, no residual problem
Recovered with residual problem
Death

MATERNAL MORTALITY

Total Maternal Death in TVMCH in the study period (22 months): 87

Maternal Death due to CVA: 12 [13.79%]

Patients with CVT showed less mortality compared to other problems like ICH and Arterial infarct.

Of 12 patients, 50% had Hypertensive disorders either in the form of pre eclampsia or chronic hypertension

Considering the intra cranial pathology, 41.5% had Intra cranial haemorrhage.

Of 12 patients, 5 patients had Cerebral Venous Thrombosis and all of them showed haemorrhagic infarct.

DISCUSSION

The occurrence of Cerebro Vascular Accident during peripartum period is a challenging, diagnostic and management problem. Since pregnancy and puerperium are pro coagulant states, women are at greater risk of Thrombo Embolic Diseases in this period.[3] More than 50% young strokes in Indian women are related to pregnancy and puerperium[14]. Ischaemic strokes are more common than haemorrhagic strokes. Cerebral Venous Thrombosis leads as first cause for stroke in pregnancy and puerperium. Bansal *et al* in India reported puerperal CVT in a frequency of 4.5/ 1000 obstetric admissions.[53] CVT has the maximum incidence in the puerperal period.

Whenever a risk factor like haematological abnormalities, immunological abnormalities, infections, dehydration ,pre eclampsia occur over the pre existing hyper coagulable state there is higher risk for Cerebro Vascular Accidents especially CVT. Unbooked status and home delivery have been proposed by many authors previously as important contributing factors. Nowadays these risk factors have been reduced due to implementation of our National health programmes.

The reported mortality due to CVT ranges from 5% to 30% which varies according to the time of detection and initiation of treatment.[11] Neuro imaging

in the form of CT or MRI should be done at the earliest, after the onset of symptoms.

This study was carried out to evaluate the risk factors, spectrum of clinical presentation and outcome of Cerebral Vascular Accidents during pregnancy and puerperium. Due to unavailability we were not able to do all the investigations. We had 87 patients who were diagnosed to have CVA in pregnancy and puerperium in a period of 22 months from January 2011 to October 2012. They were investigated to the maximum possibility, analyzed and the following observation was noted.

AGE DISTRIBUTION:

Highest incidence of CVA occurred in the age group of 19-25 years. It contributes to 55.17% of total cases. This result is comparable to the results of the study conducted by Ameri *et al*., in 1992 with 61%.[3,6] Lanska et al has published a report mentioning that 15-25 yr age group is more vulnerable to obstetric stroke.[50] Age groups 26-30 yr and 31-35 yr had similar incidences 17.24%. Youngest age of incidence was 17 years and the patient was a case of Takayasu arteritis. Incidence among> 36 yr age group may appear low but the mortality was high (40%), when compared to lower age groups.

PARITY:

Among 87 patients, maximum incidence was noted in primi gravida and primi para which was 43.67%. G_2 group which includes second gravida and second para showed the incidence of 27.58%. The rate of grand multiparity has

been reduced now a days which explains low incidence of CVA in grand multipara.

SOCIO ECONOMIC STATUS

Maximum occurrence of CVA was noticed in Class IV and V socio economic states, which was 70.10%. This may be because most of the patients admitted to Tirunelveli Medical Collage Hospital belong to lower socio economic classes. Another explanation is that risk factors like anemia, poor nutrition, unbooked status and lack of awareness are more prevalent in lower socio economic classes. Prakash BC and Bansal C mentioned that reasons for higher incidence of CVA in lower socio economic groups need to be researched further.[53]

AREA OF DISTRIBUTION

Among 87 patients with pregnancy related CVA 71.26% of patients are from rural area. This may be because of delayed identification of risk factors due to illiteracy and lack of awareness and poor accessibility to health care system. Wrong beliefs like post partum water restriction is prevalent in rural areas of Tirunelveli.

TIME OF PRESENTATION

In our study 80% of CVA occurred in the post partum period. This is comparable to the results of study by Jeng JS *et al*., which is 73%.[15] More than half of post partum CVA had occurred at II week of postpartum [44.82%]. Prakash BC and Bansal C have reported that postpartum CVA usually occurs 7-

10 days after delivery.[53] Among 20% of antepartum strokes most of them had PRES and higher occurrence was noticed during third trimester [13.79%]. One patient, who is a case of Systemic Lupus Erythematosus with renal failure, had stroke in first trimester.

MODE OF DELIVERY

In the present study, 55.71% of patients with puerperal CVA had undergone caesarean section for various reasons. One patient had Subtotal Hysterectomy due to atonic PPH. Lanska DJ and Kryscio RJ mention that Caesarean section is an independent risk factor for obstetric CVT as it increases the risk by 3 times.[16] Another explanation is that most of these patients had Caesarean sections for the indications like pre eclampsia, diabetes and elderly primi which are proven risk factors for CVT. Instrumental delivery and caesarean section may increase the risk due to post surgical decline in protein C level because of surgically induced tissue damage.[1] They also lead to prolonged immobilization which causes reduced blood flow to the legs resulting in venous stasis.

PREDISPOSING FACTORS

CVA in pregnancy is usually a multi factorial disorder. When two or more risk factors co exists in a patient, the chance for CVA is high. In this study we found that Anemia [62.06%], Hypertensive disorders [44.82%], Caesarean delivery [44.82%], Dehydration [27.58%] and Transfusion [11.49%] are the important predisposing factors for CVA. Sharshar T *et al.*, has mentioned that

pre eclampsia and eclampsia are responsible for 25% to 45% of pregnancy related CVA. Brown *et al* found that women with preeclampsia were 60% more likely to have ischemic stroke than women without it.[30]

Independent risk factors for CVT are the peripartum period, caesarean delivery, increasing maternal age, hyper emesis, infections, transfusions and maternal hypertension[16]. In Tirunelveli, people have a custom of severe water restriction in the postpartum period due to some wrong beliefs. It increases the risk of CVT when patients have other co morbid conditions like anemia, pre eclampsia, immunological abnormalities and fever.

CLINICAL FEATURES

1. HEADACHE:

We observed that headache was the most common symptom which usually precedes the neurological event. In our study 67.81% of our patients presented with headache at the time of admission which is comparable to the study by Kumar S *et al* with 66% and Nagaraj *et a*l with 71%.[63] Convulsions and Altered sensorium are the next two common symptoms with 59.77% and 56.32% respectively. The inference is that patients with severe, persistent headache during peripartum period should not be left unevaluated even in the absence of other neurological features.

2. LEVEL OF CONSCIOUSNESS:

In our study we observed that 43.67% were conscious at the time of admission and during the disease course. 56.32% of patients presented with

altered sensorium. Of those patients with altered level of consciousness 42.5% of patients were drowsy, 9% were stuporous and 4.5% were comatose. This result is comparable with Nagaraj *et al* from Tamilnadu who has reported altered sensorium in obstetric stroke as 57.53%. Neki S *et al* has reported 56% of altered sensorium in pregnancy related CVA.

3. SEIZURES:

Fifty percent of patients with CVT have either focal or generalized seizures. They may be localized at the onset but may later become generalized. Rarely may they persist after an acute phase is over. Their early appearance is the hallmark of bad prognosis. In the present study 60% of patients presented with convulsions either at the time of admission or during hospital stay. It is similar to the report by Kumar S *et al* who has observed 66% in 85 patients. Of 52 patients with seizures in our study, 78.84% was Generalized Tonic Clonic Seizures and 21.15% was Focal seizures.

4. NEUROLOGICAL SIGNS:

In the present study 31.03% of patients had neurological deficits like hemiparesis/hemiplegia and cranial nerve palsies. This is comparable to the results of study by Bousser *et al* which is 35%.[57] Focal neurological deficit comprises hemiparesis usually with facial sparing (as “face area” in cerebral cortex is drained by sylvian vein which is a tributary of cavernous sinus) and lower limb more severely affected than upper limb.

5. FEVER:

In our study 32.18% of patients had fever either before or at the onset of stroke. Infection and fever are one of the important predisposing factors for CVT. When deep cerebral veins are affected patients may have high grade fever due to involvement of Pons.[56] One of the patients in our study had large brainstem infarct and persistent hyper pyrexia.

6. VISUAL DISTURBANCES:

Among the 87 patients studied 12.64 % had visual disturbances in the form of blurring of vision, diplopia or transient loss of vision. 6 Th cranial nerve compression due to raised intra cranial pressure can lead to lateral rectus palsy and diplopia.[1] Posterior Reversible Encephalopathy Syndrome causes transient loss of vision.

7. BLOOD PRESSURE:

We observed that 45% of patients with obstetric CVA suffered from Hypertensive disorders. Of them, 31.03% had blood pressure >/= 140/90 and < 160/110. Only 13.79 % of the patients had BP >/= 160/110. James *et al* have reported that only 20% of cases with CVA related to Pre Eclamptic Toxemia exhibit sustained diastolic pressures of > 105 mm Hg before the event of stroke.[6]

INVESTIGATIONS

ANAEMIA:

Brig Kumaravelu S *et al* has observed in 2008 that haematological abnormalities especially anemia is an important predisposing factor for peripartum CVA.[77] In our study 62.06% of patients were anemic. Among them 24% were severely anemic with haemoglobin level < 7g/dl. Risk factors like pre eclampsia, infection and transfusion are prevalent in patients with low haemoglobin levels and this explains the increased incidence of CVA in patients with anemia.

RADIOLOGICAL FINDINGS:

Radiological investigations were carried out for all patients. 54% had findings suggestive of CVT. 16% had findings suggestive of Posterior Reversible Encephalopathy Syndrome. Eight patients had typical clinical feature of CVT but imaging studies were normal. Sirinivasan K has encountered 50 cases of severe CVT among 10000 deliveries. It has been estimated that the prevalence rate in developing countries is approximately 10 times more than that in developed countries. Among those with CVT, 44.68 % had Hemorrhagic infarct and 31.91% had Non hemorrhagic infarct. Similar observations were noted by Dixit et al 48.4% and 32% respectively[3]. Empty Delta sign, presented in 27% indicates Superior Sagittal Sinus involvement.11 patients had mass effect with midline shift which is a sign of bad prognosis.

SINUSES INVOLVED:

In Magnetic Resonance Venogram hyperintense area was noted in the affected sinuses instead of hypointense flow void. MR Angiogram showed filling defect due to mass effect caused by hemorrhage/infarct. In Cerebral Venous Thrombosis, we observed that Superior Sagittal Sinus is the most common site of involvement.70.21 % patients had SSS involvement followed by Transverse sinus with 34.04 %. Similar observations were obtained by Strolz E et al with 72.2% and Ameri et al with 72%.

MANAGEMENT:

All the patients with CVA admitted in Intensive Care Unit. Multi disciplinary treatment was made involving the Obstetrician, Physician, Neurophysician, Radiologist and Physiotherapist.

Unfractionated Heparin was given in a dose of 5000 units subcutaneously thrice daily for patients with Arterial infarct or CVT. Anti convulsants, anti edema measures and anti hypertensives were given in an appropriate manner. Coagulation profile was monitored periodically before and during heparin therapy.

Measures were taken to prevent aspiration, bed sores and infections. The risk factors like anemia, dehydration and fever were corrected simultaneously. After the acute phase, stable patients were shifted to ward and anti coagulation was maintained with Tablet. Warfarin.

OUTCOME:

69% of patients recovered without neurological deficit within four weeks. 17.24% of patients had neurological deficits like hemiparesis. Patients with CVT/ venous infarct had good outcome in the form of complete recovery. Residual defects were noted in patients with haemorrhage and arterial infarcts. The mortality rate was 13.79%

MORTALITY:

Total Maternal mortality in Tirunelveli Medical College Hospital during the study period was 87, out of which 12 deaths (13.79%) have occurred due to Cerebral Vascular Accidents. Maximum deaths occurred due to intra cranial hemorrhages which accounts for 41.5% of total deaths due to CVA. We observed that all 5 patients who died of CVT had haemorrhagic infarct. Daif A *et al* and Treadwell SD *et al* have mentioned haemorrhagic infarct and empty delta sign as poor prognostic indicators.[1,76] In the present study we observed that higher maternal age, Hypertensive disorders, Haemorrhagic infarct and Intra cranial haemorrhage as poor prognostic factors.

Bousser MG has reported mortality rate of 5-30% due to obstetric CVA which varies according to the time of intervention[57]. The mortality from all-cause CVT is 3–10%, though the rate is significantly low for pregnancy related CVT. When deaths occur, they usually result from Trans tentorial herniation or intracranial haemorrhage.

SUMMARY

Observations obtained through our study are summarized below.

- Highest incidence of stroke was noted in the 18-25 year age group with 55.17% .
- Maximum occurrence of CVA was noted in Primi gravida and Primipara with 43.67%
- 70% of Cerebral Vascular Accidents occurred in lower socio economic group (Class IV &V)
- 71.26% of patients belong to rural background
- Incidence of CVA was higher in the postpartum period (80.45%) , specifically in the second week of postpartum which was 44.82%
- 55.71% of patients with postpartum CVA had undergone LSCS.
- Anemia, as a risk factor, was noted in 62.06% of patients. Second common risk factor was Hypertensive disorders which was present in 44.82%.
- Most common clinical feature was headache followed by convulsions
- More than half of the patients (56%) had altered sensorium
- Generalized seizures are more common than Focal seizures
- In the present study most common cause for pregnancy related stroke was CVT accounting for 63%
- In CVT, Superior Sagittal Sinus thrombosis was the most common pathology, noted in 72.72%

- Complete recovery was noted in 68.96% of patients and PRES and Venous infarct had good prognosis. The mortality was 13.79%. Patients with ICH or Haemorrhagic infarct had high mortality.

CONCLUSION

CVA is one of the serious complications of pregnancy and puerperium and it is a recognized cause for maternal mortality. It has a wide spectrum of clinical presentation.

CVT is the commonest cause for pregnancy related 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.

Classical symptoms of CVT are headache, convulsions and altered sensorium. Investigations should be aimed at identification of not only the diagnosis also the etiology like thrombophilias. The ideal imaging modality for CVT is MRI with MR Venography.

Patients with arterial or venous infarct are treated with Heparin followed by oral anti coagulants. Of all the patients with pregnancy related CVA, patients with CVT have better prognosis except those with haemorrhagic infarct.

SUGGESTIONS

- People should be made aware of the risk factors and early symptoms of CVA.
- 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 in our day to day practice.
- Patients with higher chance for CVA should be referred to the tertiary care center without delay.
- At the onset of symptoms, treatment should be initiated at the earliest.
- Postpartum headache deserves prompt and focused evaluation.
- Specific protocols may be designed to reduce the devastation caused by stroke related to pregnancy and puerperium.

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PROFORMA

CVA IN PREGNANCY AND PUERPERIUM

HISTORY :

Name : Age :

IP No. : Address :

Contact number:

Socio economic status :

Time of presentation : Antenatal / Postnatal

If AN, LMP: EDD: Gestational Age :

If PN, Mode of deliver :

Postnatal day:

Significant AP/IP/PP History:

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/ MR Venogram-

Other Investigations –

DIAGNOSIS:

TREATMENT:

OUTCOME:

MASTER CHART

S.NO	NAME	AGE	1P.NO	SOCIOECONOMIC STATUS	AREA	OBS.CODE	TIME OF PRESENTATION	GA	MODE OF DELIVERY	PP WEEK	RISK FACTORS	SYMPTOMS	BP	CONSCIOUSNESS	SEIZURES	TEMPERATURE	FUNDUS	Hb LEVEL	RADIOLOGICAL FINDING	CVT FEATURE	SINUS INVOLVED	OUTCOME
1	Mariammal	26	2460	IV	R	P1L0	PP	NA	LN	II WK	PE/AN	H/C/AS/NS	140/90	Stuporous	GTCS	AF	P	7	ICH	NA	NA	RD
2	sundari	20	3007	IV	R	Primi	AP	34 WK	NA	NA	Severe PE	H/C/F/V	150/110	Conscious	GTCS	F	N	10	PRES	NA	NA	RND
3	Panneerselvi	21	3989	V	R	P1L1	PP	NA	Instrumental	I WK	AN/BT	C/AS/ND	120/80	Drowsy	Focal	AF	N	4.6	Normal	NA	NA	RND
4	devi	28	5977	V	U	P3L2	PP	NA	LN	II WK	AN	H/AS/F/NS	110/80	Drowsy	NA	F	N	7		NHI/CS	TS/SG	RND
5	Jayalakshmi	23	6705	V	R	P2L2	PP	NA	LN	III WK	PE/DH	C/AS/ND/NS	140/90	Comatose	GTCS	AF	P	9	VI/CVT	HI/ME/E	SS/TS/ST	D
6	mumtaj	30	8907	V	R	P3L3	PP	NA	LN	II WK	AN/ BT	H/AS/F	120/80	Drowsy	NA	F	N	3.6	VI/CVT	HI/E	SS	RND
7	mariasudha	21	10232	III	R	P1L1	PP	NA	LSCS	II WK	AN/DH	H/VD/AS	100/70	Drowsy	NA	AF	N	6.8	VI/CVT	NHI	SS	RND
8	santhanamari	21	11765	IV	R	G2A1	AP	32 WK	NA	NA	PE	H/C/AS/V	150/100	Drowsy	GTCS	AF	P	10.2	ICH	NA	NA	RD
9	Kanniammal	25	9671	V	U	P2L3	PP	NA	LN	I WK	Twin	C/ND	130/80	Conscious	GTCS	AF	N	10.2	VI/CVT	NHI/CS/E	SS	RND
10	Prema	18	10879	V	R	Primi	AP	28 WK	NA	NA	Severe PE	H/V/VD	150/110	Conscious	NA	AF	AS	11	AI	NA	NA	RD
11	Sivakami	22	12430	II	U	P1L1	PP	NA	Instrumental	IV WK	GHT	H/VD/ND	140/100	Conscious	NA	AF	N	10.5	PRES	NA	NA	RND
12	Muthumari	35	11005	V	R	P4L4	PP	NA	LSCS	II WK	CHT/AN	C/F/AS/V	140/90	Drowsy	GTCS	F	N	7.9	PRES	NA	NA	RND
13	Magesh	21	11915	III	U	P2L2	PP	NA	LN	III WK	AN/F	AS/F/ND	100/70	Comatose	NA	F	N	8	AI	NA	NA	D
14	Shanmugavadivu	41	17446	V	R	P3L2	PP	NA	LSCS	II WK	PE/PPH/STH/ BT	H/C/AS/NS	140/90	Stuporous	GTCS	AF	N	11	VI/CVT	HI/ME/E	SS/SG/ST	D
15	Ponmalar	24	17993	V	R	P1L1	PP	NA	LSCS	I WK	AN	H/C/AS	120/70	Drowsy	GTCS	AF	N	7.9	Normal	NA	NA	RND
16	Jasmine	21	18108	IV	R	P1L1	PP	NA	LN	III WK	AN	H/ND	110/70	Conscious	NA	AF	N	6.9	AI	NA	NA	RD
17	Valli	32	20086	V	R	P3L3	PP	NA	LN	II WK	AN/BT	AS/F/NS	110/70	Drowsy	NA	F	N	3.9	VI/CVT	NHI	TS	RND
18	Zareena	19	21254	IV	U	P1L1	PP	NA	LSCS	II WK	PE/AN	H/C/AS	140/100	Drowsy	GTCS	AF	P	8.5	ICH	NA	NA	RD
19	Subbulakshmi	30	22085	III	R	P2L2	PP	NA	LSCS	I WK	AN	H/C/F/V	120/70	Conscious	Focal	F	P	6.5	VI/CVT	HI/E	SS	RND
20	Kavitha	25	22294	V	U	P1L1	PP	NA	Instrumental	IV WK	GDM	H/C/AS	120/80	Drowsy	GTCS	AF	N	13	VI/CVT	NHI/ME/E	SS	RND
22	Mariammal	32	25438	V	R	P3L3	PP	NA	LN	IV WK	AN/DH	H/C/F/ND	110/70	Conscious	Focal	F	N	8	VI/CVT	ED/E	SS/SG	RND
23	Arumugakani	38	26077	IV	R	P4L4	PP	NA	LSCS	II WK	AN/BT	AS/NS	110/70	Drowsy	NA	AF	N	5	VI/CVT	HI	TS	RND

24	Lakshmi	22	29854	III	U	P1L1	PP	NA	LSCS	I WK	DH	H/C/ND	120/70	Conscious	Focal	AF	N	12.1	Normal	NA	NA	RND
25	Mythili	24	30921	V	R	P1L1	PP	NA	LSCS	II WK	F/DH	H/AS/F	100/70	Drowsy	NA	F	N	10.3	VI/CVT	ED/E	SS/JS	RND
26	Parameshwari	28	32756	V	R	G2P1L1	AP	34 WK	NA	NA	AN	C/AS/F/NS	130/80	Drowsy	GTCS	F	N	7	ICH	NA	NA	RD
27	Kavipriya	23	35490	III	R	P2L2	PP	NA	Instrumental	II WK	PPH/BT/DH	H/C/V	110/70	Conscious	GTCS	AF	N	10.7	VI/CVT	HI/ME/E	SS	RND
28	Meena	17	36054	I	U	Primi	AP	24 WK	NA	NA	TA	ND/NS	120/80	Conscious	NA	AF	N	11	AI	NA	NA	RD
29	Muthuselvi	24	39551	IV	R	P1L2	PP	NA	LSCS	II WK	Twin/PE	H/C/AS	130/100	Stuporous	GTCS	AF	N	12.2	VI/CVT	ED/CS/E	SS	RND
30	Kannimariyal	34	41257	V	U	P2L2A1	PP	NA	LSCS	II WK	AN/DH	AS/ND	110/70	Drowsy	NA	AF	N	8	VI/CVT	ED/E	SS	RND
31	Grace	24	44872	V	R	P1L1	PP	NA	LSCS	II WK	PPH/BT	AS/F/V	100/70	Drowsy	NA	F	N	11	VI/CVT	NHI	TS/ST	RND
31	Poomala	25	23448	IV	R	Primi	AP	12 WK	NA	NA	SLE Flare/RF	ND/F	120/80	Conscious	NA	F	N	10.1	AI	NA	NA	D
32	Thavamani	26	48220	IV	R	P1L1	PP	NA	LSCS	III WK	AN	H/C/NS	110/80	Conscious	GTCS	AF	N	8.6	VI/CVT	HI/ME/E	SS	RND
33	Mariammal	35	50023	V	R	P3L3	PP	NA	LN	II WK	CHT/AN	H/F/V	150/90	Conscious	NA	F	AS	7.4	AI	NA	NA	RD
34	Vijayarani	29	51389	I	U	G4P1L1A2	AP	26 WK	NA	NA	SeverePE/F	C/AS/F	160/100	Stuporous	GTCS	F	P	11	ICH	NA	NA	D
35	Niraimathi	31	52431	IV	R	P3L2	PP	NA	LSCS	II WK	AN/DH	H/C	110/70	Conscious	GTCS	AF	N	8	VI/CVT	NHI/E	TS	RND
36	Manjula	18	54882	III	U	P1L1	PP	NA	LN	II WK	AN/GHT	H/C/NS	150/90	Conscious	GTCS	AF	N	8.8	Normal	NA	NA	RND
37	Esakkiammal	21	55012	V	R	P2L2	PP	NA	LN	IV WK	AN/BT	AS/F	110/70	Drowsy	NA	F	N	5.8	VI/CVT	HI/CS	TS	RND
38	Palanimuthu	27	56301	IV	R	P3L3	PP	NA	LN	I WK	PE/AN	H/C/V/ND	140/90	Conscious	Focal	AF	P	9	VI/CVT	HI/ME/E	SS/SG	D
39	Muthumari	31	59350	V	R	G4P1L1A2	AP	35 WK	NA	NA	PE/AN	H/C/V	150/90	Conscious	GTCS	AF	N	6.6	PRES	NA	NA	RND
40	Deepa	20	60328	II	U	P1L2	PP	NA	LSCS	II WK	Twin/PE/DH	H/VD/ND	140/110	Conscious	NA	AF	N	10.4	PRES	NA	NA	RND
41	Piramachi	25	7036	IV	R	P1L1	PP	NA	Instrumental	II WK	PE/AN/DH	H/C/AS/ND	140/100	Comatose	GTCS	AF	N	8.8	VI/CVT	HI/ME/E	SS/SG/JS	D
42	Sudha	21	9821	V	U	Primi	AP	32 WK	NA	NA	SeverePE/AN	C/ND/AS	160/100	Drowsy	Focal	AF	N	8	PRES	NA	NA	RND
43	Annaselvi	24	13058	III	R	P1L1	PP	NA	LN	II WK	AN/DH	H/C/AS/NS	110/80	Drowsy	GTCS	AF	N	7.8	VI/CVT	NHI/E	TS	RD
44	Pandiselvi	28	19236	V	R	P2L2A1	PP	NA	LSCS	II WK	PE	H/AS	140/90	Drowsy	NA	AF	N	10.1	PRES	NA	NA	RND
45	Rajeshwari	22	20621	II	R	P2L1	PP	NA	LSCS	I WK	AN	H/C/ND	110/70	Conscious	GTCS	AF	N	7.4	VI/CVT	ED/CS	SS	RND
46	Ramalakshmi	30	22979	IV	R	P4L3	PP	NA	LSCS	II WK	SeverePE	AS/F/V/ND	160/100	Stuporous	NA	F	N	10.7	ICH	NA	NA	D
47	Suganthi	33	23011	III	U	P3L3	PP	NA	LN	II WK	AN	H/F/V/NS	120/80	Conscious	NA	F	P	7	VI/CVT	ED	SS/SG	RND
48	Lathamary	23	23894	V	R	P2L2	PP	NA	LSCS	III WK	AN/DH	H/C/NS	100/70	Conscious	GTCS	AF	N	7.4	VI/CVT	NHI/CS	SS	RD

49	Ushadevi	20	24145	IV	U	P1L1	PP	NA	LSCS	I WK	PE/DH	H/C/AS/V	130/100	Drowsy	Focal	AF	N	10.2	VI/CVT	HI/E	TS	RND
50	Juliet	27	25379	III	U	G2P1L1	AP	36 WK	NA	NA	PE/AN	H/C/AS	140/100	Drowsy	GTCS	AF	N	8.8	PRES	NA	NA	RND
51	Anitha	20	25870	V	R	P1L1	PP	NA	LSCS	II WK	AN/ HD/DH	ND/VD	120/60	Conscious	NA	AF	N	8.9	Normal	NA	NA	RND
52	Thangaselvi	31	26232	IV	R	G2A1	AP	34 WK	NA	NA	SeverePE	H/C/AS/NS	160/110	Stuporous	GTCS	AF	P	11	ICH	NA	NA	RD
53	Mariammal	25	27399	V	R	P1L1	PP	NA	Instrumental	I WK	AN/DH	H/C/AS/ND	120/70	Drowsy	Focal	AF	N	7	VI/CVT	NHI/ME/E	SS	RD
54	Banu	33	27883	III	U	P3L4	PP	NA	LN	II WK	Twin/AN	H/C/F/V	130/80	Conscious	GTCS	F	N	8.6	VI/CVT	ED/CS	SS	RND
55	Mallika	23	28001	II	U	P2L0	PP	NA	LSCS	I WK	Severe PE	H/C/AS/NS	150/100	Drowsy	GTCS	AF	N	11.2	PRES	NA	NA	RND
56	Muneeshwari	21	28562	V	R	P1L1	PP	NA	LSCS	II WK	AN	H/C/ND	120/80	Conscious	Focal	AF	N	7.8	VI/CVT	HI/CS/E	SS/JS	RND
57	Chitra	22	28992	IV	R	P2L1	PP	NA	LSCS	III WK	SeverePE/AN	H/V/F	160/100	Conscious	NA	F	N	8	ICH	NA	NA	D
58	Poomari	31	29034	III	R	P3L3	PP	NA	LN	II WK	AN/BT	AS/ND	110/70	Drowsy	NA	AF	N	2.8	AI	NA	NA	RD
59	Devipriya	18	29870	V	R	Primi	AP	37 WK	NA	NA	PE/AN	H/C/AS/V	140/100	Drowsy	GTCS	AF	P	7.4	VI/CVT	HI/ED	SS	RND
60	Krishnammal	22	30104	IV	R	P2L2	PP	NA	LSCS	II WK	AN/BT	H/C/F/V	120/80	Conscious	GTCS	F	N	5.8	VI/CVT	ED/E	SS	RND
61	Muppidathi	36	31289	V	R	P4L3	PP	NA	LSCS	I WK	AN	H/C/F/NS	120/80	Conscious	GTCS	F	N	8	VI/CVT	NHI/E	TS	RND
62	Chinnakkannu	27	31873	IV	R	P1L1	PP	NA	LSCS	II WK	AN	H/C/ND/NS	120/80	Conscious	GTCS	AF	N	8.2	Normal	NA	NA	RND
63	Gomathi	23	32010	III	U	G2P1L1	AP	36 WK	NA	NA	PE	H/C/VD	150/90	Conscious	GTCS	AF	N	12.3	PRES	NA	NA	RND
64	Parameshwari	39	32313	V	R	P4L2	PP	NA	LN	I WK	DM/PE/ IUD	AS/NS/ND	140/100	Comatose	NA	AF	N	10.1	ICH	NA	NA	D
65	Aabithabegum	25	34187	II	U	P1L0	PP	NA	LSCS	II WK	AN/PE/DH	H/C/NS	150/100	Conscious	GTCS	AF	N	7	VI/CVT	HI	TS/JS	RND
66	Geetha	27	35087	IV	R	P2L2	PP	NA	LN	I WK	AN/DH	H/C/AS	110/70	Drowsy	GTCS	AF	N	7.2	VI/CVT	HI/E	TS	RND
67	Helen	25	37284	V	R	P1L1	PP	NA	LSCS	I WK	AN/DH	AS/V	110/70	Drowsy	NA	AF	N	8	VI/CVT	NHI/CS	SS	RND
68	Padma	23	38871	IV	R	G2A1	AP	37 WK	NA	NA	PE	H/C/F/VD	140/90	Conscious	GTCS	F	N	10.4	PRES	NA	NA	RND
69	Tamiselvi	22	40035	III	R	P1L1	PP	NA	LSCS	II WK	AN	H/AS/NS	110/80	Drowsy	NA	AF	N	7.4	VI/CVT	ED	SS	RND
70	Eswari	32	41095	V	U	P3L2	PP	NA	LSCS	I WK	PE/DH	H/C/AS	140/90	Drowsy	GTCS	AF	P	10.2	VI/CVT	HI/ME/E	SS/SG	RD
71	Maripriya	20	43828	IV	R	P1L1	PP	NA	LSCS	II WK	AN	H/C/VD	110/80	Conscious	GTCS	AF	N	8	VI/CVT	HI/E	TS	RND
72	Vijaya	31	45294	II	U	P3L2	PP	NA	LN	III WK	AN/PE	H/C	140/100	Conscious	GTCS	AF	N	8.9	AI	NA	NA	RD
73	Santhanamari	21	46003	III	R	P2L2	PP	NA	LN	II WK	DH	AS/ND	110/70	Drowsy	NA	AF	N	11	Normal	NA	NA	RND
74	Kuttiammal	24	49320	V	R	G2P1L1	AP	35 WK	NA	NA	AN/PE	C/AS/V	140/90	Stuporous	GTCS	AF	N	8.8	PRES	NA	NA	RND
75	Kala	30	52924	IV	U	P1L1A2	PP	NA	LSCS	I WK	PE/DH	H/AS	140/100	Drowsy	NA	AF	N	10.8	VI/CVT	NHI/ME/E	SS	RND

76	Anandhi	25	56538	III	R	P1L1	PP	NA	LSCS	II WK	PPH/BT	C/VD	110/80	Conscious	Focal	AF	N	12	VI/CVT	HI	SS	RND
77	Ramalakshmi	34	57882	V	R	P1L1	PP	NA	LSCS	I WK	CHT	H/AS/NS	140/90	Drowsy	NA	AF	P	10.1	ICH	NA	NA	D
78	Rejitha	24	58221	V	R	P1L0	PP	NA	LSCS	I WK	AN	AS/F/V	120/80	Drowsy	NA	F	N	8.2	VI/CVT	ED/CS	SS/ST	RND
79	Vadivu	20	59023	V	R	G2A1	AP	36 WK	NA	NA	PE/AN	H/C/VD	160/90	Conscious	GTCS	AF	N	9	PRES	NA	NA	RND
80	Dhanalakhmi	29	60048	III	R	P2L2	PP	NA	LN	II WK	Not Found	AS/F	120/70	Drowsy	NA	F	N	10.3	VI/CVT	HI/E	SS	RND
81	Kavitha	23	61036	IV	R	P2L2	PP	NA	LSCS	I WK	AN/DH	H/AS/F	110/70	Drowsy	NA	F	N	6.8	VI/CVT	NHI/ED/E	SS	RND
82	Sakthi	21	61785	V	U	Primi	AP	28 WK	NA	NA	SeverePE	C/ND/NS	160/110	Conscious	GTCS	AF	P	11	VI/CVT	NHI/CS	TS	RND
83	Jothimani	36	62340	III	R	P3L3	PP	NA	LN	II WK	AN/ HD	H/VD	110/70	Conscious	NA	AF	N	8.8	VI/CVT	ED	SS	RND
84	Umarani	25	62941	V	R	P1L1	PP	NA	Instrumental	II WK	PE/AN/DH	H/C/F/AS	150/110	Stuporous	GTCS	F	P	8.2	VI/CVT	HI/ME	SS/TS	D
85	Vennila	20	64558	II	U	P2L1	PP	NA	LSCS	II WK	AN	C/ND/F	110/70	Conscious	Focal	F	N	7.4	VI/CVT	HI/E	TS/SG	RND
86	Sumathi	31	66121	V	R	P3L3	PP	NA	LN	I WK	AN	AS/V/ND	120/80	Drowsy	NA	AF	N	7	Normal	NA	NA	RND
87	Nagajothi	22	67923	IV	R	P1L1	PP	NA	LSCS	II WK	PE/DH	H/AS/F/NS	160/100	Drowsy	NA	F	N	11	PRES	NA	NA	RND

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