A STUDY OF THROMBOEMBOLIC DISORDERS COMPLICATING PREGNANCY AND PUERPERIUM AND ITS MATERNAL AND FETAL OUTCOMES - PROSPECTIVE OBSERVATIONAL STUDY IN A TERTIARY CARE CENTRE

Dissertation submitted to

THE TAMIL NADU DR. M. G. R MEDICAL UNIVERSITY

In partial fulfilment of the regulation for

the award of the degree of

M.S. BRANCH II

OBSTETRICS AND GYNAECOLOGY

Reg. Number: 221816202

THANJAVUR MEDICAL COLLEGE

THANJAVUR- 613004

THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY

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MAY 2022

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This is to certify that the dissertation titled "**A STUDY OF THROMBOEMBOLIC DISORDERS COMPLICATING PREGNANCY AND PUERPERIUM AND ITS MATERNAL AND FETAL OUTCOMES - PROSPECTIVE OBSERVATIONAL STUDY IN A TERTIARY CARE CENTRE** " is a bonafide work done by Dr. R.CHAMRUTHA , Post graduate student, Department of Obstetrics and Gynaecology, Thanjavur Medical college, Thanjavur – 04, during the period JANUARY 2020 TO JUNE 2021 in partial fulfillment of rules and regulations of the Tamilnadu Dr. M.G.R Medical University, for the award of M.S. Degree Branch II (Obstetrics and Gynaecology) examination to be held in May 2022.

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Approval No.: 557

This is to certify that The Research Proposal / Project titled

A study of thrombo embolic disorders complicating pregnancy and puerperium and its maternal and fetal outcomes

submitted by Dr. R. Chamrutha , Post-graduate **of**

Dept. of Obstetrics & Gynaecology **Thanjavur Medical College, Thanjvur**

was approved by the Ethical Committee.

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DECLARATION

I solemnly declare that this dissertation titled **"A STUDY OF THROMBOEMBOLIC DISORDERS COMPLICATING PREGNANCY AND PUERPERIUM AND ITS MATERNAL AND FETAL OUTCOMES - PROSPECTIVE OBSERVATIONAL STUDY"** was done by me at **Department of Obstetrics and Gynaecology, Thanjavur Medical College, Thanjavur,** during year 2019-2022 under the guidance and supervision of **Prof. Dr. R.RAJARAJESWARI, MD., DGO., DNB.** This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of **M.S. BRANCH II (Obstetrics and Gynaecology).**

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ACKNOWLEDGEMENT

I gratefully acknowledge and sincerely thank **Prof**. **Dr. G. RAVIKUMAR MS., M.Ch.,DNB(Gen)., MRCS(Ed)., FICS., FIME.,** The Dean, Thanjavur Medical College and hospital, Thanjavur for permitting me to conduct the study and use facilities of the institution for my study. I wish to express my respect and sincere gratitude to my beloved teacher and Head of the Department, **Prof. Dr. R. RAJARAJESWARI, MD., DGO., DNB.,** Department of obstetrics and Gynaecology, Thanjavur Medical College, Thanjavur for her valuable guidance and encouragement during the study and also throughout my course period.

I sincerely thank Associate Professors **Dr. S. UDAYA ARUNA, MD., DGO** and **Dr. J. PRABHA. MD(O&G)** for their constant support and guidance throughout the study.

I am bound my ties of gratitude to Asst. Prof. **Dr. ANJU PADMASEKAR, MD (O&G)** for her valuable guidance in conducting this study.

I wish to express my sincere thanks to all the Assistant Professors of Obstetrics and Gynaecology Department for their support during the study.

I thank the Secretary and the Chairman of Institution Ethical Committee, Thanjavur Medical College, Thanjavur.

I would be failing in my duty, if I don't place my sincere thanks to those patients who were the subjects of my study. Above all I thank God Almighty and my parents for immense blessings.

CONTENTS

INTRODUCTION

INTRODUCTION

Thromboembolic disorders accounts for 0.5to 2.0 per 1000 pregnancies, which leads to 1.1 deaths per 1,00,000 pregnancies. The risk of thromboembolism in pregnancy is four to five times higher than that of non-pregnant women.

Early recognition of signs and symptoms and prompt treatment reduce the morbidity and mortality due to thromboembolic disorders.

In 1856, Rudolf Virchow, postulated Virchow triad–venous stasis, hypercoagulability and endothelial changes. Pregnancy itself predisposes the risk of thromboembolism.

Around 70% of the Deep vein thrombosis in antenatal patients occurs within less than 15 weeks of gestation during the antenatal period .Most of the Deep vein thrombosis occurs in iliofemoral vein, which is more prone to embolism and difficult to diagnose.

Cortical venous thrombosis, thrombosis of dural venous sinuses and cerebral veins affects more commonly during the puerperal period, due to the pro thrombotic state. More commonly the patient presents with headache, seizures, papilledema, altered sensorium and focal neurological deficits. Cantu from Mexico reported 59% cortical venous thromboses are puerperal. International Study on Cerebral and Dural Sinus Thrombosis reported 20% are obstetric cerebral venous thrombosis; Compared to Mexico, India showed highest frequency of cases. Cross et al[1] states that “If the patient survives acute episode, recovery is rapid and complete“.

Cerebral sinus venous thrombosis is considered to be a medical emergency,[2] mode of onset highly variable, and spectrum of its clinical manifestations is extremely wide.

Pulmonary embolism is rare, affects 1 in 7000 pregnancies. Pulmonary embolism is mostly fatal if not treated promptly.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Normal pregnancy is accompanied by increased concentrations of factors VII, VIII, X, and IX and von Willi brand factor and by pronounced increases in fibrinogen. Factors II and V are unchanged. Free protein S, the active, unbound form, is decreased during pregnancy secondary to increased levels of its binding protein, the complement component C4b. Plasminogen activator inhibitor type 1 (PAI-1) levels increase 5-fold. Levels of PAI-2, produced by the placenta, increase dramatically during the third trimester. Markers of thrombin generation such as prothrombin F1+2 and thrombin-anti thrombin (TAT) complexes are increased. These changes, which may not completely return to baseline until more than 8 weeks postpartum, begin with conception. So does the risk of thrombosis.

Epidemiology

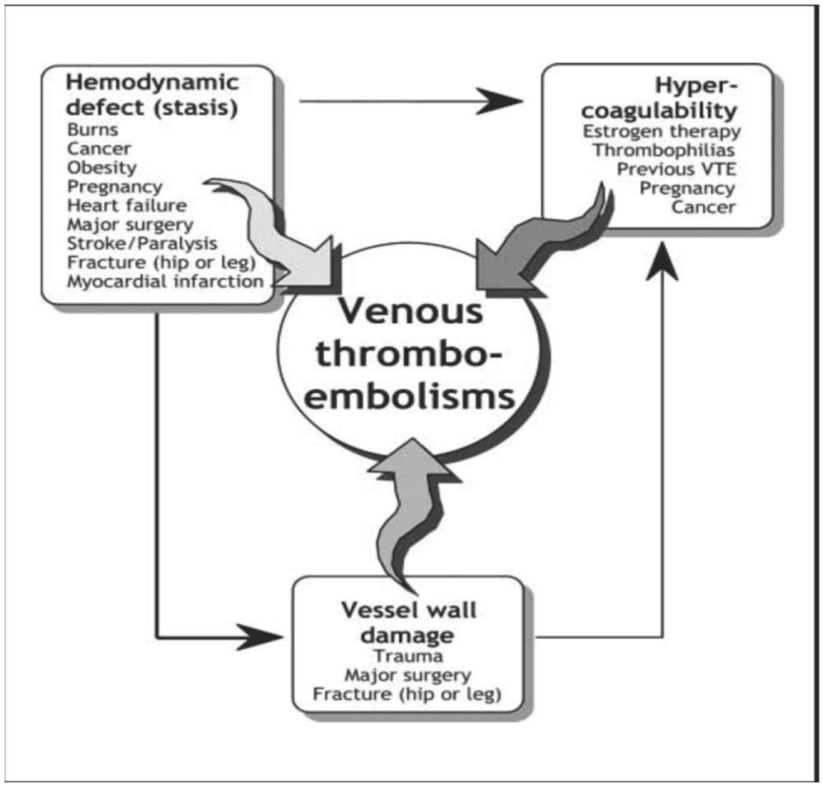
The thrombosis is the pathological presence of a clot in a blood vessel causes obstruction of blood flow through the circulation. Thrombosis is considered as distinct disease characterized by different pathogenic mechanisms and underlying risk factors.

Thromboembolic disorder accounts for 0.5to 2.0 per 1000 pregnancies, which leads to 1.1 deaths per 1,00,000 pregnancies .The most common sites of thrombosis are deep vein thrombosis of the leg or pelvis or pulmonary embolism , Less frequent sites includes portal vein, the splanchnic vein, the cerebral veins and the veins of the upper extremity. The incidence of VTE is slightly higher in women at younger age group, because of increased incidence due to pregnancy and female hormonal intake.

The thrombosis during pregnancy is due to homeostatic changes that occur during the pregnancy. During normal pregnancy, the clotting factors fibrinogen, VII, VIII, Von Willi brand factor, IX, X, and XII are all increased, results in hypercoagulable state, which increases the risk of thrombosis.

Moreover, the mechanical obstruction by the growing uterus compromises venous return and increases the susceptibility of pregnant and postpartum women for developing thromboembolisms. Moreover, pregnancy combined with either heritable or acquired forms of thrombophilia constitutes a cumulative risk of thrombosis.

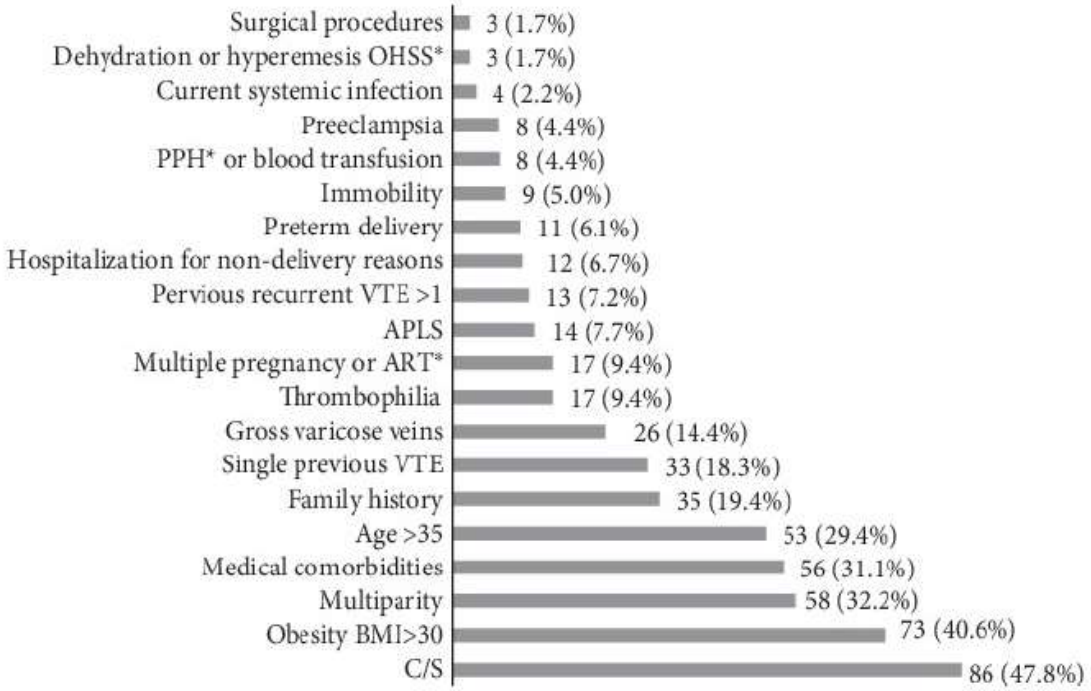
Pathophysiology



Risk Factors of Pregnancy-Associated Thrombosis Based on RCOG Risk Assessment

Cesarean section was the topmost prevalent risk factor among study participants, which was observed in 86 (47.8%) patients, followed by obesity (73 (40.6%) patients), multiparity (>3 children; 58 (32.2%patients), the presence of medical comorbidities (such as T2DM, gestational DM, and hypertension; 56(31.1%) patients), age >35 years (53 (29.4%) patients), and family history (19.4% of patients).

Risk factors that were less frequent include other surgical procedures (3 (1.7%) patients), dehydration, ovarian hyper stimulation syndrome (OHSS; 3 (1.7%) patients), systemic infection (4 (2.2%) patients), preeclampsia (8 (4.4%) patients), and postpartum hemorrhage (PPH) or blood transfusion (8 (4.4%) patients.



CORTICAL VENOUS THROMBOSIS

The term “primary” or “idiopathic” cortical venous thrombosis is considered when no specific etiological factor is evident. “Secondary” venous thrombosis from a variety of causes that include injury, infection, haematological disturbances, dehydration. In India, cortical venous thrombosis in puerperal period is very common, 10-12 times higher than in the West.

History

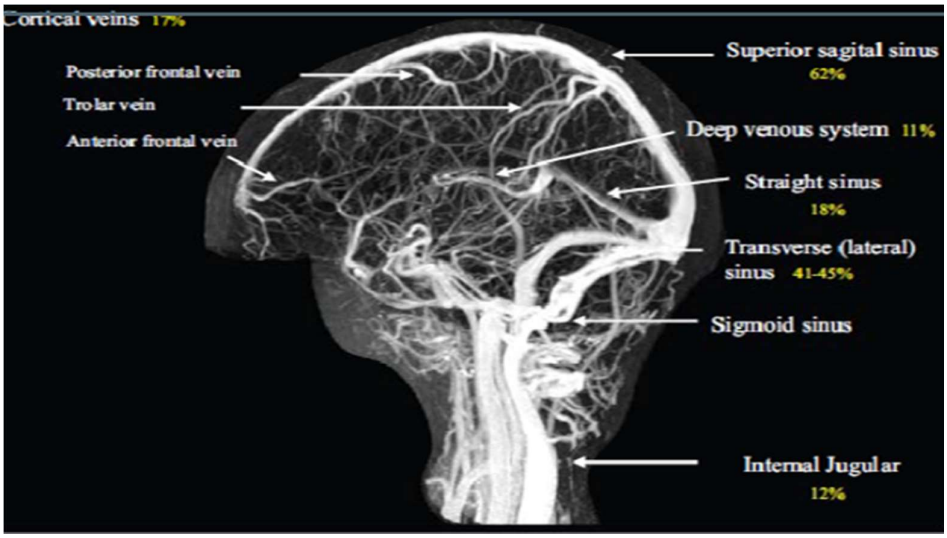
Ribes[3] first reported primary venous sinus thrombosis in 1825. In 1828, John Abercrombie of Scotland described a 24-year old lady died in puerperal period, Autopsy revealed ischemic and hemorrhagic infarcts with thrombosed and sclerosis cortical veins. Kalbag and Woolf, Purdon Martin, Si Charles Symonds and others gave a precise clinical description of CVT after 1940[3]. Several large series with confirmation of diagnosis by angiograms, surgical exploration, and autopsy and recently with CT and MRI studies have been reported from the Indian subcontinent.[4, 5]

Epidemiology

The true incidence of CVT is unknown. Ehlers and Courville found only 16 superior sagittal sinus thrombosis in a series of 12,500 autopsies.[6] Recently large clinical series, the true incidence if CVT is probably considerably higher than that thought from autopsy series .People of all age groups may be affected but slight preponderance in young women because of specific causes like oral contraceptives, pregnancy and puerperium. Puerperal CVT has been reported to account up to 15-20%

of young stroke. It is the commonest cause of stroke in young women in India. In a study sample of 230 cases of CVT seen over a period of 8 years at National Institute of Mental Health and Neuro Sciences (NIMHANS), Bangalore, 200 cases were in the puerperal period.[7]

Relevant Venous Anatomy



The cerebral veins that empty into dural sinuses, and then drain into two internal jugular veins, drain blood from the brain.[8]

The Dural venous sinuses are valve less, translocated venous blood channels and may be divided into a superior group related to the vault and a basal group found at the skull base.

The superior group-sagittal, transverse and straight sinuses are the main components.

The basal group-cavernous, petrosal and sphenoparietal sinuses. The Dural sinuses most commonly thrombosed are the superior sagittal sinus (SSS), lateral sinus and cavernous sinus.

Superior Sagittal Sinus (SSS)

Superior Sagittal Sinus lies in the anterior border of falx cerebral and runs from the foramen caecum to the occipital protuberance, where it joins with straight sinus lateral. The Superior Sagittal Sinus is triangular in cross section, increases in size from before backwards. In the majority of individuals, most of its flow is directed to the right transverse sinus with the straight sinus draining to the left transverse sinus. Cortical veins enter perpendicular to the Superior Sagittal Sinus anteriorly but the angle becomes shallower more posteriorly with the veins entering against the direction of flow. Superior Sagittal Sinus and other sinuses play a major role in CSF circulation because they contain most of the arachnoid villi and granulations (pacchionian bodies) in which much of the CSF absorption takes place.

Lateral Sinuses (LS)

The lateral sinuses extend from the torcular herophili to the jugular bulbs and consist of the transverse and sigmoid portions. They drain blood from the cerebellum, brain stem and posterior portion of cerebral hemispheres. The transverse sinuses commence at the torcula and lie within the outer margin of the tentorium. The right is usually 'dominant' and larger than on the left, receiving almost the entire output of the superior sagittal sinus. The sinus on one side can be poorly developed or even absent. The transverse sinuses become the sigmoid sinuses at the posterior petrous edge continuing towards the jugular bulb. The straight sinus lies at the junction of the falx and the tentorium where the straight, transverse and Superior Sagittal Sinus meets.

Numerous lateral sinuses, anatomic variations may be misinterpreted in sinus occlusions on angiography. In Hacker's study[9], transverse portions were not visualized on ipsilateral carotid angiograms in 14% of cases on left side and 33% on right side, whereas sigmoid portions, which may be directly injected via cerebral veins, failed to fill in 4% of cases on left side and were always demonstrated on right.

Cavernous Sinus

This sinus drains venous blood from the orbits through the ophthalmic veins and from anterior part of the base of the brain via the sphenoparietal sinus and middle cerebral veins. They empty into both superior and inferior petrosal sinuses and ultimately into internal jugular vein. Because of their situation, cavernous sinuses are often thrombosed in relation to infections of face or sphenoid sinusitis. In contrast to other sites, infection is the leading cause of cavernous sinus thrombosis.

Petrosal and Sphenoparietal Sinuses

The superior petrosal sinuses are situated at the junction of the tentorium and the petrous bone. They drain to transverse sinuses. The inferior petrosal sinuses lie between the clivus and petrous apex and run medial to the superior sinus joining the jugular bulb. The sphenoparietal sinus is a medial extension of the sylvian vein and courses around the greater sphenoid wing.

The Supratentorial Venous System: Cerebral veins: Three groups of veins that draw blood supply from the brain are: (1) superficial cerebral, (2) deep cerebral and (3) veins of post-fossae.

- Superficial cerebral veins: and the cortical veins - frontal, parietal and occipital superior cerebral veins drain the cortex, ascending to the Superior Sagittal Sinus. The middle cerebral veins drain into the cavernous sinuses. Troland's greater anastomotic vein connects the Superior Sagittal Sinus to middle cerebral veins, which are they connected to LS by the vein of labbe. The cortical veins present some peculiarities that are important to know to understand some of the clinical features of CVT – they have thin walls, no muscle fibers and no valves. These features allow for dilation and reversal of the direction of blood flow when sinuses into which they drain are occluded. They are linked by numerous anastomoses, allowing development of collateral circulation (angiographically visible as corkscrew vessels). This probably explains the good prognosis of the venous thrombosis.

- Deep Cerebral Veins The internal cerebral and basal veins both join to form the great vein of Galen, which continues as straight sinus drain blood from the deep white matter of the cerebral hemispheres and from basal ganglia. In contrast to the superficial system, the deep system anatomy is constant and is always visualized on angiography, so that thrombosis is easily recognized.

- Veins of Posterior Fossa There are three groups: (i) Superior veins draining into the galenic system. (ii) Anterior veins draining into petrosal sinuses and 9 (iii) Posterior veins draining into the torcular or neighboring straight sinus and lateral sinus. They are variable in course and angiographic diagnosis of their occlusion is extremely difficult.

Causes of CVT

In the pre-antibiotic era, infection was commonly associated with CVT. Recently various causative factors involved are infectious, inflammatory diseases, neoplasm, Coagulation abnormalities, and pregnancy and endocrine conditions. Puerperium is associated with a six fold increase in risk of venous thromboembolism. Even in developed countries, despite extensive investigations, in 25% cases, no cause is found.

Pathogenesis of cerebral venous thrombosis –

Hemorrhagic infarction occurs in approximately 10-50% of cases, principally affecting the cortex and adjacent white matter. This is thought primarily due to elevated venous and capillary pressure caused by the persistence of thrombus.

Various theories involved in the pathogenesis of the cerebral venous thrombosis.

- Infective theory
- Embolism
- Local endothelial damage
- Hypercoagulability

- Infective theory

The septic thrombosis is the one where a purulent infection occurs with thrombophlebitis. The examples are cavernous sinus thrombosis following facial infection and lateral sinus thrombosis following otitis media. In Daif A et al.[10] studied 40 cases of CVT, infection was the cause of CVT in 7% of cases, whereas

it was the cause in 16% and 17% of cases reported by Bousser et al.[34] and Shell and Rathe, [11] respectively.

- Martin-Batson Theory of Embolic Thrombosis

  The pathogenesis of puerperal venous thrombosis, the studies of Batson (1940) and extension of the results of that study by Martin (1941). Martin (1941) argued that under circumstances of increased intra-abdominal pressure, the thrombi from parturient mother could pass into intracranial sinuses via the vertebral plexus. Once the thrombus reaches the sagittal sinus, where blood flow is slow, it acts as a nidus for further thrombosis. [12]

  This theory fails to explain

  - The basic mechanism of puerperal thrombosis.
  - The fact that Superior sagittal sinus is most frequently involved, although vertebral plexus of veins communicate with the occipital and petrosal sinuses and not SSS.
  - Delayed onset of symptoms.

- Kendall's Theory of Local Damage (1948)

  He suggested that damage to endothelial lining occurs during periods of breath holding and straining, which occur during the second stage of labor.[13]

- Stasis

Bailey (1971) noted that in many pathologically proven SSS thrombosis, the thrombus was oldest in the middle fifth of the sinus and in some cases only the central portion was involved suggesting involvement of certain local factors. In this region the superior cerebral veins enter the sinus in a direction opposite to the direction of blood flow in the sinus. Also there is sudden widening of the sinus at this point and may contribute to the localization.

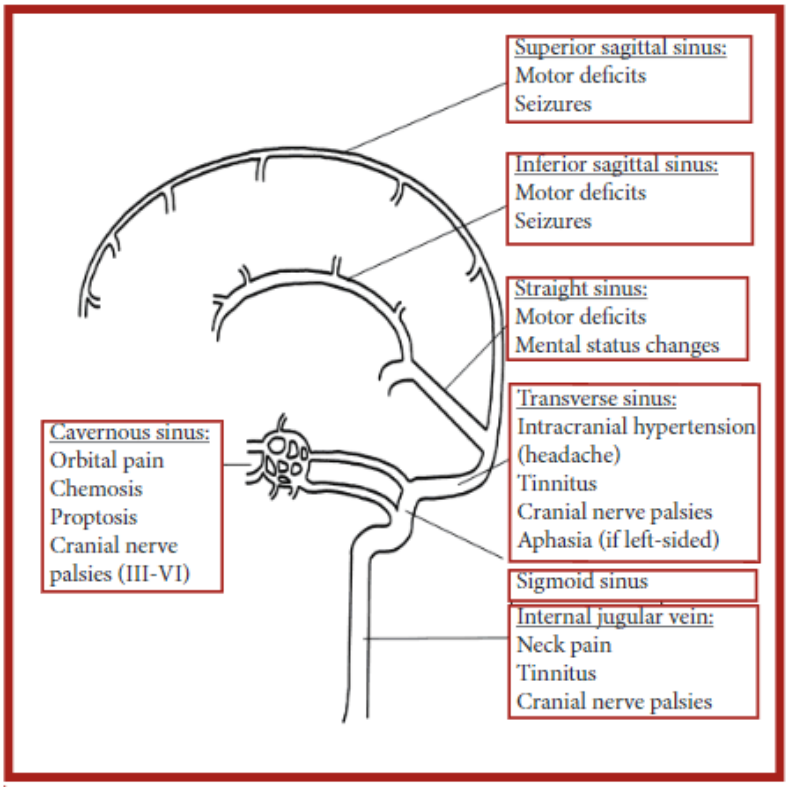
- Theory of Hypercoagulability

  During pregnancy normally the hemostatic balance changes in the direction of hypercoagulability, thus decreasing bleeding complications in connection with delivery. The most important initial factor for acute hemostasis at delivery is, however, uterine muscle contractions, which interrupt blood flow. Increased endogenous thrombin generation, acquired protein C resistance, slightly decreased activated partial thromboplastin time (APTT) and increased prothrombin complex level (PT) measured as INR of less than 0.9, activation of platelets, release of betathromboglobulin and platelet factor 4 and increased thrombomodulin levels have been reported in normal pregnancy. Most blood coagulation factors and fibrinogen increase during pregnancy. Factor XI is the only blood coagulation factor that decreases during pregnancy. Fibrinolytic capacity is diminished during pregnancy, mainly because of markedly increased levels of plasminogen activator inhibitor 1 (PAI-1) from endothelial cells and plasminogen activator inhibitor-2 (PAI-2) from the placenta. The total hemostatic balance has been studied by analysis of prothrombin fragment 1 and 2, thrombin-antithrombin complex, fibrinopeptide A, soluble fibrin, D-dimer and plasmin-antiplasmin complex. There

is activation of blood coagulation and a simultaneous increase in fibrinolysis without signs of organ dysfunction during normal pregnancy. These changes increase as pregnancy progresses. Fibrinolysis improves and increases fast following childbirth and expulsion of the placenta, resulting in increased D-dimer levels. These changes are self-limiting at normal delivery. The hemostatic changes normalize after delivery within 4 to 6 weeks. Platelet count and free protein 5, however can be abnormal for a longer period.[13,14,15,16,17,18]

Pathophysiologically, there are important differences between arterial and venous thrombosis. CVT has been described as a continuing process in which the balance of prothrombotic and thrombolytic processes is disturbed, leading to progression of the venous thrombus in time.[19]

Clinical Features of Cerebral Venous Thrombosis



Headache

Most prominent and frequently presenting symptom in 70-80% of the patients. [20, 21, 22, 23, 24, 25] There are no specific features. It may be acute, like a thunderclap headache[21], sub-acute or chronic. In most of the cases, it is associated with nausea, vomiting and other neurological symptoms. It is probably caused by leakage of blood to the surface stimulating pain-sensitive fibers in the dura or due to rise of intracranial pressure.

Fever

This is often present with septic CVT or those secondary to systemic infections. Although not mentioned as an important feature in two large international studies, [21, 23] frequently seen in Indian patients.[22, 24, 26, 27] Pyrexia is noted in 15-60% of such cases, does not necessarily imply infection and is probably caused by an aseptic thrombotic process.[26, 27] Superior sagittal sinus thrombosis in infants and children may present only with fever and convulsions which may be confused for febrile seizures.[26]

Seizures

These occur more frequently in CVT than in arterial stroke. 12-15% of patients present with seizures and in 39-68% they occur sometime during course of illness. They are more common in patients with focal deficit and also when onset is acute or subacute. Seizures may be focal, multimodal or generalized, and are commonly due to 'irritation' of the cortex when there is a hemorrhagic venous infarct. Status epilepticus is the presenting feature in some cases.[25] It's incidence varies from as low as 3.5% to as high as 42% in two Indian series.[28,29]

Focal Deficits and Impaired Sensorium

Focal signs vary with extent and site of thrombosis. They may be motor or sensory, unilateral or bilateral and fluctuating. They are seen in approximately 60% of cases.[21] Altered sensorium is usually due to raised intracranial pressure (ICP) which is often preceded by headache and severe focal deficits. Although moderate drowsiness is usually reversible with therapy, progressive worsening of

sensorium is associated with a poor prognosis. Aphasia, mutism and cerebellar infarcts are also described.[21, 33] Cranial nerve palsies are uncommon; but a total ophthalmoplegia is seen with cavernous sinus thrombosis and ninth and tenth nerve palsies with thrombosis of the internal jugular vein.[30]

Papilloedema

Thrombosis of the dural venous sinuses leads to an increase in venous pressure and alters the pressure gradient leading to poor CSF absorption through the Pacchionian granulations. Hence there is rise of ICP and papilloedema. The incidence of papilloedema is variable and depends on the aetiology, rate and site of venous thrombosis. It is less common in puerperal CVT with acute presentation of headache, seizures, focal deficits and altered sensorium. Ameri and Bousser found puerperal CVT in 50% of French patients,[31] 45 cases with sub-acute onset a clinical picture stimulating idiopathic intracranial hypertension can be as high as 83-100%.[21, 22] Other uncommon clinical features are signs of meningeal irritation, cortical blindness, akinetic mutism, dystonia and associated venous thrombosis in the lower limbs or other parts of the body.

Nagaraj et al.[20] grouped clinical features of CVT into four categories, depending on the topographic venous involvement.

- Presentation with seizures, focal deficits and progressively deteriorating consciousness. Thrombosis involves the dural sinuses as well as cortical veins producing cerebral infarction.

- Presentation with symptoms and signs of raised intracranial tension namely headache, vomiting and papilloedema. If thrombosis is confined to dural sinuses, the course is slow and prognosis is favourable.

- Occasionally thrombosis predominantly involves cortical veins and patient may present with features of space occupying lesion.

- Rarely thrombosis predominantly involves the deep venous system. Patient manifest symptoms of raised Intracranial pressure, focal deficits, choreo-athetosis, ocular signs and coma. It runs a fulminant course.

Table 2: Clinical features of cerebral venous thrombosis

Sl. No.	Clinical features	Nagaraja (1980) n=138(%)	Bansal (1983) n=135(%)	Srinivas (1987) n=200(%)
1.	Fever	62	16	11
2.	Headache	48	24	57
3.	Vomiting	36	24	-
4.	Seizures (a) GTCS (b) Focal (c) Status epilepticus	 29 17 -	 50 22 -	 35 23 14
5.	Diplopia	1	-	-
6.	Dysphasia	25	5	-
7.	Nuchal rigidity	3	10	13
8.	Deep leg vein thrombosis	10	-	-
9.	Altered sensorium	41	43	81
10.	Papilloedema	35	16	14
11.	Ocular palsy	-	2	11
12.	Motor deficit (a) Monoplegia (b) Hemiplegia (c) Paraplegia	 5 43 27	 9 37 3	 69

In another study conducted by Ameri et al.[44] in a series of 110 patients with CVT, main signs and symptoms reported are the following:

Table 3: Clinical features described by Ameri et al.

Sl. No.	Clinical features	Ameri et al. (1992) n=110(%)
1.	Headache	83 (75%)
2.	Papilloedema	54 (49%)
3.	Motor or sensory deficit	38 (34%)
4.	Seizures	41 (37%)
5.	Drowsiness, mental changes, confusion or coma	33 (30%)
6.	Dysphasia	13 (12%)
7.	Multiple cranial nerve palsies	13 (12%)
8.	Cerebellar incoordination	3 (3%)
9.	Nystagmus	2 (2%)
10.	Hearing loss	2 (2%)
11.	Bilateral or alternating cortical signs	3 (3%)

Table 4: Sites of venous occlusion in CVT

Sl. No.	Sinuses affected	Ameri et al. (1992) n=110(%)
1.	SSS	79 (72%)
2.	Lateral sinus Right Left Both	78 (70%) 29 (26%) 29 (26%) 20 (18%)
3.	Straight sinus	16 (4.5%)
4.	Cavernous sinus	3 (2.7%)
5.	Cerebral veins Superficial Deep Cerebellar veins	42 (38%) 30 (27%) 9 (8%) 3 (3%)
6.	One sinus only SSS LS SS	14 (13%) 10 (9%) 1 (1%) 1 (1%)
7.	Isolated cortical veins plus cerebral or cerebellar veins	39 (35%)

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

NEURO IMAGING:

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

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

Direct Signs of Cerebral Venous Thrombosis

- The **cord sign** which is visible on CT scans without contrast enhancement represents the thrombosis 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*. [41] It is evident in contrast CT images. It appears as a filling defect in the posterior part of SSS in case of occlusion.

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 hypo density suggests cerebral edema.
- Hypo density 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 hyper dense lesions in 10% to 50%cases.
    - In rare cases, there can be a subarachnoid haemorrhage or subdural hematoma along with other features. Sometimes it may be the only sign of CVT.
    - Non - haemorrhagic venous infarcts appear as focal hypo dense 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.

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

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

Trans cranial venous Doppler:

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

Dimer ASSAY:

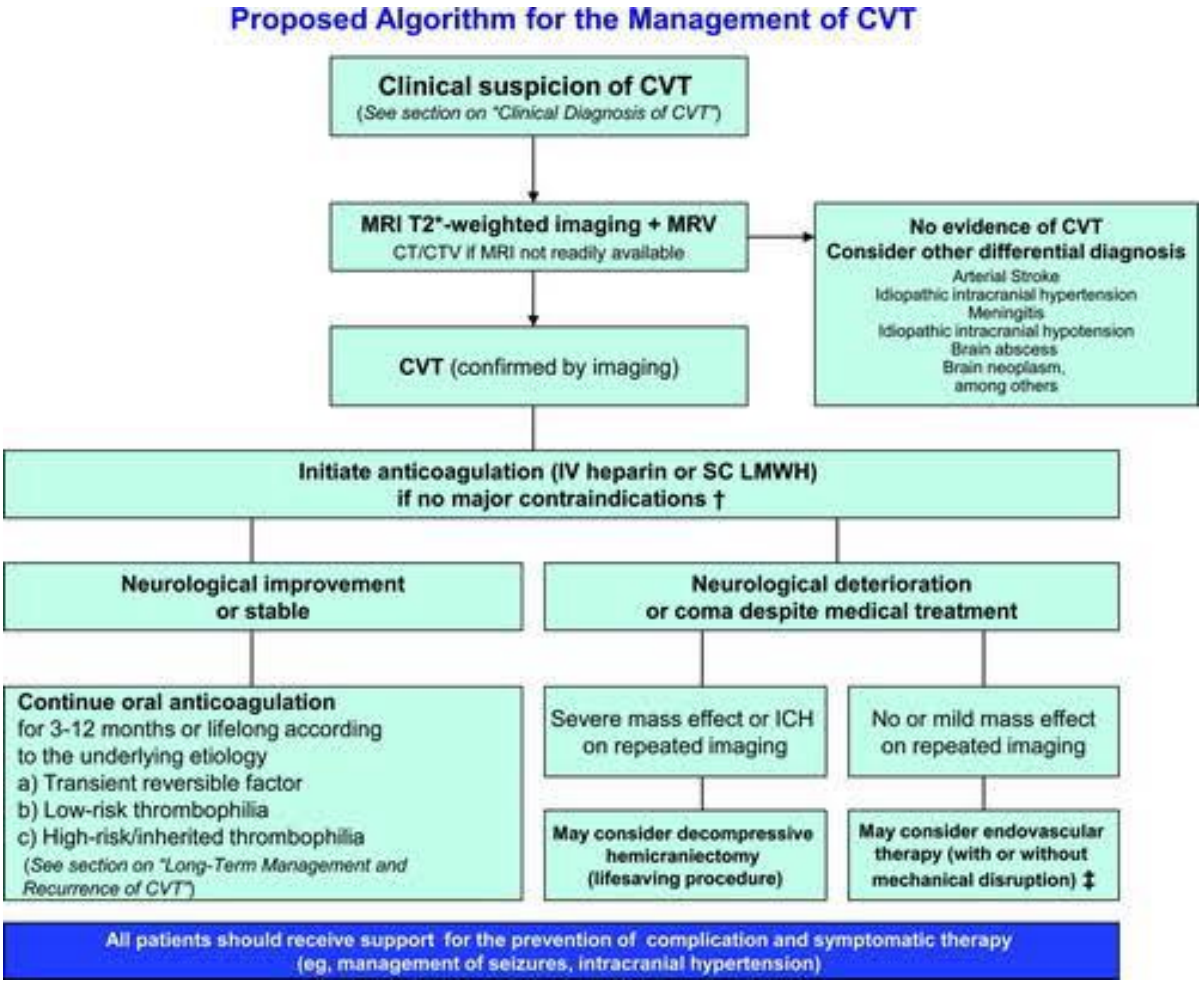
In most patients with recent CVT there is elevation of D Dimer concentration. A low value will rule out the diagnosis.

LUMBAR PUNCTURE:

It won't helpful in establishing the diagnosis of CVT. It may be used to rule out other causes like meningitis.

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

THE AMERICAN STORKE ASSOCIATION 2019 GUIDELINES [35] recommends the following for management in CVT.



Deep vein thrombosis

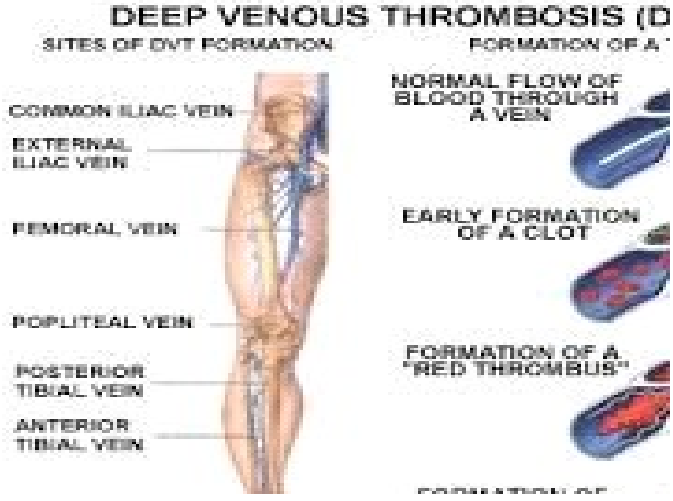
Risk of thrombosis is 2-3 times more in puerperium compared to pregnancy. Risk is equal throughout all the three trimesters.

Approximately 80% of venous thromboembolic events during pregnancy are deep vein thrombosis (DVT) and 20% are pulmonary emboli. Approximately one third of pregnancy-related DVT and half of pregnancy-related pulmonary emboli occur after delivery. When DVT occurs during pregnancy, it is more likely to be proximal, massive, and in the left lower extremity. Distal thrombosis are as likely to occur on the right as on the left, but proximal thrombosis occurring under the influence of oestrogen are more likely to be on the left. This left-sided predominance is thought to be attributable to a relative stenosis of the left common iliac vein.

Absolute risk of VTE in pregnancy is 0.025–0.1%

- ✓ Sixty-five percent of DVT occur antenatal
- ✓ Half of antenatal DVT occur prior to 15 weeks
- ✓ More than half occur in ileofeomral vein
- ✓ Rate of VTE is greatest in puerperial period, which is more on left side

Relevant anatomy



Clinical features -

The two most common initial symptoms, present in more than 80% of women with pregnancy-related DVT, are pain and swelling in an extremity[35]

Reasons

- Impaired venous flow due to obstruction
- Transient hypercoagulable state
- Increased fibrinogen level
- Acquired functional resistance to APC
- TPA levels reduced. PAI 1&2 increased.

Caesarean increases the risk of DVT compared to normal vaginal delivery. Other risk factors for puerperal DVT are maternal age advancement, suppression of lactation, hypertension, and assisted delivery.

CLINICAL ASSESSMENT FOR DVT DONE BY WELL'S SCORE

- Calf swelling ≥3 cm compared to symptomatic calf (measured 10 cm below tibial tuberosity)
- Active cancer (treatment within last 6 months or palliation)
- Swollen unilateral superficial veins (nonvaricose in symptomatic leg)
- Unilateral pitting edema in symptomatic leg
- Localized tenderness along the deep venous system
- Previous documented DVT
- Swelling in entire leg
- Paralysis or paresis or recent cast immobilization of lower extremity

- Recently bedridden for more than 3 days, or major surgery requiring regional or general anesthesia in past 12 weeks
- Any other alternative diagnosis at least likely

Clinical Probability for DVT Depends on the Score

- <0—low
- 1–2—intermediate
- >3—high

DIAGNOSIS

- Clinical prediction score
- D-dimer test in patients with low probability for DVT or PE,
- D-dimer test is a good option if negative it indicates very low likelihood. It has a very high false positive rate but a good negative predictive value
- Ultrasound is recommended in patients with intermediate to high probability for DVT. Compression duplex ultrasound is the primary diagnostic test for DVT.

Clinical diagnosis is notoriously inaccurate for diagnosing DVT. Doppler ultrasonography has a sensitivity and specificity of 95% for proximal DVT. Doppler ultrasonography is more accurate for detecting symptomatic, proximal, and first-time DVTs than for detecting those that are asymptomatic, distal, or recurrent. Other Radiological studies are contraindicated in pregnancy

Diagnostic criteria for DVT are:

- Soft tissue mass within the venous lumen
- Noncompressibility of vein
- Decrease or absent flow in the vessel Sometimes if the first ultrasound is negative but the index of suspicion is very high, anticoagulation may be continued till performance of the second test. If on repeat testing DVT is negative treatment is stopped.
- Impedance plethysmography
- CT and MRI venography mainly useful in pelvic vein thrombosis.
- Routine test like platelet count, aPTT, RFT, LFT should be done prior to starting anticoagulation
- Thrombophilia screening has to be done, but can be delayed in acute episode, as there would be no change in immediate management
- Radioactive fibrinogen test, not recommended in pregnancy and lactation due to risk of radiation exposure.

Foetal Surveillance in Patients with DVT

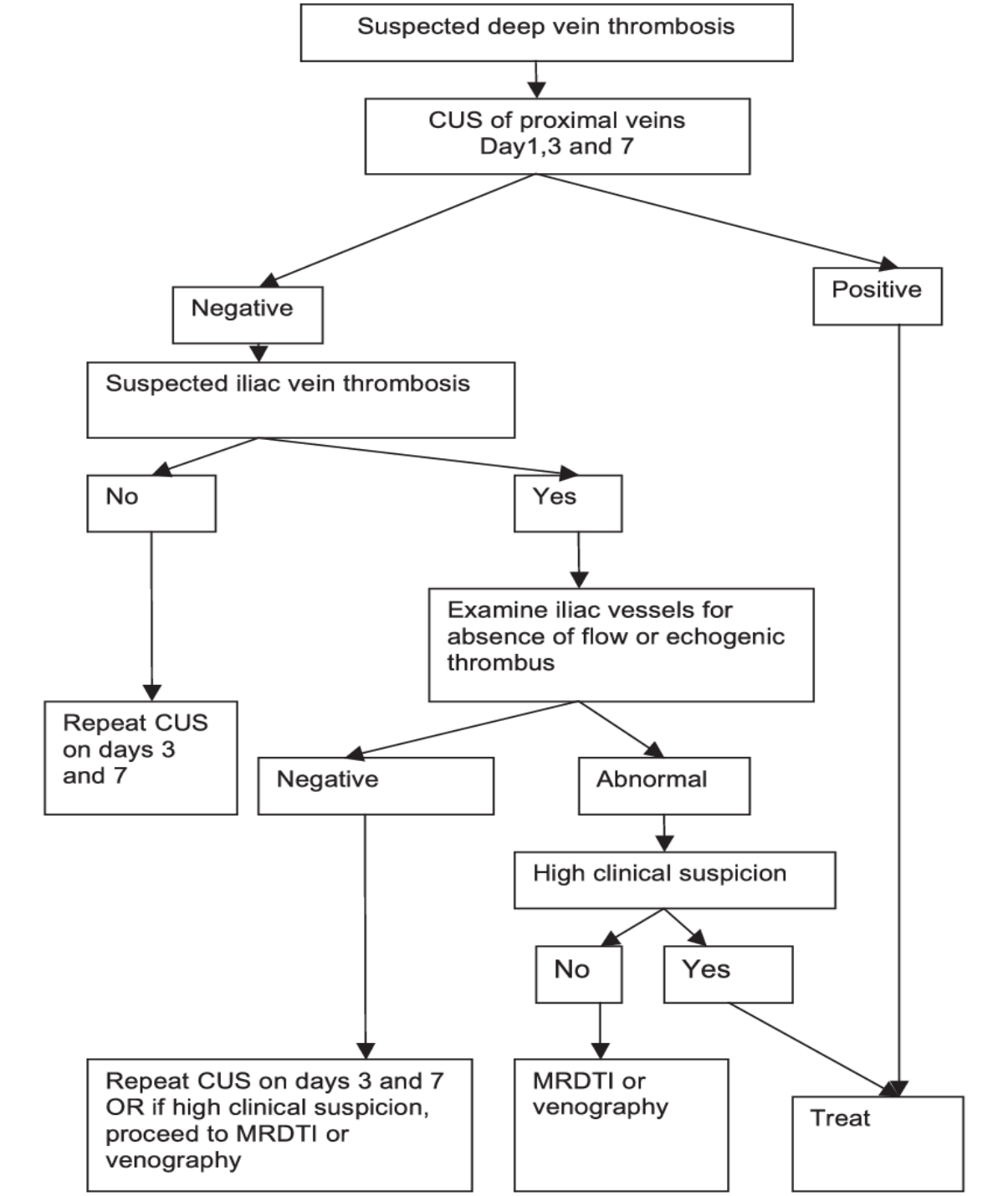
Patients with thrombophilia or other risk factors for VTE are at a risk of placental thrombosis, uteroplacental insufficiency, IUGR, sudden foetal death, abruption, and hence poor perinatal foetal outcome.

Patients who were receiving anticoagulation in the periconceptional period with warfarin are at a risk of congenital foetal anomalies. Thus, foetal surveillance should be done.

- Detailed anomaly scans at 18–20 weeks
- Doppler study at 24–28 weeks for placental insufficiency
- Ultrasonography assessment at regular interval in third trimester for growth assessment
- After confirming fetal well-being on term delivery should be planned and appropriate measures taken.

MANAGEMENT –

1. **MEDICAL -** Anticoagulation therapy
2. **SURGICAL –**
   - Venous Thrombectomy (ilio femoral DVT)
   - Pulmonary Embolectomy
3. **ENDOVASCULAR -** Inferior Venacava filter placement
4. **NON PHARMACOLOGICAL MEASURES** -
   - Adequate Hydration
   - Early Mobilisation
   - Graduated Compression Stockings
   - Pneumatic compression devices



CUS - Compression ultrasonography

MRDTI - Magnetic Resonance Direct Thrombus Imaging (MRDTI) sequence

Pulmonary embolism

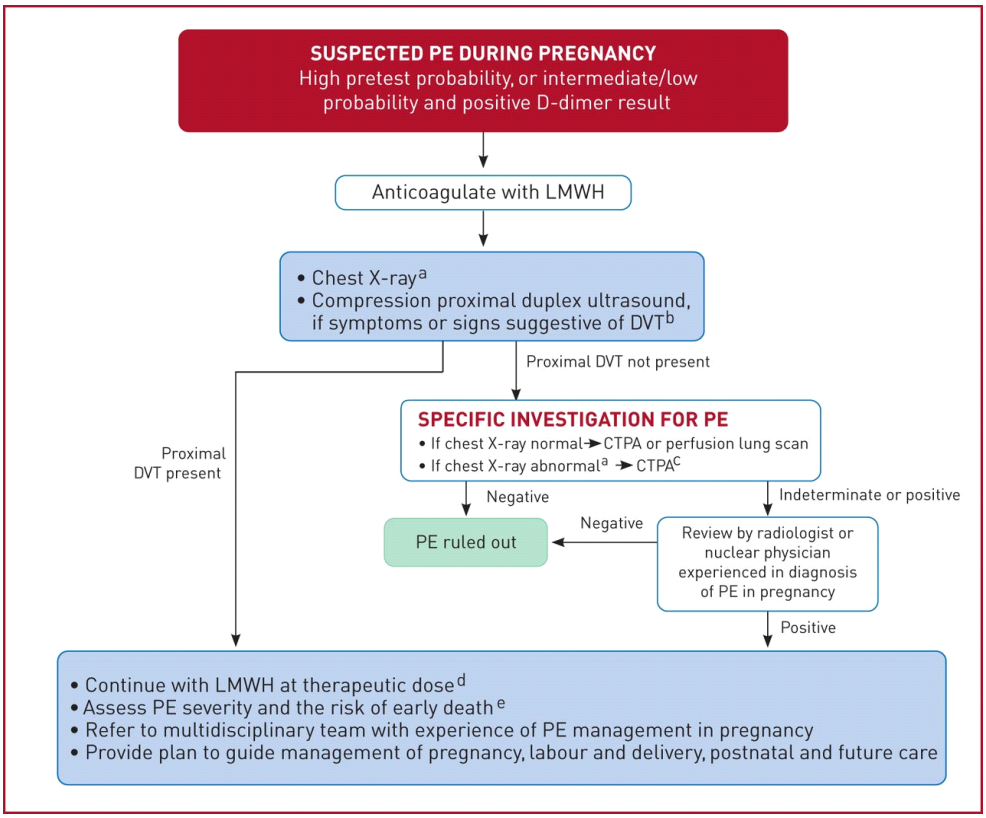
The most important complication of acute DVT is Pulmonary Embolism.

Asymptomatic presentation can be up to 80%. Approximately 90% of PE is caused by proximal lower extremity venous thrombosis. Some autopsy series states that the combination of DVT and PE is 1.8 times more common than DVT alone. Symptomatic presentation of PE is not only dependent on amount of vessel occlusion, but also the cardio respiratory reserve of the patient. The risk of death in symptomatic PE is 18 fold higher than DVT patient alone.

Patients with multiple risk factors at high risk of DVT or PE should receive thromboprophylaxis with both pneumatic compression devices and unfractionated heparin or low-molecular-weight heparin

The preferred agents for anticoagulation in pregnancy are heparin compounds. Neither heparin nor low-molecular-weight heparin crosses the placenta, and both are considered safe in pregnancy. Unique aspects of anticoagulation in pregnancy include an increase in maternal blood volume of 40% to 50% and an increase in the volume of distribution. An increase in glomerular filtration results in increased renal excretion of heparin compounds, which are eliminated by this route. Additionally, there is an increase in protein binding of heparin. During pregnancy, both unfractionated heparin and low-molecular-weight heparins have shorter half-lives and lower peak plasma concentrations, usually necessitating higher doses and more frequent administration

ESC (EUROPEAN SOCIETY OF CARDIOLOGY) GUIDELINES,[19] 2019, FOR DIAGNOSIS AND MANAGEMENT OF ACUTE PULMONARY EMBOLISM IN PREGNANCY



Clinical features of pulmonary embolism-

1. Dyspnea

2. Chest pain

3. Cough -are the most frequent symptoms

Fever, tachycardia, abnormal pulmonary signs, and peripheral vascular collapse are the most common physical findings.

Cyanosis, hemoptysis, syncope, and the various manifestations of acute cor pulmonale are less commonly observed.

Diagnosis of Pulmonary Embolism -

PE is detected based on the patient's medical history, a physical exam, and test results. The tests used to detect a pulmonary embolism are:

- ✓ Ultrasound of the leg
- ✓ Computed tomography (CT) scan
- ✓ Lung ventilation perfusion scan-for initiation of treatment
- ✓ Pulmonary angiography-definitive diagnosis
- ✓ Blood tests
- ✓ Echocardiography
- ✓ Electrocardiogram
- ✓ Chest X-Ray
- ✓ Chest MRI

MANAGEMENT -

LMWH is the treatment of choice for pulmonary embolism during pregnancy, it does not cross the placenta, and consequently does not confer a risk of foetal haemorrhage or teratogenicity.

Unfractionated Heparin is also safe in pregnancy, LMWH has more predictable pharmacokinetics and a more favourable risk profile.

Fondaparinux may be considered if there is an allergy or adverse response to LMWH, although solid data are lacking and minor transplacental passage has been demonstrated. Warfarin may be associated with central nervous system anomalies in the foetus throughout pregnancy.

The management of labour and delivery requires particular attention. In women receiving therapeutic LMWH, strong consideration should be given to planned delivery in collaboration with the multidisciplinary team to avoid the risk of spontaneous labour while fully anticoagulated. The incidence of spinal haematoma after regional anaesthesia is unknown in pregnant women under anticoagulation treatment. If regional analgesia is considered for a woman receiving therapeutic LMWH, ≥24 h should have elapsed since the last LMWH dose before insertion of a spinal or epidural needle (assuming normal renal function and including risk assessment at extremes of body weight).

In high-risk situations, for example in patients with recent PE, it is recommended that LMWH be converted to UFH ≥36 h prior to delivery. The UFH infusion should be stopped 4 − 6 h prior to anticipated delivery and the activated partial thromboplastin time should be normal (i.e. not prolonged) prior to regional anaesthesia.

Anticoagulant treatment should be administered for ≥6 weeks after delivery and with minimum overall treatment duration of 3 months. LMWH and warfarin can be given to breastfeeding mothers.

High-risk, life-threatening PE during pregnancy is a rare, but potentially devastating, event. A recent systematic review included 127 cases of severe PE during pregnancy

(and until 6 weeks post-partum) treated with thrombolysis, thrombectomy, and/or ECMO. Both high- and intermediate-risk PE cases were included, and 23% of women experienced cardiac arrest. Reported survival rates were 94 and 86% following thrombolysis and surgical thrombectomy, respectively; however, these favourable rates may reflect reporting bias. Following thrombolysis, major bleeding occurred in 18 and 58% of cases during pregnancy and in the post-partum period, respectively. Finally, foetal deaths occurred in 12 and 20% of the cases following thrombolysis and thrombectomy, respectively.Thrombolytic treatment should not be used peri-partum, except in the setting of life-threatening PE. Typically, UFH is used in the acute treatment of high-risk PE.

Although the indications for vena cava filters are similar to those for non-pregnant patients, there is limited experience with their use in pregnancy and the risk associated with the procedure is increased.

Role of a multidisciplinary pregnancy heart team

A team of multidisciplinary colleagues should collaborate in the planning of antepartum, peripartum, and post-partum care pathways for women with cardiovascular diseases, including Pulmonary Embolism.

VALVULAR THROMBOSIS

Anticoagulation for women with mechanical valves during pregnancy

Preconception

- Discuss with the patient an anticoagulation strategy for pregnancy including a thorough discussion of the risks/benefits of each strategy and arrive at a preformed plan individualized based on patient preference and risk profile
- Counsel patient to monitor for pregnancy by tracking menstrual cycles and frequent pregnancy tests and contact her cardiologist as soon as pregnancy is known.

Pregnancy: Conception to 12 weeks

- If the warfarin dose is <5 mg, it may be continued
- If the warfarin dose is ≥5 mg or if the patient prefers to avoid warfarin altogether during pregnancy, switch to weight-based LMWH BID and adjust dose based on weekly peak/trough monitoring for goal peak anti-Xa levels of 0.6–1.2 IU/mL 4 hours post-dose. If trough levels are <0.6 IU/mL with therapeutic peaks, dose TID.

Pregnancy: Week 13–35

- All patients can be switched to warfarin.
- For patients who prefer to avoid warfarin during pregnancy, LMWH can be continued with careful weekly monitoring as above.

Pregnancy: Week 36

Switch patients on warfarin to LMWH or IV UFH. LMWH should be monitored weekly, with a goal peak anti-Xa level of 0.7– 1.2 IU/mL 4 hours post dose. If trough levels are <0.6 IU/mL with therapeutic peak, dose TID.

Labor and postpartum

- 36 hours prior to induction or caesarean delivery, all patients should be switched to IV UFH
- 6 hours prior to delivery, IV UFH should be stopped
- Restart IV UFH 4–6 hours after delivery (provided the risk of bleeding is not prohibitive from an obstetric perspective)
- Once safe to start long term anticoagulation from bleeding perspective, bridge with LMWH or IV UFH to warfarin with careful INR monitoring postpartum, especially in breastfeeding patients.

Anticoagulant therapy in pregnancy [43, 42]

Indications for Anticoagulation

1. Patients with mechanical heart valves

2. Chronic atrial fibrillation

3. Deep vein and pulmonary embolism

• *Warfarin:* Causes foetal skeletal stippling, craniofacial deformities, microcephaly, optic atrophy, limb defects. Exposure in first trimester causes dorsal or ventral midline dysplasia Warfarin embryopathy consists of mid face and nasal hypoplasia, optic atrophy, hypoplasia of digits, stippled epiphysis (chondrodysplasia punctata), and mental illness

• It also causes 1st trimester abortions, still births, neonatal deaths and foetal haemorrhages

Recommendations of the Seventh ACCP Consensus Conference on Antithrombotic therapy for Prophylaxis in Patients with Mechanical Heart Valves

1. Aggressive adjusted-dose UFH, given every 12 h subcutaneously throughout pregnancy; mid-interval activated partial thromboplastin time maintained at >2 x control levels, or anti-Xa heparin level maintained at 0.35 to 0.70 IU/ml.

OR

2. LMWH throughout pregnancy, in doses adjusted according to weight or as necessary to maintain a 4-h postinjection anti-Xa heparin lelevel of about 1.0 IU/ml

OR

3. UFH or LMWH, as above, until the 13th week, change to warfarin until the middle of the third trimester, then restart UFH or LMWH therapy until delivery from Bates et al .

ACCP=American College of Chest Physicians; LMWH= low molecular weight heparin; UFH=unfractionated heparin

AIM AND OBJECTIVES

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To evaluate the incidence, demography, risk factors, clinical features, diagnosis and management of thromboembolic disorder in pregnancy and puerperal period, and to study the maternal and perinatal outcomes.

The objectives of this prospective study in pregnancy and puerperium are to examine the incidence, risk factors, fetomaternal outcomes, complications of thromboembolic disorders, in Thanjavur medical college Hospital, Thanjavur from January 2020 to June 2021.

MATERIALS AND METHODS

MATERIALS AND METHODS

Institutional Ethical Committee approval was obtained for the study

STUDY DESIGN - PROSPECTIVE OBSERVATIONAL STUDY

STUDY PERIOD - 18 MONTHS (JANUARY 2020-JUNE 2021)

STUDY POPULATION - 100

INCLUSION CRITERIA - All confirmed cases (both clinical and radiological) of thromboembolic disorders in pregnancy and puerperal period during the study period, admitted at RMH and THANJAVUR MEDICAL COLLEGE HOSPITAL.

EXCLUSION CRITERIA - All patients without definitive diagnosis of thromboembolic disorders are excluded.

- Women without radiological evidence of CVT.
- Women who presented with complaints after 6 weeks puerperium.

METHODOLOGY

All confirmed cases of thromboembolic disorders admitted over a period from January 2020 to June2021 in Thanjavur Medical College Hospital, Thanjavur was studied. Basic demographic data regarding name, age, parity, number of live child, type of antenatal care, mode of delivery, were recorded. Results of the procedure with respect to incidence, risk factors and complications associated with thromboembolic disorders are inferred.

Data Collection:

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

- Age of the mother, BMI, parity, presence of predisposing factors, mode of delivery, time of presentation, treatment given, hospital course, outcome were noted. .
- Radiological confirmed cases -

  Cortical venous thrombosis-Neuroimaging in the form of CT brain and MRI brain with MRA and MRV was done in all patients with clinically confirmed cases of cortical venous thrombosis

  Deep vein thrombosis-Doppler ultrasound is done in patient with Deep vein thrombosis

  Pulmonary Embolism-CT Pulmonary Angiogram was performed.

  Mitral valve thrombosis-Echocardiography done

  Brachial Artery thrombosis-Doppler sonography

  Cephalic vein thrombosis - Doppler sonography

- Investigations like complete blood count, erythrocyte sedimentation rate (ESR), blood urea, blood sugar, serum creatinine, serum electrolytes, lipid profile, X-ray chest, Electrocardiogram, ELISA for Human Immunodeficiency Virus (HIV),VDRL, coagulation profile including

bleeding time, clotting time, prothrombin time, activated partial thromboplastin time were done in all patients and investigations like antinuclear antibody (ANA), antiphospholipid antibodies, done in certain patients as needed.

OBSERVATION AND RESULTS

OBSERVATION AND RESULTS

A total of 100 cases of Thrombosis were evaluated in the present study

Table1. Based on Period of Gestation
N=100

S.No	Period	Patients	%
1	Antenatal	10	10
2	Post Abortal	10	10
3	Post Natal	29	29
4	Post Caesarean Section	51	51
	Total	100	100

In the present study, majority of the thrombosis contribute to puerperal group, post Caesarean section 51%, post-natal 29% and post-abortal 10%.

Figure1. Based on Period of gestation

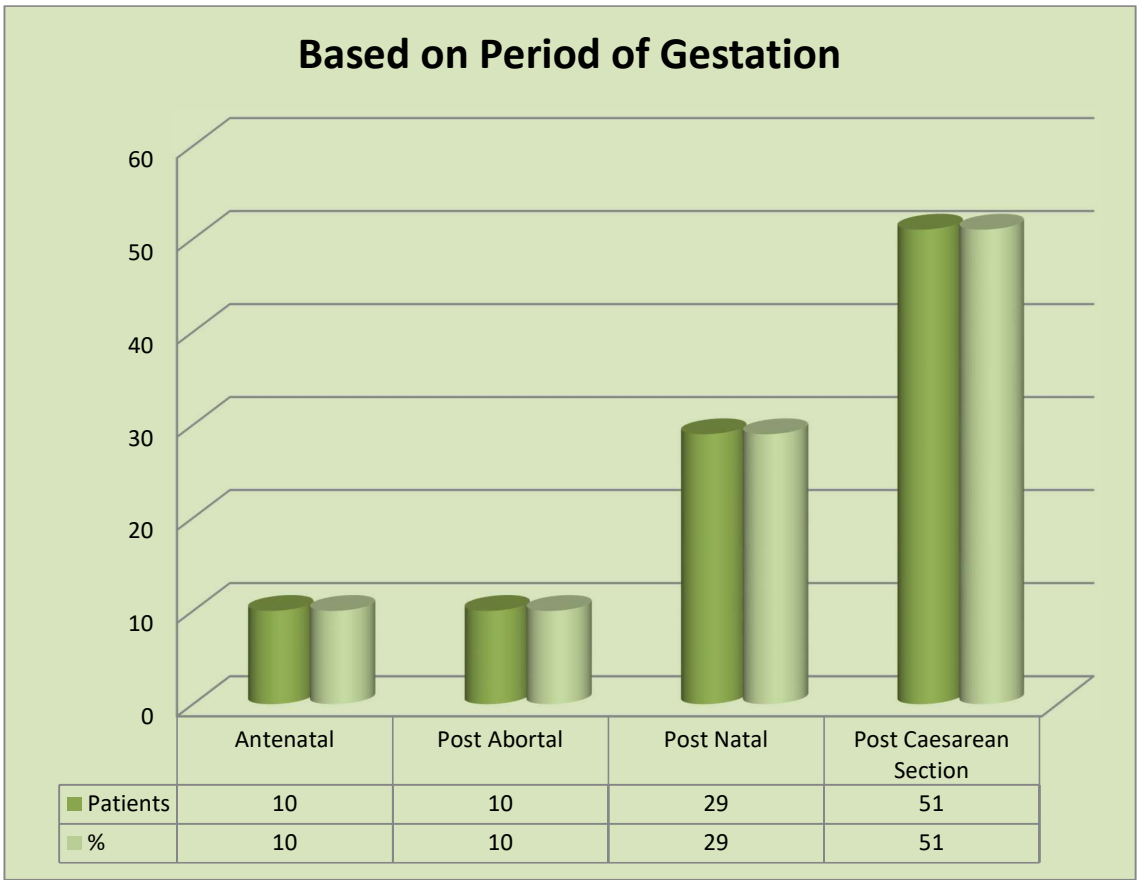


Table2. Based on the mode of Delivery

N=80

S.No	MODE OF DELIVERY	PATIENTS	PERCENTAGE
1	NORMAL VAGINAL DELIVERY	26	32.5
2	ASSISTED VAGINAL DELIVERY	3	3.75
3	CAESAREAN SECTION	51	63.5

In the present study, majority of the thrombosis occurs following caesarean section 63.5%, normal vaginal delivery 32.5%, and assisted vaginal delivery 3.75%

Figure2. Based on the mode of Delivery

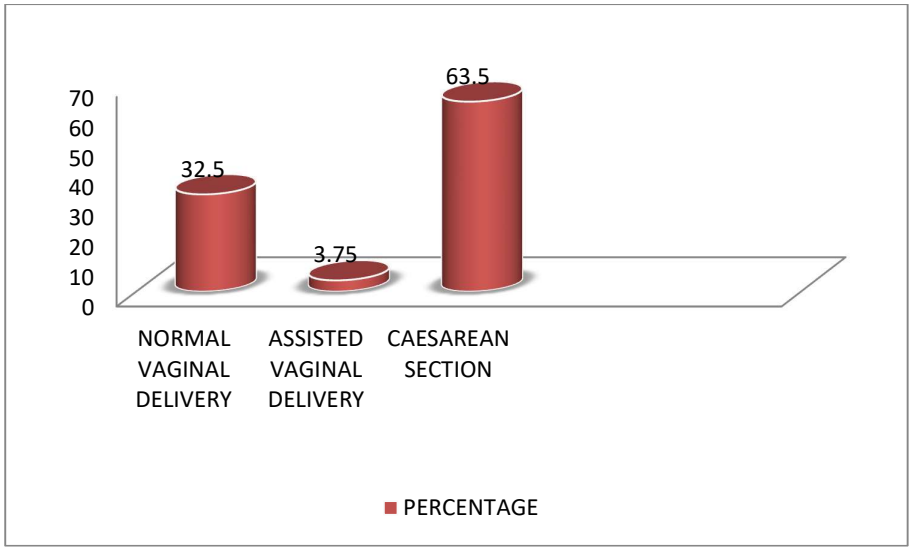


Table3. Based on Parity

S.No	Based on Parity	Patients	%
1	Primi	37	37
2	Multi	63	63
	Total	100	100

In the present study, thromboembolism risk increases during multi para contributing to 63% (out of 100 cases).

Figure3. Based on Parity

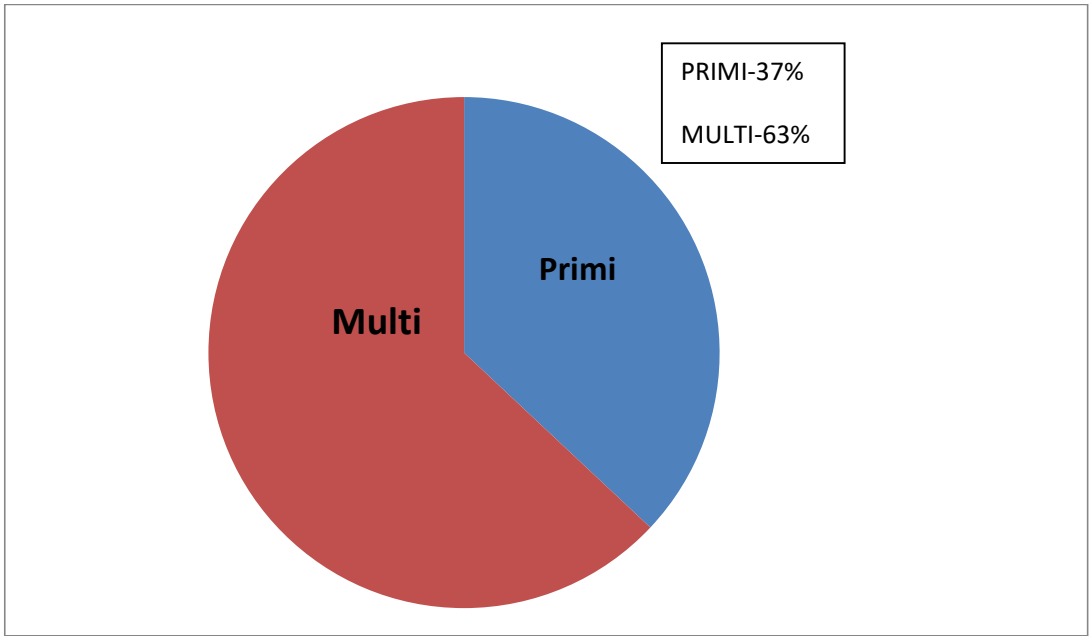


Table4. Age
N=100

S.No	Age	Patients
1	16 -25	34
2	26-35	56
3	> 35	10
	Total	**100**

Majority of the Thromboembolic patients are in the age group 26-35 contributing to 56 patients. The youngest being 16 years and the oldest being 45 years. Mean age found to be 30.5

Figure4. Age

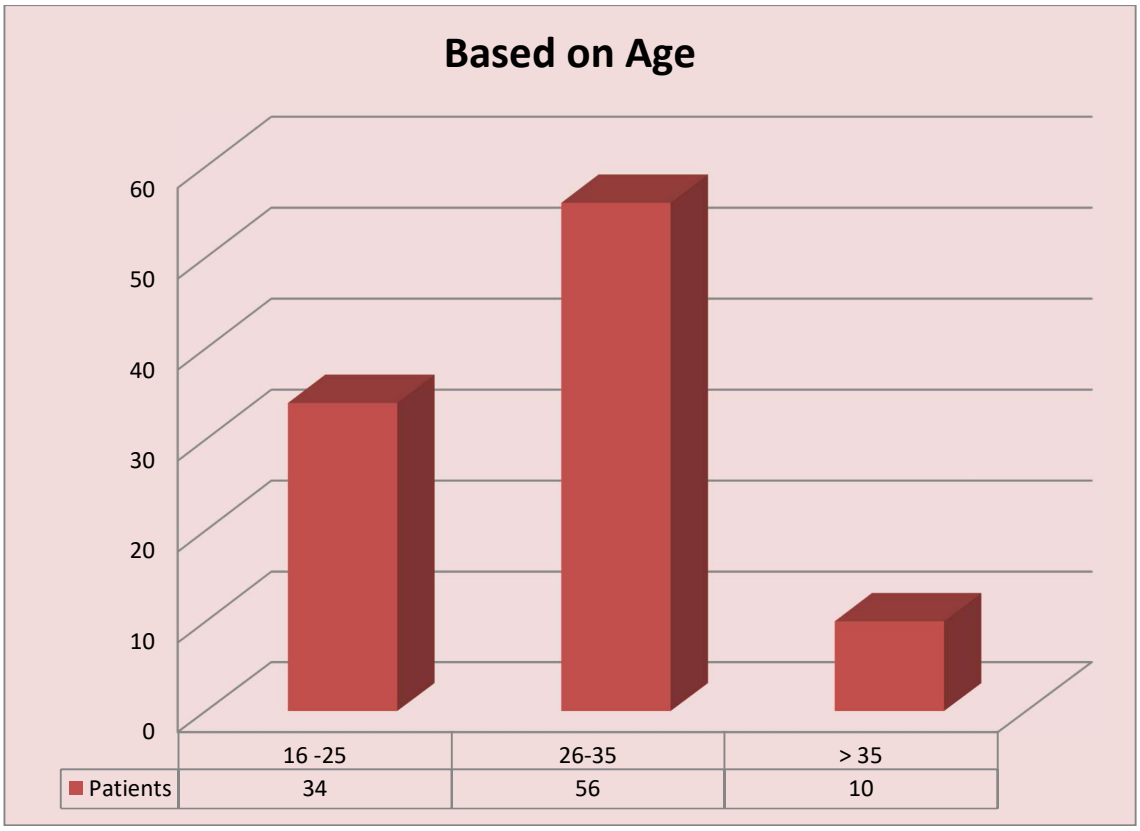


Table5. BMI
N=100

S.No	BMI	Patients	%
1	<18.5	2	2
2	18.6 - 25	40	40
3	25.1 - 30	42	42
4	> 30.1	16	16
	Total	**100**	**100**

In the present study, majority of the patients are overweight contributing to 42%, obese patients contributing to 16%

Figure5. BMI

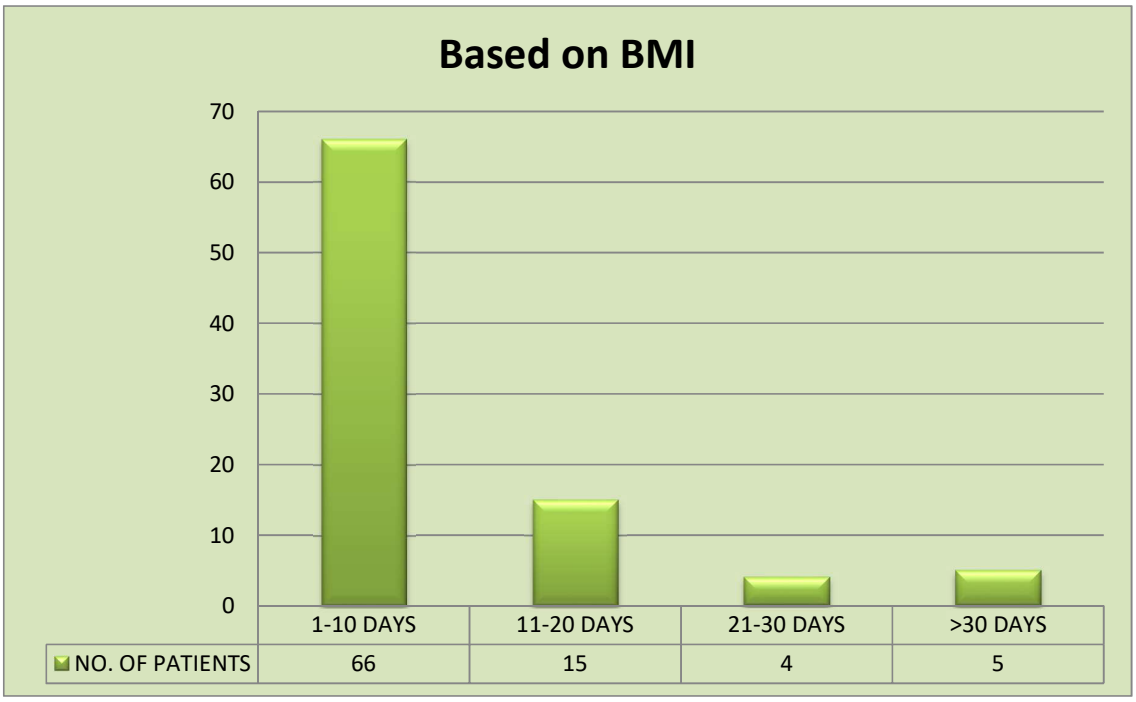
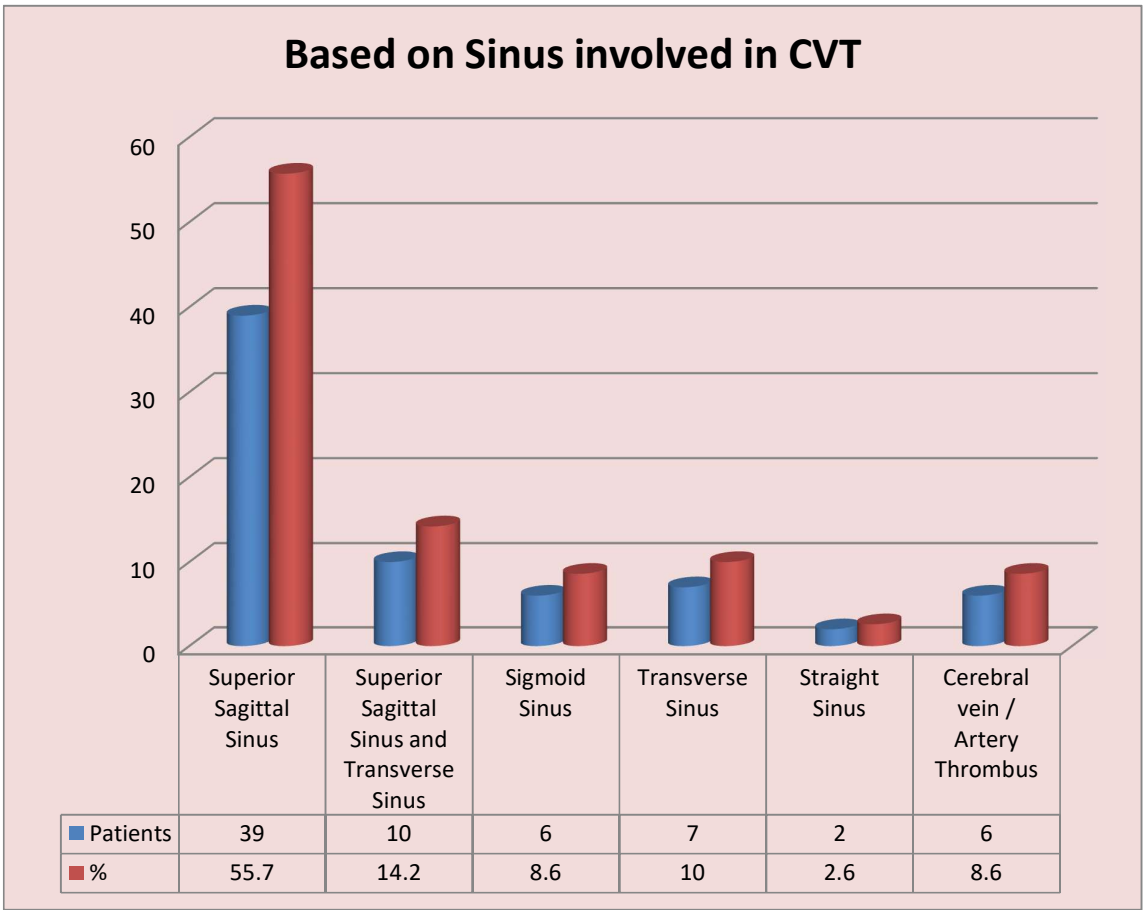

Based on BMI
70
60
50
40
30
20
10
0
1-10 DAYS
11-20 DAYS
21-30 DAYS
>30 DAYS
NO. OF PATIENTS
66
15
4
5

Table6. Based on Sinus involved in CVT

S.No	Based on Sinus involved in CVT	Patients	%
1	Superior Sagittal Sinus	39	55.7
2	Superior Sagittal Sinus and Transverse Sinus	10	14.2
3	Sigmoid Sinus	6	8.6
4	Transverse Sinus	7	10
5	Straight Sinus	2	2.6
6	Cerebral vein Thrombus	6	8.6

In the present study, most common sinus involved was Superior Sagittal Sinus contributing to 55.7%, followed by both superior sagittal sinus and transverse sinus contributing to 14.2% among 70 patients

Figure6. Based on Sinus involved in CVT



	Superior Sagittal Sinus	Superior Sagittal Sinus and Transverse Sinus	Sigmoid Sinus	Transverse Sinus	Straight Sinus	Cerebral vein / Artery Thrombus
Patients	39	10	6	7	2	6
%	55.7	14.2	8.6	10	2.6	8.6

Table7. Type of thrombosis

N=100

S.NO	PATIENTS	No of Patients
1.	ARTERIAL THROMBOSIS	1
2.	VENOUS THROMBOSIS CVT-70 DVT-21 MESENTERIC VEIN THROMBOSIS-1 CEPHALIC VEIN THROMBOSIS-4	96
3.	PULMONARY EMBOLISM	1
4.	MITRAL VALVE THROMBOSIS	2

In the present study, during my study period, majority of the thrombosis are venous thrombosis 96 cases, arterial thrombosis 1 case reported, pulmonary embolism -1 case, Mitral valve thrombosis-2 cases reported from January 2020 to June 2021.

Figure7. Depending on Type of thrombosis

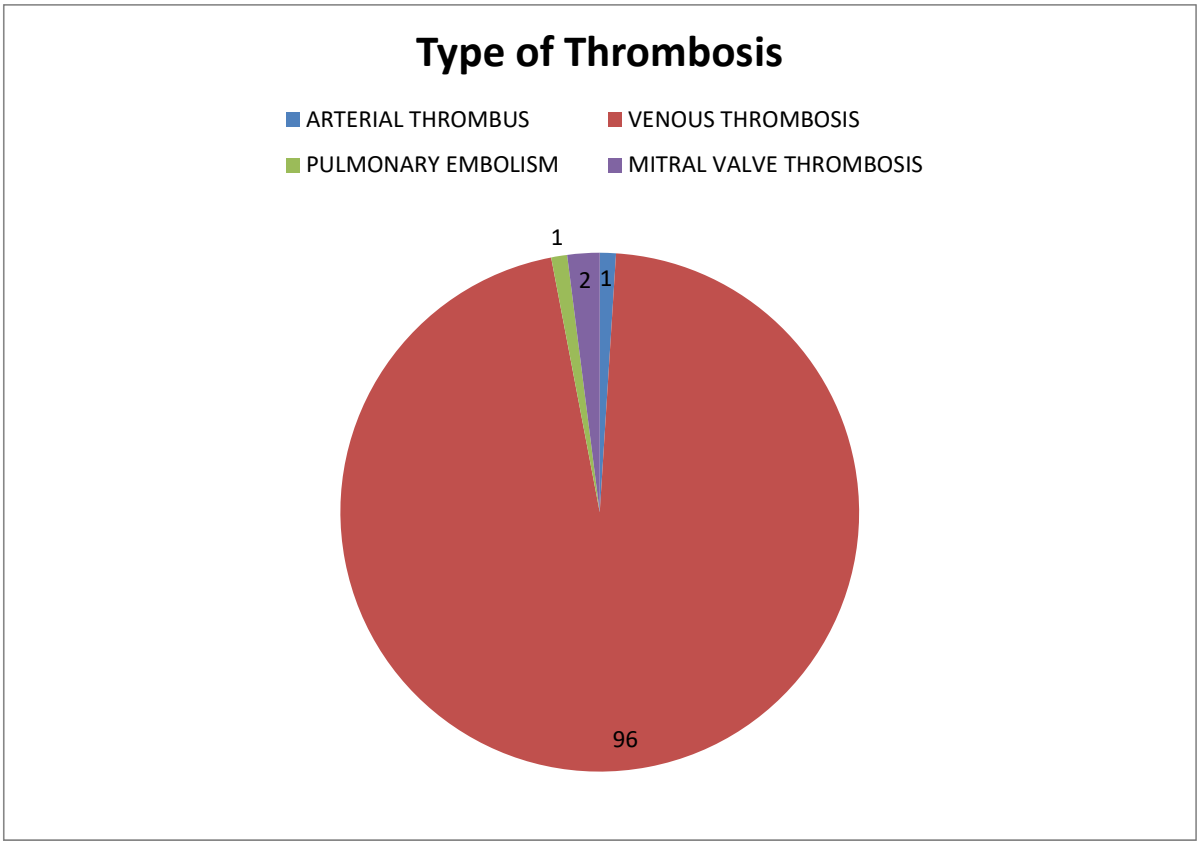


Table8. Based on period of onset of symptoms in puerperium

N=90

DURATION	NO. OF PATIENTS	PERCENTAGE
1-10 DAYS	66	73.3
11-20 DAYS	15	16.6
21-30 DAYS	4	4.4
>30 DAYS	5	5.5

In the present study out of 90 puerperal patients, 73.3% of thromboembolic disorders occur during 1-10days after delivery,16.6% occurs 11-20 days after delivery,4.4%occurs 21-30 days after delivery,5% occurs 30 days after delivery.

Figure8. Based on period of onset of symptoms in puerperium

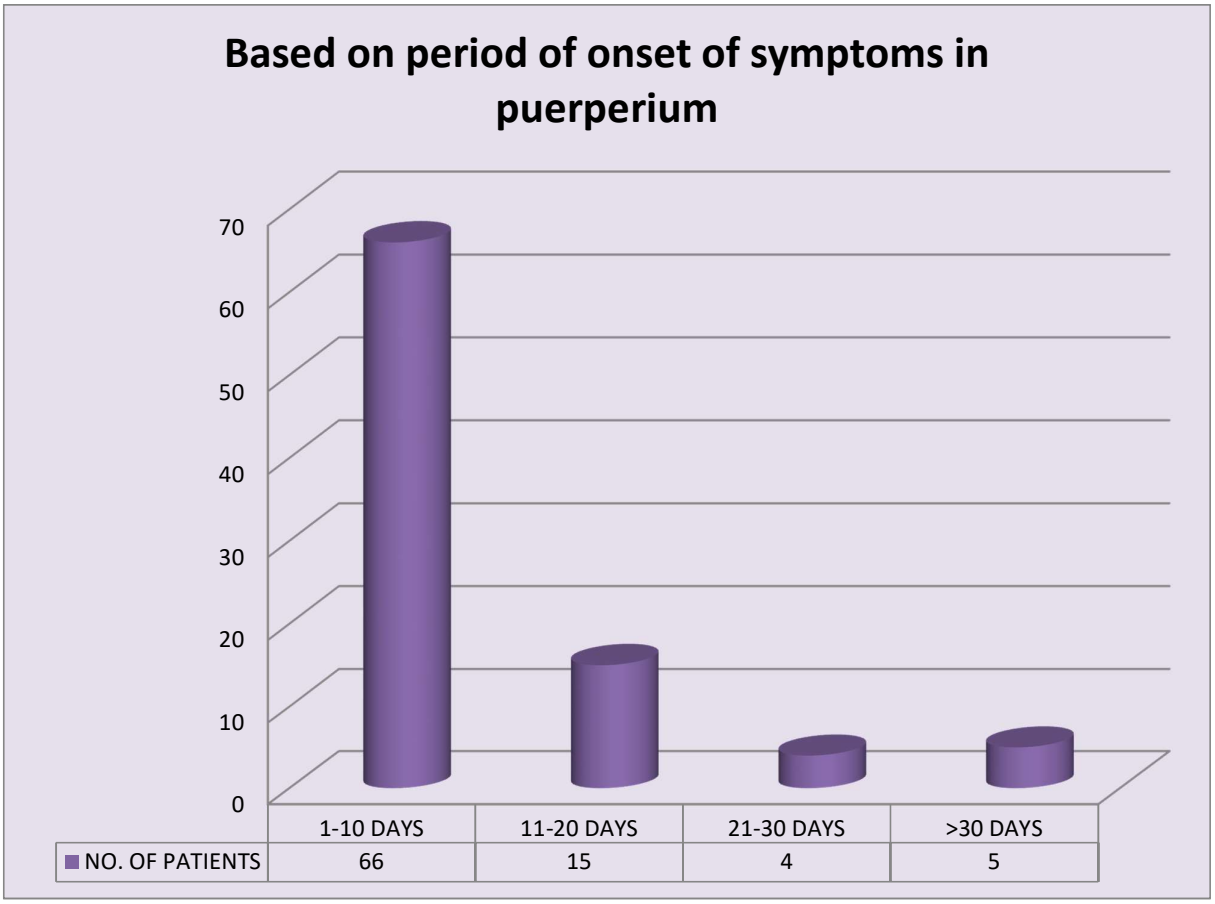


Table9. Based on HAEMOGLOBIN on admission
N=100

S.No	HB gm/dl	Patients	%
1	5 to 8	19	19
2	8.1 - 10	54	54
3	> 10	27	27
	Total	100	100

In the present study, out of 100 patients majority of the patients are anaemic with moderate anaemia of 54%, severe anaemia of 19%.

Figure9. Based on HB on admission

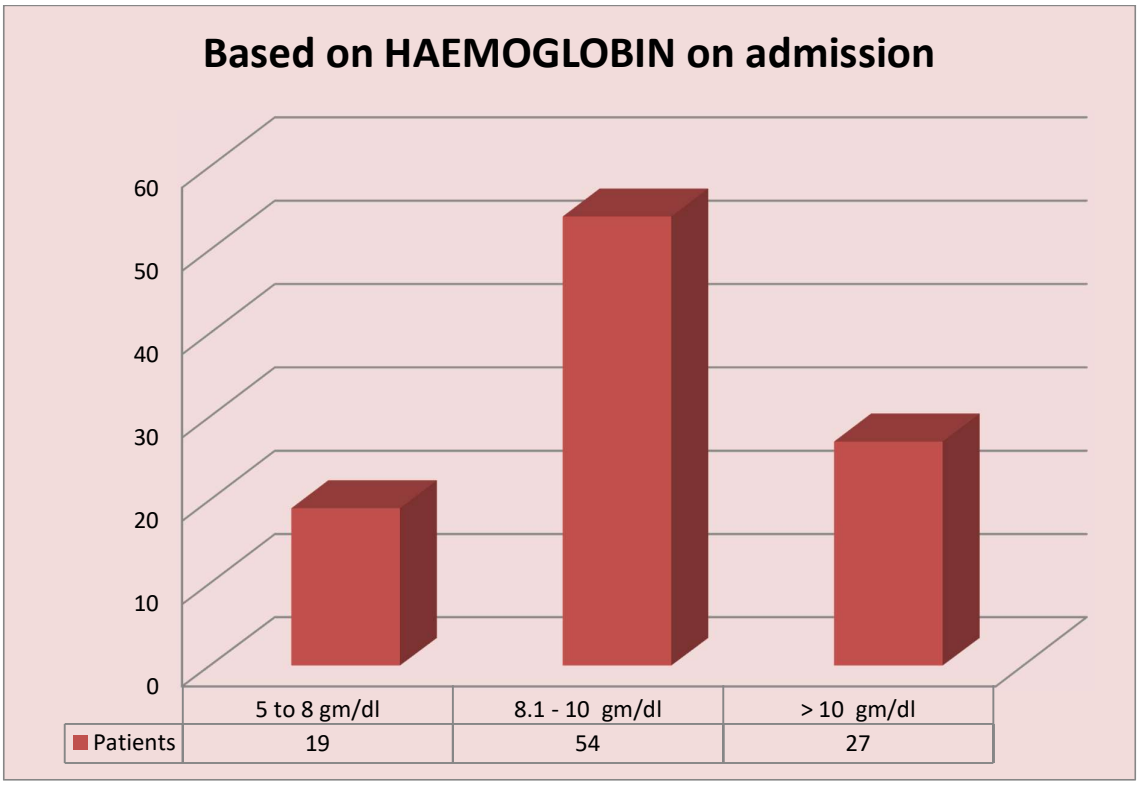


Table10. Incidence of Arterial thrombosis (BRACHIAL ARTERY THROMBOSIS)

S.NO	PATIENTS	No of Patients
1.	ANTENATAL (1-BRACHIAL ARTERY THROMBOSIS)	1

In our hospital, one case of BRACHIAL ARTERY THROMBOSIS reported during antenatal period, during the study period.

A 29/f, primi, with 10 weeks gestation, known case of Rheumatic Heart disease, presented with complaints of left upper limb pain and swelling, Doppler ultrasonography revealed left brachial artery thrombosis, left brachial embolectomy done at TMCH and the patient discharged alive.

Table11. Incidence of Pulmonary Embolism

S.NO	PATIENTS	No of Patients
1.	post caesarean (1-**Pulmonary Embolism**)	1

In our hospital, one case of Pulmonary Embolism reported during the study period.

A 31year/f,multi,P3L3/POST LSCS/POD -30/presented with complaints of breathing difficulty, Pulmonary Angiography revealed Right lower lobe pulmonary embolism, Thrombolysis done with inj.streptokinase, then changed to warfarin and the patient discharged alive.

Table12. Incidence of Mitral valve thrombosis

N=2

S.NO	Mitral valve thrombosis PATIENTS	PATIENTS Count	%
1	POST ABORTAL	1	50
2	POST NATAL	1	50

In our hospital, two cases of Rheumatic Heart disease with history of mitral valve replacement on anticoagulants with artificial Mitral valve thrombosis reported, during the study period.

Figure12. Incidence of Mitral valve thrombosis

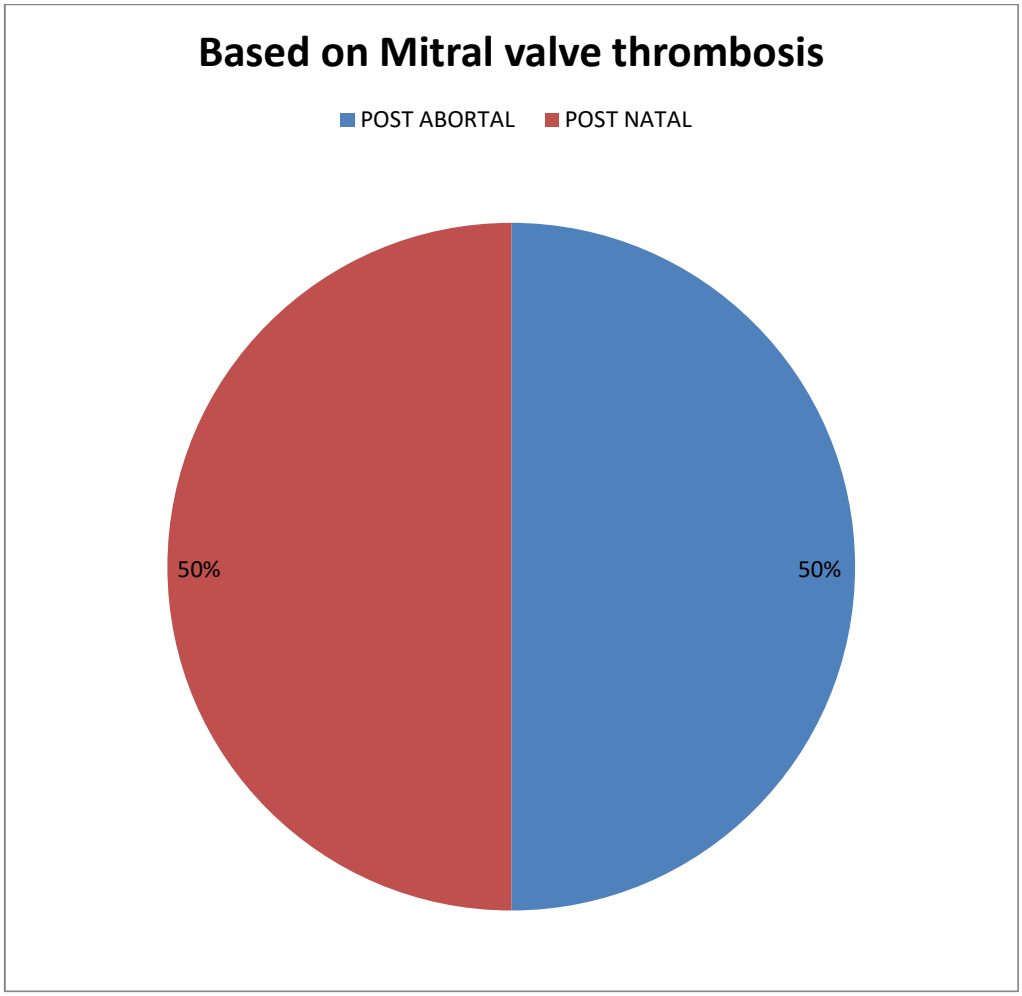
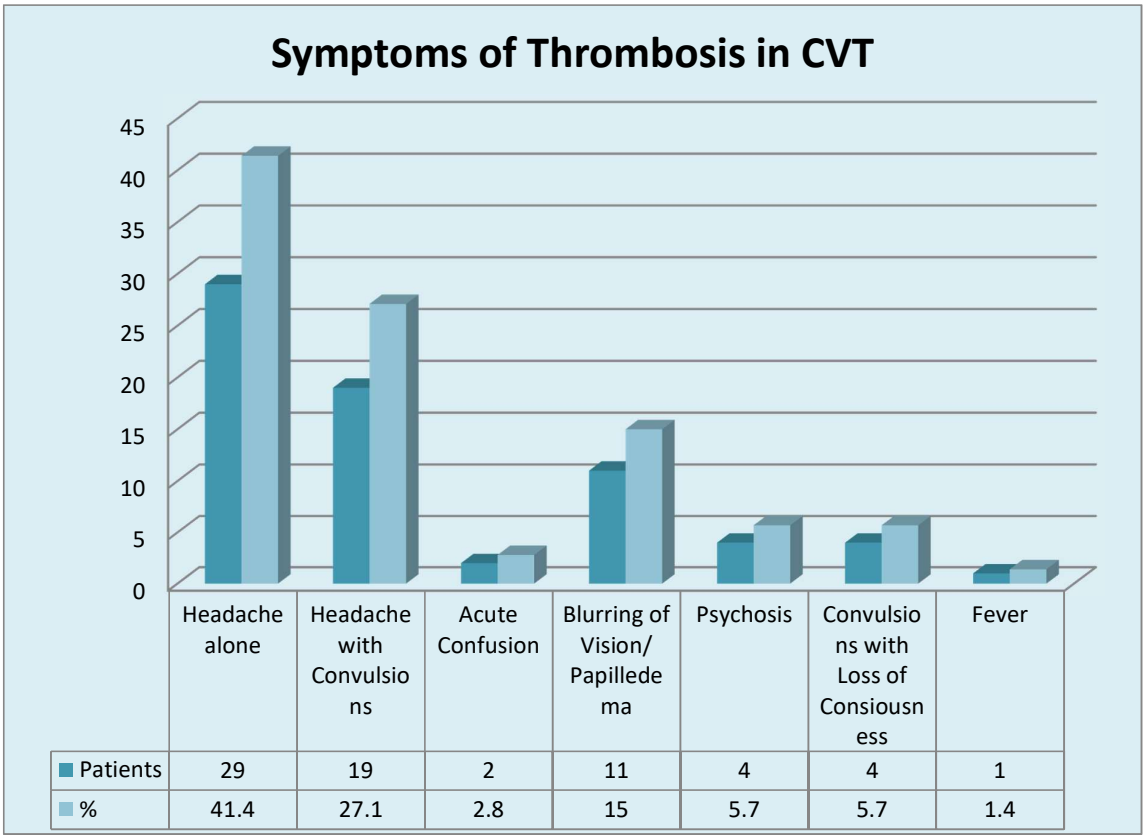


Table13. Symptoms of Thrombosis in cortical vein thrombosis
N=70

S.No	Symptoms of Thrombosis	Patients	%
1	Headache alone	29	41.4
2	Headache with Convulsions	19	27.1
3	Acute Confusion	2	2.8
4	Blurring of Vision/ Papilledema	11	15
5	Psychosis	4	5.7
6	Convulsions with Loss of Consciousness	4	5.7
7	Fever	1	1.4

In the present study, most common symptom is headache contributing to 41.4% followed by convulsions contributing to 27%, Blurring of vision contributing to 15%.

Figure13. Symptoms of Thrombus in cortical vein thrombosis



	Headache alone	Headache with Convulsions	Acute Confusion	Blurring of Vision/ Papilledema	Psychosis	Convulsions with Loss of Consiousness	Fever
Patients	29	19	2	11	4	4	1
%	41.4	27.1	2.8	15	5.7	5.7	1.4

Table14. Based on RISK FACTORS

RISK FACTORS	PATIENTS	%
1. HYPERTENSION(GHT/CHRONIC TH)	54	54
2. HYPOTHYROID	8	8
3. DIABETES(GDM/DM)	12	12
4. PROLONGED IMMOBILIZATION	8	8
5. DEHYDRATION	5	5
6. HEART DISEASE	3	3
7. APLA	3	3
8. COVID CASES	2	2
9. SEPSIS	3	3
10. VENTILATOR	2	2

In our study we found Hypertensive disorders (54%), diabetes (12%), Dehydration (5%), sepsis (3%), ventilator (2%) are the most important predisposing factor for thrombosis.

Figure14. Based on RISK FACTORS

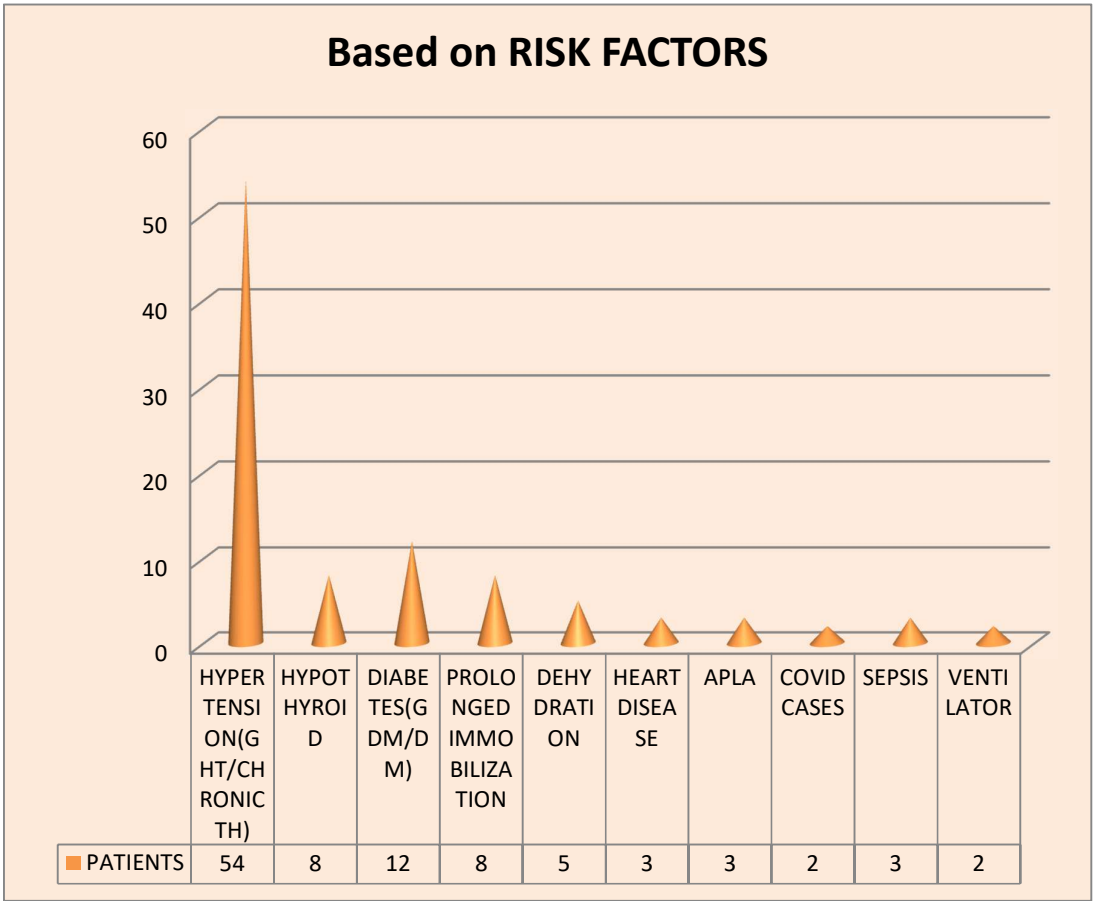


Table15. Based on treatment modality
N=100

Treatment Mode	PATIENTS	%
LMWH	47	47
INJ.UNFRACTIONATED HEPARIN	53	53

In the present study, in our hospital, 47% of patients received LMWH, 53% patients received INJ.UNFRACTIONATED HEPARIN.There is no difference in prognosis, as all these patients had good prognostic outcomes.

Figure15. Based on treatment modality

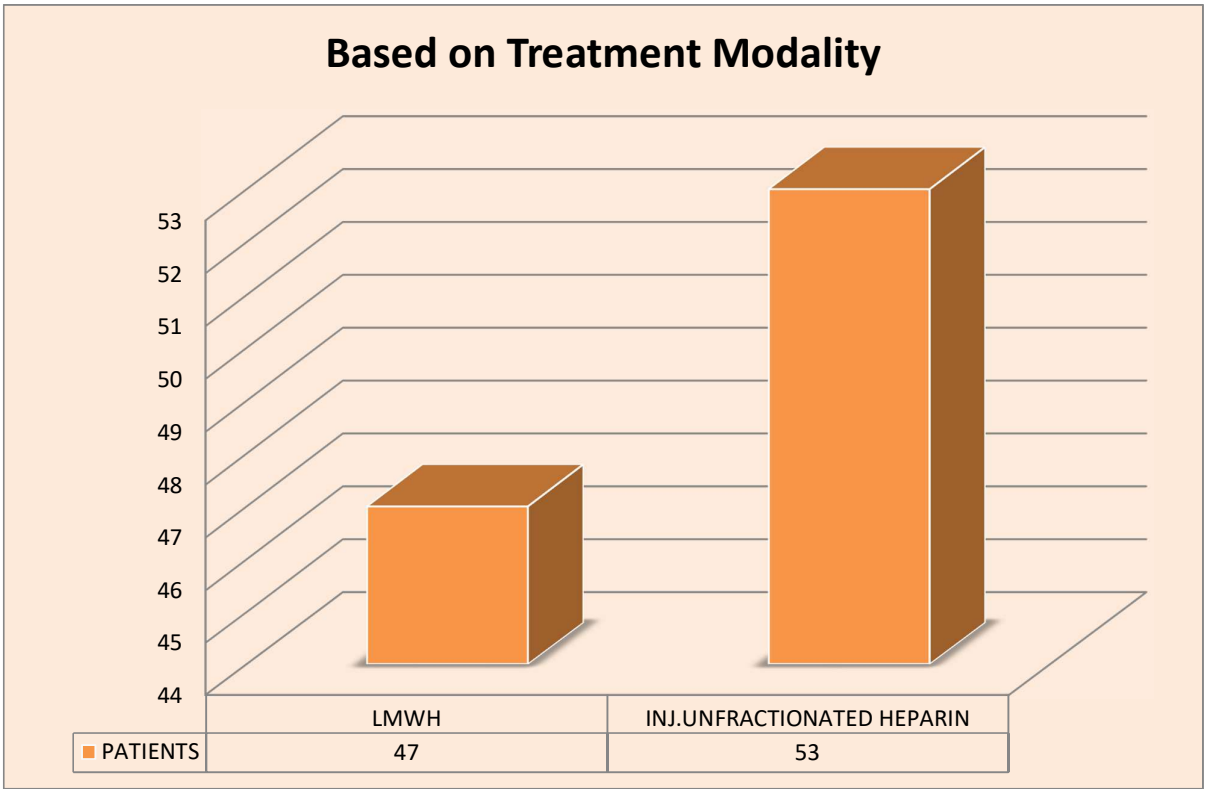


Table16. Based on perinatal outcome

S.NO	b.wt	total
1	1.0 to 2kg	12
2	2.0 to 3.okg	42
3	>3kg	26

In the present study of out of 80 babies delivered, 42 babies between birth weight 2.0-3.0kg, 26 babies >3kg, 12 babies between birth weight 1.0-2.0 kg out of this 80 babies, 74 babies discharged alive.

Figure16. Based on perinatal outcome

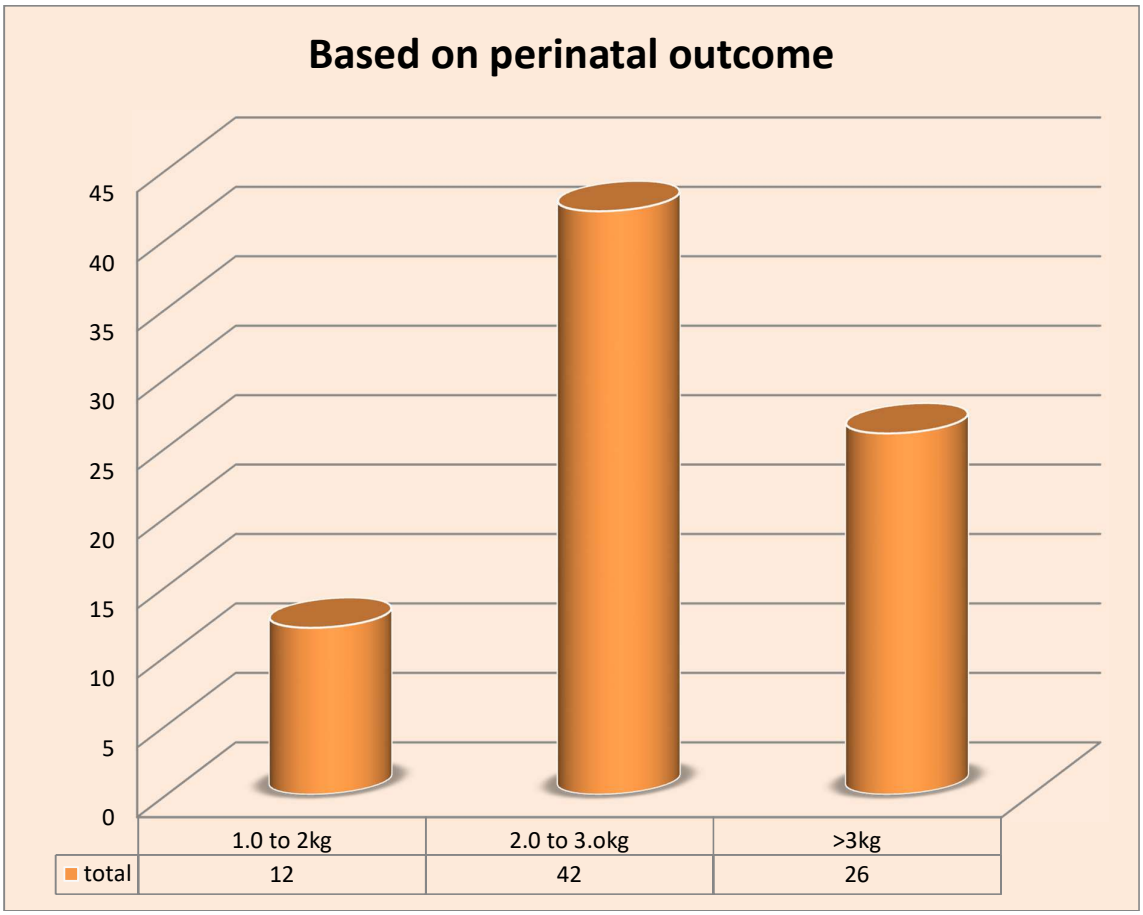


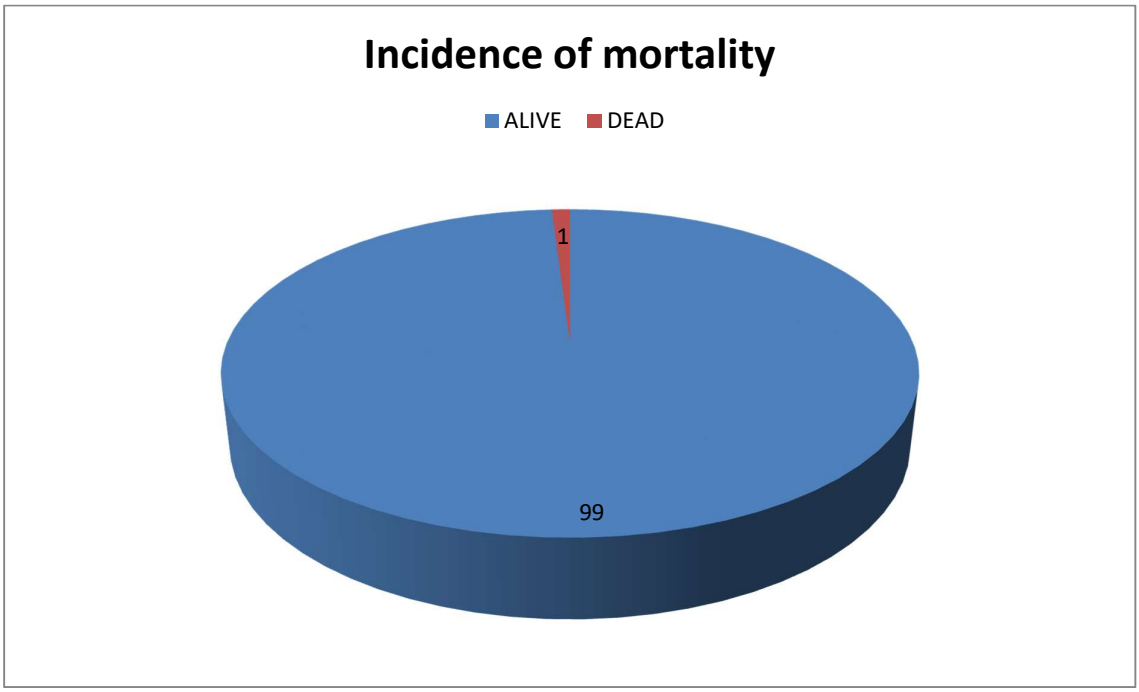
Table17. Incidence of mortality

Mortality	PATIENTS	%
ALIVE	99	99
DEAD	1	1

In the present study of 100 patients, 1 patient died and 99 patients were alive comprising 99%. We observed that 99% of patients showed good prognosis in the form of recovery.

The mortality was 1%. A case of 28/f,P1L0/Emergency LSCS done at Jeyankondam GH/COVID POSITIVE/on POD-1,patient presented with acute abdomen and vomiting, Laparotomy revealed Mesenteric vein thrombosis with extensive small bowel gangrene, small bowel resection anastomosis done ,but patient succumbed to the illness.

Figure17. Incidence of mortality



DISCUSSION

DISCUSSION

In this study, totally 100 cases were studied.

1. The present study showed highest thromboembolic disorder within the age group of 26 to 35 years.
2. Increased BMI increases the risk of thromboembolism.
3. In the present study, majority of the patients are overweight contributing to 42%, with obese patients contributing to 16%.

   Finkelstein et al proposed, there is positive association between increasing BMI and escalating risk of thromboembolism.

   Olie et al found increasing BMI was related to increasing incidence of recurrent thromboembolism.
4. In the present study, majority of the thromboembolic disorder contribute to puerperal group, post LSCS 51%, post-natal 29% and post-abortal 10%,ante natal 10%
5. In the present study, out of 100 patients majority of the patients are anaemic with moderate anaemia of 54%, severe anaemia of 19%.
6. In our hospital, one case of pulmonary venous thrombosis reported following post LSCS period. Patient survived and did follow up for 6 months complete.

   In a study by Chang et al, long term outcome in pregnancy related DVT; they found 42% of women with lower limb extremity developed pulmonary thromboembolism.

7. In the present study, out of 70 patients with cortical vein thrombosis, majority of the cortical vein thrombosis occurs at the post-natal period contributing to 21.4%, followed by post LSCS contributing to 61.4%.

   Kumar s et al.[37] (2003) had found that 65 out of 85 cases (76%) of Puerperal CVT. The present study of CVT group is comparable with Kumar S et al.

8. Headache was the most common symptom in the present study accounting for 41.4% of patients. The present study was comparable with Kumar S et al. (2003) with 66% of patients

Study by James et al found only 20% of patients of CVT related to pre eclamptic toxemia exhibit sustained diastolic pressure of >105mmHg before the stroke develops[39.]

Authors	Number of cases studied	Percentage with headache
Daif et al. (1994)	40	82
Nagaraj et al. (1999)[20]	905	71
Kumar S et al. (2003)	85	66
Neki S et al. (2003)	14	85.5
Mehta SR et al. (2003)	43	77.8
Strolz E et al. (2005)	79	73.4
Present study	70	41.4

9. In the present study, 27% of cases had seizures which are comparable with Mehta SR et al.

Authors	Number of cases studied	Percentage with seizures
Kumar S et al. (2003)	85	67
Mehta SR et al. (2003)	43	26.6
Strolz et al. (2005)	79	39.2
Present study	70	27.1

10. In the present study of CVT group, the superior sagittal sinus is most commonly involved accounting 55.7% followed by transverse sinus with 10%, followed by cerebral vein 8.6% which is comparable with Ameri et al (72%) and Strolz E et al. (72.2%)

Sinuses involved	Daif et al. (1994)	Strolz E et al. (2005)	Present study
Superior sagittal sinus	85%	72.2%	55.7%
Transverse sinus/Sigmoid sinus	2.5%	38%	10%
Straight sinus	7%	7.6%	2.6%
Internal cerebral vein	10%	6.3%	8.6%

11. Prakash BC and Bansal C[18] reported that postpartum CVT occurred 7-10 days after delivery, which is similar to our study group.

12. Brown et al showed women with pre eclampsia were 60%, more likely to have ischemic stroke than women without it.

    This study is more comparable with Brown et al, in which women with hypertensive disorders were 54%

13. All cases of DVT presented with the same symptoms of leg pain, and swelling.

14. In the present study, thromboembolism risk increases during multipara contributing to 63% (out of 100 cases).

15. In the present study, in our hospital, 47% of patients received LMWH, 53% patients received INJ.UNFRACTIONATED HEPARIN. There is no difference in prognosis, as all these patients had good prognostic outcomes.

SUMMARY

SUMMARY

Important clinical signs to suggest Thromboembolic disorder are wide.

Thromboembolism is one of the most serious complications of puerperium and it is a recognized rising cause for maternal mortality.

Though hypercoagulable state of pregnancy creates the risk, it is possible to prevent cerebral venous thrombosis by timely identification and correction of risk factors. Important risk factors are anaemia, preeclampsia, infection and dehydration.

According to the study,

- Highest incidence of Thromboembolic disorders was observed in age group 26- 35years.
- Maximum number of thromboembolic disorder occurred in Multiparous women.
- Majority of women with thromboembolic disorder are overweight contributing to 42%,followed by obesity 16%
- Incidence of postpartum thromboembolism was highest in first week of postpartum with 73.3%.
- Out of 100 patients, 54% of the patients are moderate anaemia, 19% of patients are with severe anaemia
- The commonest risk factor noted was hypertensive disorders of pregnancy 54%.
- Puerperium forms important causes of thromboembolic disorder.

- Headache was the most common symptom followed by convulsions in CVT group
- Radiologically most common sinus involved in CVT was superior sagittal sinus 55.7%
- In postpartum cerebral venous thrombosis Superior sagital sinus was most commonly involved.
- Management with Unfractionated heparin/injection Low molecular weight heparin followed by oral anticoagulants is appropriate and the prognosis is generally favourable.

CONCLUSION

CONCLUSION

- The risk factors should be corrected accordingly like anaemia with blood transfusion, BP monitoring, early mobilisation in the puerperal period, dehydrated patients with Intravenous fluids.
- The risks of thromboembolism (0.05 Vs. 12%) are higher in incidence as compared to non-obese
- During every antenatal visits, weight & B. P. recording with appropriate sized cuff. Obese women should be evaluated for gestation diabetes at 1st visit. Testing should be repeated in second & third trimester if previous findings were normal.

- Women with severe obesity(BMI>35kg/m2) with one additional risk factor for hypertensive disease should be prescribed Aspirin 75mg per day from 12 weeks of gestation in order to prevent further risks of thromboembolism.

- Heparin is restarted 4-6 hours after normal delivery and 24 hours after caesarean section. Oral anticoagulants can be started concomitantly. Breast feeding is not contraindicated. Patient should be encouraged to ambulate early and is discharged after a week if stable. Appropriate contraceptive advice should be given before the woman is discharged

- Thromboprophylaxis in Peripartum Period x Anticoagulation to be stopped before elective LSCS and labor. UFH to be stopped 12 hours prior, LMWH 24 hours prior x Platelet count should be done with UFH therapy x For epidural analgesia last dose of prophylactic LMWH should not be earlier than 12 hours and 6 hours for UFH x UFH has shorter half-life, hence managing in labor is easier. So some people stop LMWH at 36 weeks and convert on UFH x Anticoagulation to be restarted 4–6 hours after normal delivery, 6–12 hours after LSCS x All patients on anticoagulations should be warned regarding vaginal bleeding. Baseline a PTT and platelet count should be done. Protamine sulfate should be ready. Fresh frozen plasma should be ready. Post-Partum Haemorrhage should be avoided.

- Thromboprophylaxis in Peripartum Period x Anticoagulation to be stopped before elective LSCS and labor. UFH to be stopped 12 hours prior, LMWH 24 hours prior x Platelet count should be done with UFH therapy x For epidural analgesia last dose of prophylactic LMWH should not be earlier than 12 hours and 6 hours for UFH x UFH has shorter half-life, hence managing in labor is easier. So some people stop LMWH at 36 weeks and convert on UFH x Anticoagulation to be restarted 4–6 hours after normal delivery, 6–12 hours after LSCS x All patients on anticoagulations should be warned regarding vaginal bleeding. Baseline a PTT and platelet count should be done. Protamine sulfate should be ready. Fresh frozen plasma should be ready. PPH should be avoided.

- On confirmation of thromboembolism, the following treatment is started:
    - Admission and monitoring in High dependency unit
    - Supportive treatment in form of hydration, analgesics and antibiotics
    - High quality graduated elastic compression stockings should be given for patients with DVT

- Risk factors like anaemia, 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. Early diagnosis and initiation of treatment reduces the mortality.

ANNEXURE

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BIBLIOGRAPHY

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PROFORMA

HISTORY:

Name:

Age:

IP No.:

Address:

BMI:

Time of presentation:

Mode of delivery:

Co- morbidity:

Symptoms:

Headache -

Nausea / Vomiting /Altered sensorium-

Fever -

Convulsions - Neurological deficit - Visual disturbance-

Difficulty in breathing/cough

Limb swelling

Pain in upper limb/lower limb

Past History:

HT / PIH / DM / Heart disease / Seizure disorder / TIA / Bleeding diathesis / Migraine / Repeated abortions / OCP intake/ Auto immune disorders

Any previous history of thromboembolism

Drug history:

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 –

CVT PATIENTS-

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 –

TSH-

Chest X-ray -

ECG –

HIV and VDRL -

APLA-

D DIMER-

LDH-

S.FIBRINOGEN LEVELS-

PT/INR-

APLA-

Radiological investigations:

ECHOCARDIOGRAPHY

DOPPLER SONOGRAPHY/CT Brain /MRI/ MRA & MRV

DIAGNOSIS:

PERINATAL OUTCOME:

TREATMENT:

PATIENT CONSENT FORM

STUDY TITLE: "**A STUDY OF THROMBOEMBOLIC DISORDERS COMPLICATING PREGNANCY AND PUERPERIUM AND ITS MATERNAL AND FETAL OUTCOMES - A PROSPECTIVE OBSERVATIONAL STUDY**"

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY, THANJAVUR MEDICAL COLLEGE, THANJAVUR

PARTICIPANT NAME: **AGE:** **SEX:**

I.P. NO:

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

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

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

I hereby consent to participate in this study.

Time: Patient name:

Date:

Place: Signature / Thumb Impression of Patient:

Name and signature of the Investigator

KEY TO MASTER CHART

BMI → Body Mass Index

GTCS → Generalised Tonic Clonic Seizure

DVT → Deep Vein Thrombosis

CVT → Cortical Vein Thrombosis

AN → Ante Natal

PA → Post Abortal

PN → Post Natal

LSCS → Lower Segment Caesarean Section

PND → Post Natal Day

POD → Post-Operative Day

MA → Mother Alive

BA → Baby Alive

BD → Baby Dead

GHT →Gestational Hypertension

GDM → Gestational Diabetes Mellitus

RHD → Rheumatic Heart Disease

APLA → Anti Phospo Lipid Antibody

HB → Haemoglobin

IP No. → Inpatient number

LOC → Level of consciousness

SSS → Superior sagittal sinus

TS → Transverse sinus

MASTER CHART

S.No	Name	Age	I.P.No	OBS Code	Antenatal/Postnatal/Post LSCS/Post Abortal	Period of Gestation	BMI	Risk Factors	Clinical symptoms	Type of Thrombosis	HB on Admission	Radiological findings	Management	Perinatal Outcome	Outcome of the patient
1	Priyadashini	18	35448	P1L1	Post LSCS	POD1	24.10	Subclnicial hypothyroid	Headache with convulsions	CVT	14	SSS and TS	UFH	BA	MA
2	Kayalivizhi	23	74760	P2L2	Post LSCS	POD5	27.90	Subclnicial hypothyroid	Leg Swelling Diffuse Swelling	DVT	9.7	Ileofemoral DVT	UFH	BA	MA
3	Chithra	31	13201	P3L3	Post LSCS	POD2	27.60	Subclnicial hypothyroid	Diffculty in Breathing	Pulmonary Embolism	9.7	Right lower lobe pulmonary embolism	UFH	BA	MA
4	Vimilarani	33	85901	P3L3A1	PA	POD2	25.40	Post MVA	Headache	CVT	9.7	SSS and TS	UFH	PA	MA
5	Sathyabama	39	4515	P3L3	Post LSCS	POD1	25.80	Recurrent Chronic HT	Headache	CVT	9.7	SSS and TS	UFH	BA	MA
6	Sathya	25	4595	P1L1A2	Post LSCS	POD 8	28.30	GHT	Acute psychosis	CVT	8.7	SSS and TS	UFH	BA	MA
7	Shanthi	37	4511	P3L3A2	PA	PAD 11	18.10	RHD Heart disease	GTCS with LOC	CVT	8.7	SSS and TS	UFH	PA	MA
8	Abirami	26	3872	P3L2	LN	PND20	23.30	GHT	GTCS with LOC	CVT	7.5	SSS and TS	UFH	BA	MA
9	Thenmozhi	35	44711	P2L2A1	PA	PAD 12	24.80	APLA	Headache	CVT	10.6	SSS and TS	UFH	PA	MA
10	Keerthiga	22	8062	P2L2	Outlet forcesps Delivery - LN	PND35	20.00	RHD Heart disease	Fever	Mitral Valve Thrombosis	11.5	Valvular Thrombosis	UFH	BA	MA
11	Vijaylakshmi	21	14930	P1L1A1	Post LSCS	POD5	22.00	Imminat Eclampsia GHT	Headache, Anemia PP Eclampsia	CVT	9.3	SSS and TS	UFH	BA	MA
12	Banupriya	28	15319	G2P1L1	AN	AN	21.50	H/o GHT 1st Pregancy	Lower limb Swelling	DVT	6.9	Left Ileofemoral DVT	UFH	AN	MA
13	Kohita	26	16436	G2P1L1	AN	AN	25.30	GDM GHT	LL Varicose veins	DVT	7.8	DVT\Ileofemoral Incompetence	UFH	AN	MA
14	Abirami	23	15535	P2L2	LN	PND6	23.10	K/C/O CVT	B/L Pupil Edemia +	CVT	10.6	SSS and TS	LMWH	BA	MA
15	Sangeetha	28	49591	Primi	AN	AN	33.30	Migaraine upper limb Right side Headace	Headache	CVT	10.5	Sigmoid Sinus	LMWH	AN	MA
16	Rani	35	49155	G2P1L1	AN	AN	37.10	Mild lower limb swelling	Right Lower Limb Swelling with Edemia warmth	DVT	10.5	Right femopopliteal DVT	UFH	AN	MA
17	Ranjitha	26	19127	G2P1L1	AN	AN	25.60	Thyroid disorder Auto immune thyroiditis imminant eclampsia	Headache blurring of vision	CVT	8.5	Cerebral vein thrombosis	LMWH	AN	MA
18	Vanitha	36	44993	G4P2L2A1	AN	AN	18.70	Actue	Vomit, Headache, irrevalent speech	CVT	10	Transverse Sinus involved	UFH	AN	MA
19	Selvarani	26	10471	P2L2	Post LSCS	POD 39	24.70	GDM GHT	Left Lower Limb swelling, warmth Distal pulses absent	DVT	10.8	Left femopopliteal DVT	LMWH	BA	MA
20	Shanthi	37	47482	P3L3A2	PA	PA Day 17	21.10	GDM GHT	Headache with ear pain	CVT	10.3	SSS	UFH	PA	MA

21	Poongudi	29	27443	P2L2	Post LSCS	POD 36	25.80	GDM GHT	Acute confustion	CVT	5.5	SSS	UFH	BA	MA
22	Abirami	29	47442	Primi	AN	AN	21.30	RHD Heart disease	Diffculty in Breathing	Brachial Artery Thrombosis	9.8	Brachial Artery Thrombosis	UFH	AN	MA
23	Sangeetha	35	24357	P4L4A1	PA	PA Day 14	27.20	Severe anemia HB 7.0	Headache with convulsions	CVT	7	Straight Sinus	UFH	PA	MA
24	Karthiga	26	24688	P2L2	Post LSCS	POD 12	37.50	GHT	Headache with convulsions	CVT	9.8	Transverse Sinus involved	UFH	BA	MA
25	Viji	27	17486	P2L2A1	Post LSCS	POD 14	28.00	GDM GHT	Acute psychosis	CVT	9.8	SSS	UFH	BA	MA
26	Savitha	20	18216	P1L1	Post LSCS	POD 22	33.50	GHT/Immobilisation	Left Lower Limb swelling	DVT	7.5	Left Iliac Vein Thrombosis	LMWH	BA	MA
27	Kumareswari	25	24686	A1	PA	PA Day 1	19.70	RHD MS/MR Post MVR Status Acute pulmonary edema	Diffcult in breathing	Mitral Valve Thrombosis	5.9	Valvular Thrombosis	UFH	PA	MA
28	Seethala Devi	25	34682	P1L1	Post LSCS	POD 5	27.80	Imminate eclampsia GHT	Headache	CVT	10.4	Sigmoid Sinus	UFH	BA	MA
29	Vidhya	26	35202	P2L2	Post LSCS	POD 32	30.00	GDM GHT	Headache with blurring of vision	CVT	11.5	Sigmoid Sinus	LMWH	BA	MA
30	Panimalar	35	19517	P2L1	LN Outside	PND21	31.20	GHT Anemia 3 packed cells	Headache with convulsions	CVT	7	SSS	LMWH	BA	MA
31	Sasikala	25	6459	P3L3	LN Outside	PND 8	21.30	Severe anemia 3 packed cell	Headache	CVT	9	Transverse Sinus involved	LMWH	BA	MA
32	Chithra	38	6298	P4L4	LN	PND 12	35.90	GHT/Dehydration	Swelling over right lower limb	DVT	9	Right femopopliteal DVT	LMWH	BA	MA
33	Maheswari	20	2932	P3L3	LN Outside	PND 7	20.00	GHT	Headache	CVT	10.5	SSS	UFH	BA	MA
34	Rosma Begam	20	7239	P2L1	LN	PND 16	40.80	GHT severe pre eclampsia	Headache anemia 2 packed cell	CVT	10.5	SSS	UFH	BA	MA
35	Keerthiga	24	8151	P1 L1	LN	PND 1	23.70	GHT/Dehydration	left lower limb swelling	DVT	10.8	Right femopopliteal DVT	LMWH	BA	MA
36	Ramya	25	4560	P2L2	LN	PND 16	36.30	GHT	Headache with convulsions	CVT	9.8	SSS	LMWH	BA	MA
37	Vinodha	24	4636	P1L1	Vaccum delivery with EPI - LN	PND 6	19.10	Chronic Hypertension	Headache numbness of left limb	CVT	9.5	Transverse Sinus involved	LMWH	BA	MA
38	Sagayajothi	42	60931	Primi	AN	AN	19.30	APLA	Bilateral Lower limb swelling	DVT	11	Bilateral popliteal vein thrombosis	LMWH	AN	MA
39	Kanagadevi	27	20267	P4L4A1	LN All NVD	PND 3	24.30	GHT	Acute psychosis	CVT	9.8	SSS	LMWH	BA	MA
40	Kalaiselvi	19	20071	Teenage Primi	AN	AN	19.10	GHT/Immobilisation	Left lower limb swelling	DVT	10	Bilateral popliteal vein thrombosis	LMWH	AN	MA
41	Sangeetha	20	18216	P1L1	Post LSCS	POD 29	26.10	GHT	Swelling of left leg	CVT	8	SSS	LMWH	BA	MA
42	Vinodha	23	29723	P1L1	LN with PPIUCD	PND 1	24.00	GHT imminate eclampsia	Headache blurring of vision	CVT	9.9	SSS	LMWH	BA	MA
43	Radha	36	30541	P3L2A1	Post LSCS	PND 5	24.70	Post ASD Closure	Pain and swelling of left upper limb	Cephalic vein Thrombosis	10	Cephalic vein Thrombosis	LMWH	BD	MA

44	Maheswari	30	31450	P1L1	LN	PND 8	27.90	GDM GHT	Headache	CVT	8.8	Straight Sinus	LMWH	BA	MA
45	Chithra	40	31988	P7L6	POST LSCS	POD 6	18.30	GDM GHT/Dehydration	Bilateral Lower limb swelling, Bilateral PP Edemia	DVT	6	Bilateral popliteal vein thrombosis	UFH	BA	MA
46	Vanitha	28	31334	P3L3	POST LSCS	POD 6	27.90	GHT	Headache	CVT	13	Transverse Sinus involved	UFH	BA	MA
47	Thangaponnu	27	34831	P3L3	LN Outside	PND 6	20.20	GHT	Headache with convulsions	CVT	9.9	SSS	UFH	BA	MA
48	Aarthi	22	29035	P3L3	LN Outside	PND 10	26.00	GDM GHT	Lower limb Swelling	DVT	7.4	Bilateral popliteal vein thrombosis	UFH	BA	MA
49	Subulakshmi	23	31596	P2L2	LN Outside	PND 8	27.70	GDM GHT	Lower limb Swelling	DVT	9	Ileofemoral DVT	UFH	BA	MA
50	Surekha	27	31805	P4L4	LNwith PPIUCD	PND 15	27.90	GHT imminate eclampsia	Headache	CVT	10.4	SSS and TS	UFH	BA	MA
51	Bhuvaneswari	28	31631	P1L1	POST LSCS	POD 6	26.20	GDM GHT	Blurring of vision	CVT	9	SSS	UFH	BA	MA
52	Suryakala	23	33140	P1L1	POST LSCS	POD 6	29.40	GHT	Headache with convulsions	CVT	10	Cerebral vein thrombosis	UFH	BA	MA
53	Jamuna	29	33153	P3L4	LN	PND 22	24.80	GHT	Headache severe Pre eclampsia	CVT	8	Transverse Sinus involved	UFH	BA	MA
54	Hemavathy	24	33504	P1L1	POST LSCS	POD 6	22.70	GHT	Headache	CVT	9.6	SSS	UFH	BA	MA
55	Sivabahigam	28	33913	P3L3	POST LSCS	POD 6	24.20	GHT	Right upper limb swelling and edemia	Cephalic vein Thrombosis	9	Uppe limb thrombosis with Cephalic vein thrombosis	LMWH	BA	MA
56	Suganya	28	35937	P1L1A1	PA	PAD 2	21.40	APLA	Headache	CVT	8	SSS	LMWH	PA	MA
57	Sowmiya	28	35949	P1L1	POST LSCS	POD 6	28.10	GHT	Headache blurring of vision	CVT	9.6	SSS	LMWH	BA	MA
58	Mariyammal	27	36076	P1L0	Vaccum delivery - LN	PND 3	28.70	Covid Positive	Blurring of vision	CVT	14	Cerebral vein thrombosis	LMWH	BD	MA
59	Rahda	28	36037	P2L2	POST LSCS	POD 6	35.70	GHT anemia imminate eclampsia	Headache	CVT	9	Transverse Sinus involved	UFH	BA	MA
60	Suganya	26	33986	P3L2	POST LSCS	POD 8	21.90	GHT	Headache	CVT	9.6	SSS	LMWH	BA	MA
61	Vijayalakshmi	24	34628	P3L3	POST LSCS	POD 8	27.90	GHT	Headache with convulsions	CVT	8.8	SSS	LMWH	BA	MA
62	Mahalakshmi	23	34723	P2L2A1	POST LSCS	POD 8	22.40	GHT	Headache	CVT	8	SSS	LMWH	BA	MA
63	Maheswari	26	40272	P3L2	POST LSCS	POD 8	24.80	GHT	Headache with convulsions	CVT	8.8	SSS	LMWH	BA	MA
64	Jayalakshmi	28	40977	P1L1	POST LSCS	POD 8	28.80	GHT Severe Anemia	Blurring of vision	CVT	6	Cerebral vein thrombosis	UFH	BA	MA
65	Mallika	30	41612	P4L4	POST LSCS	POD 8	27.60	Subclnicial hypothyroid	Headache with convulsions	CVT	9.8	SSS	LMWH	BA	MA
66	Ranjani	28	2413	P2L2A1	POST LSCS	POD 6	22.20	GHT/Immobilisation	Right lower Limb Swelling	DVT	9.7	Bilateral popliteal vein thrombosis	LMWH	BA	MA

67	Subarna	26	40899	P2L2	Post LSCS	POD 1	32.00	GDM GHT	Headache	CVT	10	Sigmoid Sinus	LMWH	BA	MA
68	Punitha	25	1545	P2L2	POST LSCS	POD 6	22.50	GHT	Headache with convulsions	CVT	9.8	Sigmoid Sinus	LMWH	BA	MA
69	Ronikamary	35	40679	P3L3	POST LSCS	POD 6	31.60	GHT	Upper limb swelling	Cephalic vein Thrombosis	9.9	Uppe limb thrombosis with Cephalic vein thrombosis	LMWH	BA	MA
70	Menaka	19	1510	P1L1	PA	PA 2	30.60		Headache with convulsions	CVT	11	Sigmoid Sinus	UFH	PA	MA
71	Subulakshmi	25	2459	P2L2	Post LSCS	POD 6	26.10	GHT	Left Lower Limb Swelling	Cephalic vein Thrombosis	6.2	Uppe limb thrombosis with Cephalic vein thrombosis	LMWH	BA	MA
72	Vembarasi	30	29671	P3L3	POST LSCS	POD 6	35.50	Subclnicial hypothyroid	Headache	CVT	9.3	SSS	UFH	BA	MA
73	Vennila	24	24757	P1L1	POST LSCS	POD 6	29.70	Overt hypothyroid	Headache with convulsions	CVT	13.6	SSS	UFH	BA	MA
74	Parisutham	34	24351	P2L2	POST LSCS	POD 6	24.40	GHT/Immobilisation	Left Lower Limb Swelling	DVT	10.8	Ileofemoral DVT	UFH	BA	MA
75	Poongulazhi	28	20805	P2L2	POST LSCS	POD 8	24.80	GHT/Immobilisation	Left Lower Limb Swelling	DVT	9.8	Ileofemoral DVT	UFH	BA	MA
76	Vennila	28	37385	P1L1	Post LSCS	POD 1	20.10	GHT GDM on MNT	Headache with convulsions	CVT	10.2	SSS	UFH	BA	MA
77	Rama	28	38851	P2L2	Post LSCS	POD 1	29.40	GHT	Headache Pupil Edemia	CVT	12	Cerebral vein thrombosis	UFH	BA	MA
78	Poongothai	29	34647	P2L2	Post LSCS	POD 36	28.70	GHT	Depression Acute confusion, Post partum psychosas	CVT	10.2	SSS	UFH	BA	MA
79	Abirami	29	34642	Primi	AN	AN	28.10	RHD Heart disease	Headache	CVT	12.6	SSS	UFH	AN	MA
80	Kannahi	21	18066	P1L1	POST LSCS	POD 8	24.70	Hypothroid	Headache	CVT	8.4	SSS	UFH	BA	MA
81	Sindhu	29	26030	P3L3	POST LSCS	POD 8	28.90	GHT/Immobilisation	Lower limb Swelling	DVT	9.8	Bilateral popliteal vein thrombosis	UFH	BA	MA
82	Syed Ratiza	26	25883	P1L2A1	POST LSCS	POD 8	31.20	GHT/Immobilisation	Lower limb Swelling	DVT	9	Bilateral popliteal vein thrombosis	UFH	BA	MA
83	Jayalakshmi	26	32164	P1L1A1	LN	PND 14	20.40	GHT imminate eclampsia	Headache	CVT	8.6	Cerebral vein thrombosis	UFH	BA	MA
84	Kavitha	31	32112	P2L2	POST LSCS	POD 8	25.10	GHT	Headache with convulsions	CVT	7	SSS	UFH	BA	MA
85	Chithra	36	28783	A1	PA	PAD 12	25.80	GHT	Headache with convulsions	CVT	12	SSS	UFH	PA	MA
86	Sabana Begam	19	27132	P1L1	POST LSCS	POD 8	28.00	GHT	GTCS with LOC	CVT	6.5	SSS	LMWH	BA	MA
87	Rasiya	35	26722	P3L3	POST LSCS	POD 8	24.80	GHT/Immobilisation	Lower limb Swelling	DVT	13.4	Ileofemoral DVT	LMWH	BA	MA
88	Vasanthi	45	26098	P1L0	LN	PND 5	28.40	Subclnicial hypothyroid	Headache Pupil Edemia	CVT	8.9	SSS	LMWH	BD	MA

89	Chinthamani	25	28047	P1L0	LN	PND 2	30.00	GHT	Acute psychosis	CVT	9	SSS	LMWH	BD	MA
90	Kodimalar	27	26004	A1	PA	PAD 9	24.70	GHT	Headache with convulsions	CVT	9.8	SSS	LMWH	PA	MA
91	Rani	28	20848	P2L2	Post LSCS	POD 10	27.60	GHT	Headache	CVT	10.4	SSS	LMWH	BA	MA
92	Janani	24	76464	P2L2	Post LSCS	POD 16	27.00	GHT/Dehydration	Lower limb Swelling	DVT	12	Ileofemoral DVT	LMWH	BA	MA
93	Kanimozhi	29	39030	P2L2A1	Post LSCS	POD 10	28.00	GHT	GTCS with LOC	CVT	9.8	SSS	LMWH	BA	MA
94	Sudha	27	40981	P1L1	LN	PND 1	25.60	GHT	Headache	CVT	9.6	SSS	LMWH	BA	MA
95	Vaitheeswari	25	41014	P2L2	LN	PND 2	30.50	GHT/Dehydration	Lower limb Swelling	DVT	10.6	Ileofemoral DVT	LMWH	BA	MA
96	Maheswari	26	40272	P3L2	LN	PND 10	21.60	Anemia	Headache with convulsions	CVT	8.4	SSS	LMWH	BA	MA
97	Habib Nisha	30	38819	P3L2	LN	PND 4	31.20	GHT	Headache	CVT	10.8	SSS	LMWH	BD	MA
98	Tamilarasi	22	37844	P1L1	LN	PND 4	20.00	GHT	Headache	CVT	10.2	SSS	LMWH	BA	MA
99	Rathika	28	24132	P2L2	LN	PND 10	26.70	Severe Anemia	Headache with convulsions	CVT	7	SSS	LMWH	BA	MA
100	Kalaiyarasi	28	40899	P1L0	Post LSCS	POD 1	26.80	Covid Positive	Acute Abdomen	Mesentric vein Thrombosis	9.9	Extensive samll bowel gangrene	UFH	BD	Dead