"STUDY ON CLINICAL PROFILE OF CEREBRAL VENOUS THROMBOSIS"

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CERTIFICATE

This is to certify that this dissertation entitled "**STUDY ON CLINICAL PROFILE OF CEREBRAL VENOUS THROMBOSIS"** is the bonafide work of **Dr. DEVAN.R** in partial fulfilment of the requirements for M.D Branch -I (General Medicine) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL - 2013 under my guidance and supervision during the academic year November- 2010 to November - 2012.

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DECLARATION

I, **Dr. DEVAN.R**, solemnly declare that the dissertation titled **"STUDY ON CLINICAL PROFILE OF CEREBRAL VENOUS THROMBOSIS"** is a bonafide work done by me at Thanjavur Medical College, Thanjavur during November 2010 to November 2012 under the guidance and supervision of **Prof. Dr. P.G.SANKARANARAYANAN, M.D.,** Unit Chief M-II, Thanjavur Medical College, Thanjavur.

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CONTENTS

LIST OF ABBREVIATIONS

AHA- American Heart Association

ASA- American Stroke Association

APLA- Antiphospholipid Antibodies

CVT- Cerebral Venous Thrombosis

CSF- Cerebro Spinal Fluid

CNS- Central Nervous System

CT- Computed Tomography

DVT- Deep Vein Thrombosis

DIC- Disseminated Intravascular Coagulation

EEG- Electroencephalography

EFNS- European Federation of Neurological Science

GCS- Glascow Coma Scale

ISCVT- International Study on Cerebral Vein and dural sinus Thrombosis

INR- International Normalised Ratio

LSCS- Lower Segment Caesarean Section

mRS- modified RANKIN SCALE

MRI- Magnetic Resonance Imaging

MRA- Magnetic Resonance Angiography

MRV- Magnetic Resonance Venography

OCP- Oral Contraceptive Pill

SSS- Superior Sagittal Sinus

SLE- Systemic Lupus Erythematosus

VDRL-Venereal Disease Research Laboratory

"STUDY ON CLINICAL PROFILE OF CEREBRAL VENOUS THROMBOSIS"

BACKGROUND:

Cerebral venous thrombosis (CVT) refers to the thrombosis of dural venous sinuses or the cerebral veins. Once considered a rare disease, CVT is now being recognized with increasing frequency especially in South Asian countries including India. It is an important cause of young strokes in India especially among woman. Though the prognosis is good in majority of patients, it is a potentially life threatening disease requiring early clinical suspicion and prompt diagnosis. However the diagnosis may be challenging in certain cases due to varied clinical presentation and there is a substantial difference in predisposing factors, presentations, therapeutic options, and outcome between developed and developing countries. The objective of our study was to study the demographic, clinical, etiological, radiological and prognostic characteristics of the disease in patients admitted with CVT in our hospital.

METHODS:

This was a cross sectional study conducted on 45 patients admitted with CVT in Thanjavur medical college hospital, Thanjavur satisfying the inclusion and exclusion criteria. Detailed history, neurological examination, brain imaging with CT and MRI with MRV, routine and specific laboratory investigations as required were done in all patients and we analysed the demographic factors, clinical presentation, etiology, radiological features and outcome in these patients. Factors associated with good and poor outcome were also analysed.

RESULTS:

In our study, CVT was 3 times more common in females than in males especially those between the age group of 21 to 30 years. Majority of patients presented subacutely. Headache was the most common symptom and papilledema was the most common sign. 28% of patients presented with headache as an isolated symptom. Other common presentations were altered sensorium, focal motor

deficit, generalized seizures and delirium. Puerperium was the leading cause of CVT in our study. Superior sagittal sinus and right transverse sinus were the most common sinuses involved by MRI with MRV. Most common finding in CT Brain was haemorrhagic infarct though CT Brain was normal in about 26 to 27 % of patients. In our study, the mortality rate was 11% and the morbidity rate was 17%. Presentation with Isolated intracranial hypertension syndrome was associated with good outcome. Age ≥35 years, GCS score of <9 and coma at presentation were associated with poor prognosis.

CONCLUSION:

1. CVT has a wide range of clinical presentation.

2. CVT should be suspected,

i) Whenever a young adult presents with symptoms and signs of raised intracranial tension with or without other neurological symptoms. Examination of the fundus to rule out papilledema is an important tool in arriving at a diagnosis in such cases of CVT.

ii) Whenever a young adult presents with stroke especially in the absence of vascular risk factors.

iii) Whenever a peripartum female presents with neurological symptoms in our setting.

iV) Whenever imaging of the brain shows haemorrhagic infarct especially in non arterial territories.

3. Diagnosis should be confirmed by MRI with MRV whenever possible.

4. In general, the prognosis is good in CVT; however extravigilance may be required in patients who present with poor prognostic factors.

KEY WORDS:

Cerebral venous thrombosis
Young peripartum females
Wide clinical presentation
Most common- Isolated intracranial hypertension
Haemorrhagic infarct in CT brain
Confirm diagnosis by MRI with MRV
Prognosis good in general

INTRODUCTION

Cerebral venous thrombosis (CVT) refers to the thrombosis of dural venous sinuses or the cerebral veins. It is a disease of young to middle-aged people and is more common in females[1,2,3]. It is a potentially life threatening condition requiring early clinical suspicion and prompt treatment.

Though most of the patients have an excellent outcome if treated early and appropriately, diagnosis may get delayed by the wide clinical spectrum of symptoms, various forms of initial presentation, obscuring of symptoms and signs by the underlying disease like meningitis and normal findings in neuroimaging.

CVT has an extremely diverse clinical features, predisposing factors, brain imaging findings and outcome[4.] There may be a substantial difference in predisposing factors, presentations, therapeutic options and outcome between developed and developing countries. For example, International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) [3]reported obstetric CVT in only 20% of cases as compared to reports from Mexico and India, which report a much higher frequency [5,6]. In addition there is variability among different developing countries[5].

The incidence of CVT is uncertain since it has a wide range of clinical manifestations [7]. Once considered a rare disease based on autopsy studies, CVT is now recognized with increasing frequency due to enhanced clinical awareness and improved non invasive imaging modalities available now. Recently Panagariya et al [8] reported that 17% of all strokes and half of all strokes in young people are due to CVT.

CVT has a specific geographic distribution, the incidence being higher in South Asia and the Middle east[9,10]. Most studies from India have reported a large number of cases; hence the incidence in India is not as rare as assumed earlier[10]. In India, CVT accounts for 10-20% of young strokes[10]. However no well designed large scale epidemiologic study on CVT has been conducted in South Asia, where it is comparatively frequent.

These factors prompted us to study the clinical profile and to evaluate the demographic, clinical, etiological, radiological and prognostic characteristics of the disease among patients admitted with CVT in Thanjavur Medical College hospital, a major tertiary care hospital for the people in and around Thanjavur district.

AIM AND OBJECTIVE

AIM

To observe the demographic factors, clinical manifestations, risk factors and also the neuroimaging, laboratory, outcome data in 45 patients of cerebral venous thrombosis.

OBJECTIVE

To evaluate the demographic, clinical, etiological, radiological and prognostic characteristics of the disease in patients admitted in our hospital.

REVIEW OF LITERATURE

INTRODUCTION

Cerebral venous thrombosis (CVT) is defined as thrombosis in the dural venous sinuses or the cerebral veins[24]. It is considered an uncommon cause of stroke and its incidence is much less common than cerebral arterial thromboembolism [24]. But in India, CVT accounts for 10-20% of young strokes [10].

The clinical features are diverse hence CVT is more challenging to diagnose than other types of stroke. Most patients have an excellent outcome if treated early and appropriately. But it is a potentially life threatening condition requiring early clinical suspicion and prompt treatment.

EPIDEMIOLOGY:

Most of the initial studies on CVT were autopsy studies performed in western countries. The largest autopsy study reported incidence of CVT as 9% [25].The estimated incidence of CVT, based on autopsy studies would be about one to two cases per million. However these autopsy studies are biased because they reflect severe fatal cases of CVT alone. They created an impression that CVT is a disease of rarity. However subsequent larger clinical studies disproved this fact.

Most of these studies were from European countries. In a hospital based series in Portugal incidence of CVT was 0.22/100000/year [26].In Hongkong the rate among admitted patients was 3.4/100000/year [27]. In Isfahan, Iran the annual frequency of CVT was 1.23/100000/year [28]. Daif et al from Saudi Arabia reported a frequency of 7 per 100,000 hospital patients, in 1995 [29].No large multi-center or multi-national data base or registry reported data from Asian countries. Studies from India on CVT have proved that CVT is more common in the developing countries of Asia than in the western world [10].

CVT is not uncommon in Asia especially in the South Asian subcontinent including India, Pakistan and Bangladesh [30]. Recently, Panagariya et al from India reported that 17% of all strokes in India are due to CVT. They also noted that half of all young strokes in India are due to CVT and that 38% of all women who experienced stroke had venous stroke [8]. Hence CVT is no longer considered a rare disease.

Review of CVT cases from Asian countries suggest differences in risk factors and outcome in these patients as compared to European studies [30] .The largest clinical series, the International Study on Cerebral vein and dural sinus thrombosis

(ISCVT) reported that 50% of these cases were related to oral contraceptive pills (OCP), 6% were due to pregnancy and 14% were secondary to puerperium [3].A study of 182 adult patients with CVT from USA reported 7% were due to pregnancy and puerperium and 5% related to OCP use[31]. A study from Pakistan reported that 17% were due to pregnancy and puerperium and 5% related to OCP use[5]. Cantu from Mexico reported 59% cases due to pregnancy and puerperium[32].

Although rare, CVT is a well recognized disorder in children, with approximately half of the cases occurring in neonates and young infants[33]. In adults CVT affects patients who are younger than those with other types of strokes and the incidence apparently decreases in older subjects. The median age in the ISCVT cohort was 37 years[10] with only 8 % of the patients older than 65 years. According to an Indian study, the mean age of patients with CVT was 32.27 years [6].

CVT is more common in females than males. Male to Female ratio in ISCVT was 1:2.9 [3]. Ameri and Bousser reported a female-to-male ratio of 1.29:1 in their study[4] similar to the observations of Ferro et al [34]. Most recent cases reported in adult women are in association with puerperium [33].

VENOUS ANATOMY

The anatomy of the cerebral veins and sinuses is subject to considerable individual variation in size and patency [35].

The Cerebral venous system includes the cerebral veins and dural venous sinuses.

Dural venous sinuses:

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

Superior group collects major part of the blood of the brain. It includes superior sagittal sinus (SSS), inferior sagittal sinus, transverse sinus, straight sinus and sigmoid sinuse. The inferior group which drains the basal and medial parts of undersurface of brain, the orbits and the sphenoparietal sinus collects at the cavernous sinus. Cavernous sinuses connect with the lateral sinuses via superior and inferior petrosal sinuses and with the pterygoid plexus.

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

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

Cerebral veins:

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

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

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

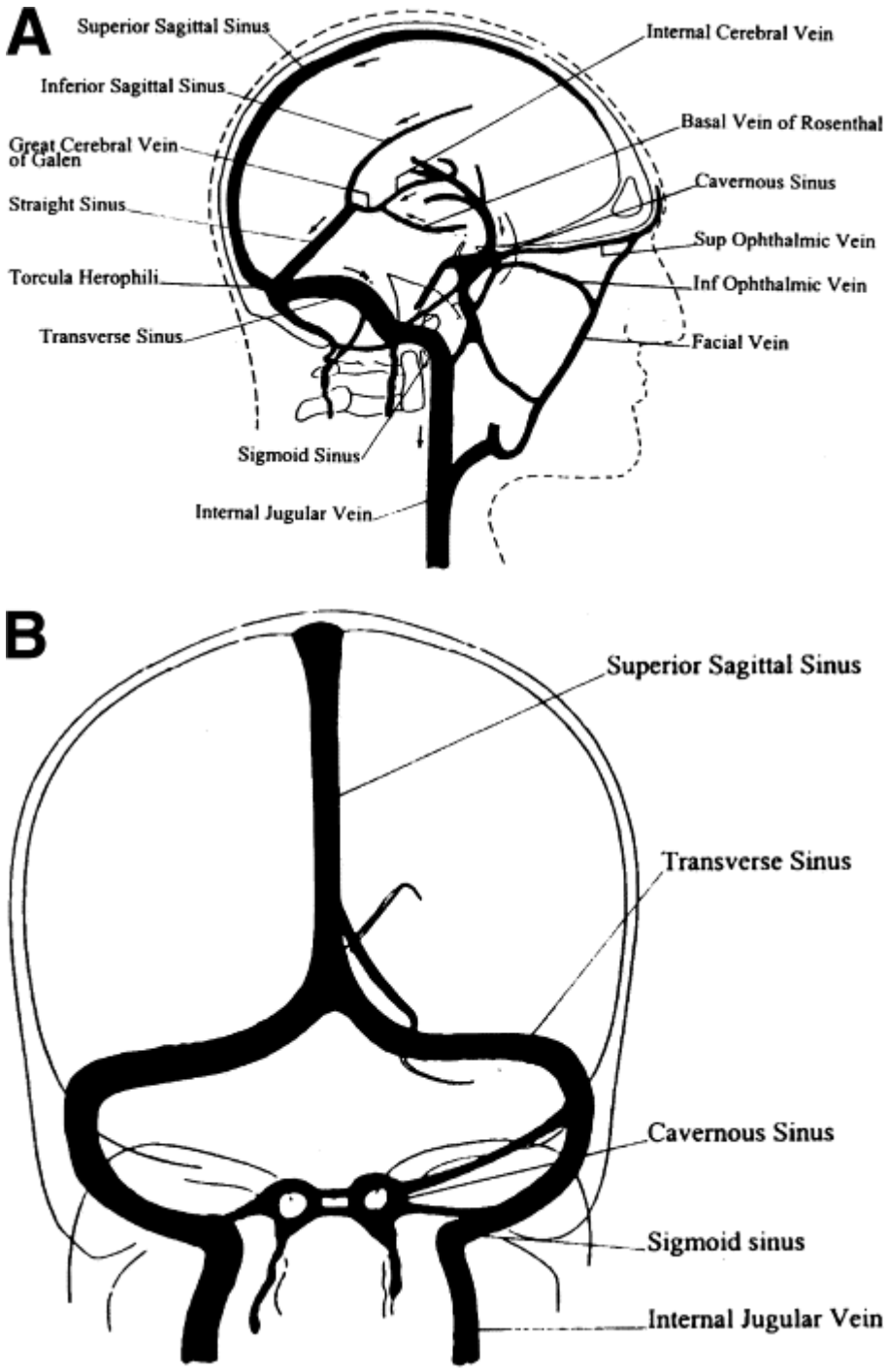
The deep cerebral veins drain the inferior frontal lobe, most of the deep white matter of cerebral hemisphere, the corpus callosum, basal ganglia and upper

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

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

Superior sagittal sinus and the lateral sinuses are the most commonly affected sinuses in CVT, followed by the straight sinus and the cavernous sinus [36,37].

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


A
Superior Sagittal Sinus
Inferior Sagittal Sinus
Great Cerebral Vein of Galen
Straight Sinus
Torcula Herophili
Transverse Sinus
Sigmoid Sinus
Internal Jugular Vein
Internal Cerebral Vein
Basal Vein of Rosenthal
Cavernous Sinus
Sup Ophthalmic Vein
Inf Ophthalmic Vein
Facial Vein
B
Superior Sagittal Sinus
Transverse Sinus
Cavernous Sinus
Sigmoid sinus
Internal Jugular Vein

PATHOPHYSIOLOGY:

Two major mechanisms [37] occurring in the pathophysiology of cerebral venous thrombosis are,

1. Thrombosis of cerebral veins or dural sinus, leading to cerebral lesions.
2. Thrombosis of dural sinus, resulting in disturbance of CSF absorption and increased intracranial pressure.

First, thrombosis of cerebral veins or sinuses results in increased venular and capillary pressure. As local venous pressure continue to raise, decreased cerebral perfusion results in ischemic injury and cytoxic edema, disruption of blood brain barrier leads to vasogenic edema and venous and capillary rupture culminates in parenchymal hemorrhage.

Obstruction of cerebral sinuses may also result in decreased cerebrospinal fluid absorption which normally occurs through arachnoid granulation into the superior sagittal sinus. Thus thrombosis of cerebral sinuses not only increases venous pressure also impairs CSF absorption and ultimately leads to increased intracranial pressure. Increased intracranial pressure aggravates venular and capillary hypertension and leads to parenchymal haemorrhage, vasogenic and cytotoxic edema.

Experimental animal data suggests that vasogenic edema occurs earlier in venous stroke than in arterial stroke and cytotoxic edema is far less common in venous stroke [38].

After venous occlusion large areas of brain may be functionally and metabolically disturbed, but not irreversibly. Reversibility is very typical of venous lesions, reflected by a favourable clinical recovery and vanishing lesions on neuroimaging[39].

Occlusion of one of the larger venous sinuses is not likely to cause localized tissue damage unless there is involvement of cortical veins or the Galenic venous system since alternate drainage routes will suffice .Thrombosis in cerebral veins, with or without dural sinus thrombosis causes multiple venous infarcts [24].

TYPES OF CEREBRAL VENOUS THROMBOSIS:

Intracranial venous thrombosis can be classified based on etiology [33] as

1. Septic and

2. Aseptic

Septic CVT most often involves the cavernous sinus and is relatively infrequent nowadays [33].

Aseptic CVT is divided into

1. Dural venous thrombosis

2. Deep venous thrombosis and

3. Cortical or Superficial vein thrombosis

ETIOLOGY:

A large number of conditions are known to cause or predispose to CVT.

They include[40]

1. Prothrombotic conditions:

Genetic

Protein S/C deficiency

Antithrombin III deficiency

Factor V Leiden mutation

G20210A prothrombin gene mutation

Plasminogen deficiency

Increased coagulation factor VIII

Acquired

Antiphospholipid antibody syndrome

Hyperhomocystinemia

2. Infections:

Central nervous system (e.g.,abscess,meningitis)

Ear, sinus, mouth, face and neck (e.g.otitis,mastoiditis,tonsillitis)

Systemic infections (e.g.sepsis,endocarditis,tuberculosis,HIV)

3. Inflammatory diseases:

Systemic lupus erythematosus

Behcet's disease

Sjogrens's syndrome

Wegner's granulomatosis

Temporal arteritis

Thromboangitis obliterans (Bueger's disease)

Inflammatory bowel disease

Sarcoidosis

4. Malignancy:

Central nervous system (meningoma,metastasis,medulloblastoma)

Solid tumor outside the central nervous system

Haematologic (leukemias,lymphomas)

5. Hematological conditions:

Sickle cell disease or trait

Iron deficiency

Paroxysmal nocturnal hemoglobinuria

Thrombocythemia (primary or secondary)

Polycythemia (primary or secondary)

Disseminated intravascular coagulation[33]

6. Pregnancy and puerperium

7. Other disorders:

Dehydration

Congenital heart disease

Nephrotic syndrome

Diabetic ketoacidosis

Thyroid disease (hyper or hypothyroidism)

Dural fistula, arachnoid cyst

8. Other precipitants:

Head trauma

Myelography, intrathecal steroids, lumbar puncture

Neurosurgical procedures

Irradiation

Jugular catheter occlusion

Drugs (oral contraceptives, hormone replacement therapy, androgens, steroids, sildenafil, l-asparaginase, cyclosporine, tamoxifen etc.)

Atleast one risk factor can be identified in more than 85% of patients with CVT and multiple risk factors in about half of the patients [3].

The more frequent risk factors are prothrombotic conditions, oral contraceptive use, pregnancy / puerperium, infection and malignancy [3].

A prothrombotic condition was identified in 34% of patients in the ISCVT cohort, being genetically determined in 22% patients [3]. Most common among them are Prothrombin gene mutation, Factor V leiden mutation and Anticardiolipin /antiphospholipid antibody syndrome [3,41]. Less common are protein C/ protein S deficiency and antithrombin III deficiency [41].

Infective causes of CVT are rare now and they account for only 6 to 12 % of cases[3,42]. In developing countries, systemic and nervous system infections may remain an important cause of CVT (18%) [5].

About 7.4% of cases of CVT are due to cancers of which 2.2% are due to CNS malignancy, 3.2% are due to solid tumors outside CNS and 2.9% are due to hematological malignancies [3].

In neonates, acute systemic illness such as perinatal complications and dehydration were frequent accounting for 84% of cases [43].

In older children, head and neck disorders, mostly infections and chronic systemic disorders account for most of the cases.

The most common risk factor in young women is oral contraceptive use. In the metaanalysis done by Dentali and colleagues [44], risk of DVT was reported to be 6 times higher in women taking oral contraceptives than those not taking OCP. Another frequent risk factor is pregnancy and puerperium[3,32,45], more commonly in less developed world regions with higher pregnancy rates [46].

In the ISCVT cohort, thrombophilia, malignancy and hematologic disorders such as polycythemia were the most common risk factors for CVT in the elderly [47]. However in 37% of elderly,no risk factor could be identified.

Despite extensive search, no underlying risk factor is found in 20% of adult patients with CVT [24].

CLINICAL FEATURES:

The clinical manifestations of Cerebral venous thrombosis are highly variable [42]. In more than 50% of the cases, it is subacute in onset, in onethird of patients it is of acute onset and in a few cases it is chronic [40].

Symptoms and signs of CVT are classified into 3 more frequent syndromes as follows,

1. Isolated intracranial hypertension syndrome which consists of headache with or without vomiting, papilledema and visual troubles [48].

2. Focal syndrome which includes focal deficit, seizures or both.

3. Encephalopathy which consists of bilateral or multifocal signs, delirium or consciousness disturbances [26,42].

Less frequent syndromes include cavernous sinus syndrome and syndromes involving multiple palsies of lower cranial nerves. Multiple transient ischemic attacks have also been reported as a manifestation of CVT [49].

In perhaps one-quarter of 'benign intracranial hypertension' patients the cause is dural sinus thrombosis[24]. In these cases, there is seldom propagation of thrombosis into the cerebral veins with venous infarction and focal neurological features. Indeed, the clinical picture seems identical to that of idiopathic benign intracranial hypertension. The prognosis is very good, although a few patients may be left blind due to optic atrophy.

Clinical signs and symptoms depend on the following factors

1. Gender of the patient [43,47].

2. Age of the patient [43,47].

3. Interval from onset to presentation [50,51].

4. Presence of parenchymal lesions.

5. Type and number of involved sinuses and veins.

In neonates, presentation is often nonspecific. Most common among them are seizures, respiratory distress, poor feeding, lethargy, hypertonia or hypotonia [52].

Older children manifest like adults. They most commonly present with head ache with or without vomiting, papilledema, sixth cranial nerve palsy, motor deficit, seizures and altered consciousness[43,53].

In elderly patients headache is less common [47] , decreased vigilance and mental symptoms are more common than younger patients[40,47].

Upto 90% of patients with CVT complain of headache which is the most frequent symtoms and often the initial one[40]. By ISCVT, head ache was the only symptom in 9% of patients with CVT[40]. Headache associated with CVT has no specific characteristics, it may be acute or chronic, localized or diffuse [54].The localization of headache has no relationship with the location of the occluded sinuses or the parenchymal lesions [55]. CVT associated headache can be more severe and acute than other types of headache in some cases requiring emergency care. The most

frequent type of headache is the intracranial hypertension variety, a severe, generalized headache worsening with valsalva's maneuvers and when the patient is lying down. Migraine with aura has also been reported[56] .Sudden headache, often with blood in the CSF, is a very rare presentation and clearly can be confused with spontaneous subarachnoid haemorrhage due to aneurysmal rupture[57]. CVT must also be included as a possible cause of persisting headache after lumbar puncture.

Papilloedema occurs in about 50% of patients [24] and is more frequent in chronic cases. Seizures, focal deficit, alterations in conscious level occur in about 30% of cases [24]. Motor weakness including hemiparesis is the most common focal finding and may be present in 40% of patients [3,58]. Seizures are more frequent in CVT than in other stroke types [40]. About 30 to 40% of patients with CVT can present with seizures either focal or generalized or with status epilepticus, especially with thrombosis of sagittal sinuses and cortical veins [58,59]. Isolated thrombosis of superficial cortical veins have marked tendency to produce partial seizures. About 13% of patients complain of visual loss [40]. Isolated cranial nerve palsy have been described with transverse sinus thrombosis[60]. Aphasia is seen in 19% of patients, for example, fluent aphasia occurs with left lateral sinus occlusion [40].

The progression of symtoms and signs in CVT is highly variable ranging from less than 48 hours to more than 30 days [25].

Dural sinus thrombosis:

Cavernous sinus thrombosis is a restricted form of CVT usually associated with sepsis spreading from the veins in the face, nose, orbits or sinuses. In general the presentation is with unilateral orbital pain, periorbital edema, proptosis, reduced visual acuity , papilloedema or 3,4,6,5 th nerve paresis. Anterior Cavernous sinus thrombosis causes marked chemosis and proptosis with cranial nerve involvement of 3,4,6, and ophthalmic division of fifth nerve. Posterior cavernous sinus thrombosis spreading to inferior petrosal sinus causes palsies of 6, 9, 10 cranial nerves without proptosis and those involving superior petrosal sinus are accompanied by 5 th cranial nerve palsy. Patients are generally toxic and ill. Thrombus can propagate to the other side and cause bilateral signs. The differential diagnosis includes severe facial and orbital infection, and carotico-cavernous fistula.

In occlusion of sagittal sinus motor deficit (46%), focal (35%) and generalized (47%) seizures are frequent and isolated intracranial hypertension syndrome (17%) is infrequent [40]. The exactly opposite scenario is observed with isolated

thrombosis of the lateral sinus. Multiple cranial nerve palsy (collet-sicard syndrome) is a rare manifestation of lateral sinus, jugular or posterior fossa vein thrombosis [60].

Superficial cortical vein thrombosis:

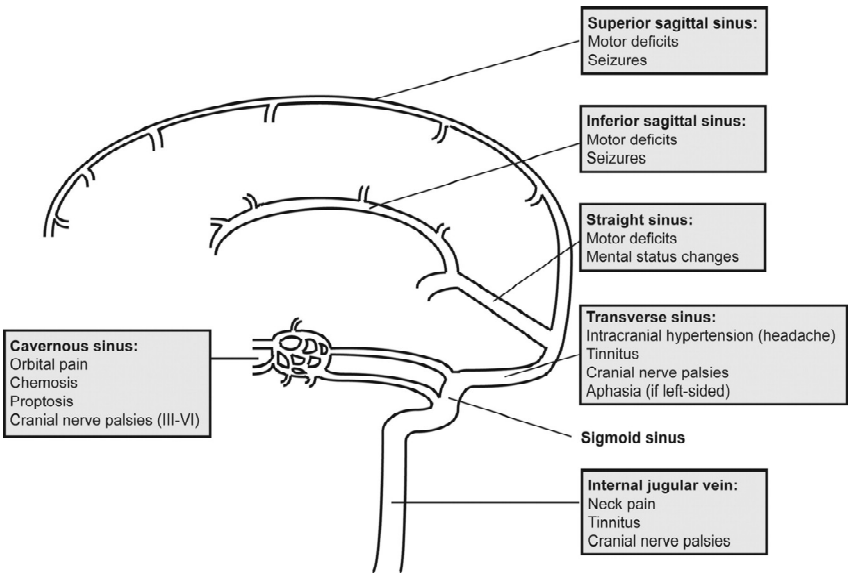
Isolated thrombosis of cortical veins produce large superficial (cortex and subjacent white matter) hemorrhagic infarctions and have marked tendency to produce partial seizures. Hemiparesis, incomplete hemianopia and aphasia are also characteristic of superficial thrombosis of cortical veins.Thrombosis of vein of Labbe causes infarction of underlying superior temporal lobe and occlusion of vein of Trolard implicates parietal cortex. The intracranial pressure is not elevated as it is in dural venous sinus occlusion. It should be suspected when there are multiple hemorrhagic infarctions in one hemisphere without a source of embolism or atherothrombosis. Isolated cortical vein thrombosis is probably underidentified and its diagnosis is difficult to confirm.

Deep cortical vein thrombosis:

Occlusion of vein of Galen and internal cerebral veins is least common and most obscure of venous syndromes. When the deep venous system is involved clinical

features are often severe with coma (67%), mental deficits (87%) and paresis (56%) that can be bilateral [61].

Major clinical syndromes according to location of cerebral venous thrombosis.



CLINICAL COURSE:

Classically the clinical course of CVT is unpredictable [41]. In the ISCVT cohort the clinical course after admission was prospectively investigated, with the following conclusions: about one-fourth of patients deteriorate in status after admission. Neurological worsening may occur several days after admission and may consist of depressed consciousness, mental state disturbance, new seizures, worsening of previous symptoms or new focal deficit, increase in headache severity or visual

loss. About one-third of patients whose status deteriorates show new parenchymal lesions on repeat neuroimaging [62] .Patients with depressed consciousness on admission are more likely to deteriorate and those with seizures at onset are more likely to have repeated seizures. Deterioration is less frequent in patients presenting with isolated headache or with isolated intracranial hypertension syndrome.

DIAGNOSIS:

Often the diagnosis is not quickly considered but is stumbled on, particularly on brain MRI, after others have been considered and then excluded.

CVT should be suspected when a patient develops signs of raised intracranial pressure with or without focal neurological deficits, papilloedema and seizures, particularly in the absence of vascular risk factors and when the CT-brain is normal .CVT may be the underlying cause in patients with features suggestive of diffuse encephalopathy, stroke, and rarely subarachnoid haemorrhage [57], psychosis, or migraine[63]. CVT should be consisdered in all cases with Hemorrhagic infarcts especially if multiple or in nonarterial vascular territories [64].

DIFFERENTIAL DIAGNOSIS: The differential diagnosis is wide and it includes arterial stroke, encephalitis, cerebral abscess, subdural empyema, and cerebral

vasculitis, as well as metabolic and toxic encephalopathies. Features differentiating CVT from arterial stroke are progression and fluctuation, more headache than usual for an arterial stroke, rather typically seizures, any infarct on brain imaging is seldom in a typically 'arterial' pattern and the patients are 'too young' for an ordinary stroke.

INVESTIGATIONS:

IMAGING:

The American Heart Association (AHA) / American Stroke Association (ASA) 2011 scientific statement on diagnosis and management of CVT recommends imaging of the cerebral venous system in patients with suspected CVT [65].

CT Brain:

CT-brain is a useful first line investigation particularly in sick patients in whom MRI is difficult to undertake. Signs detected in CT brain are divided into direct and indirect. Indirect signs are more frequent and include hemorrhagic or non hemorrhagic infarcts outwith the usual arterial territory, edema and intense contrast enhancement of falx and tentorium. Sometimes sub-arachnoid hemorrhage is seen, which is most unusual in either arterial infarcts or primary

intracerebral haemorrhage [66]. Specific but less common changes include the direct signs, which correspond to visualization of thrombus itself, like empty delta sign indicating superior sagittal thrombosis, the cord sign and the dense triangle sign [67]. All though CT brain is the most frequently performed imaging modality it can be normal in upto 25 to 30 % of cases [40,24] and direct signs are seen in only in one-third of patients [65]. Thus CT brain has poor sensitivity compared to MRI brain.

After intravenous contrast there may be gyral, falcine or tentorial enhancement and occasionally, the 'empty delta' sign (hypodensity in the middle of the posterior part of the superior sagittal sinus representing an area of no filling due to thrombus).

The imaging changes in deep CVT are particularly striking with bilateral deep hemorrhagic infarction

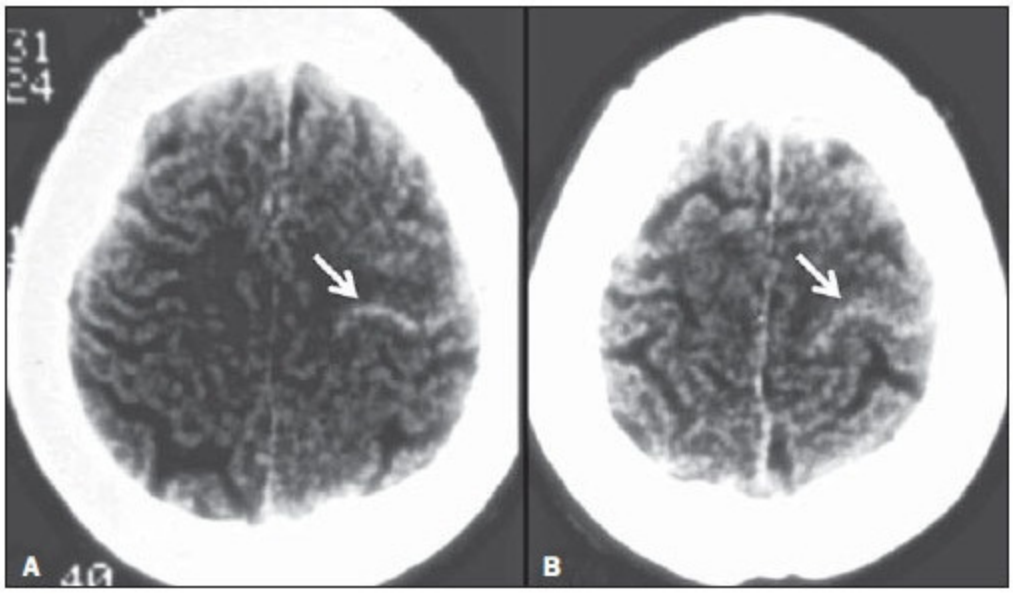


Figure1: CT brain showing CORD SIGN at the site of thrombosis of left superficial frontal vein.

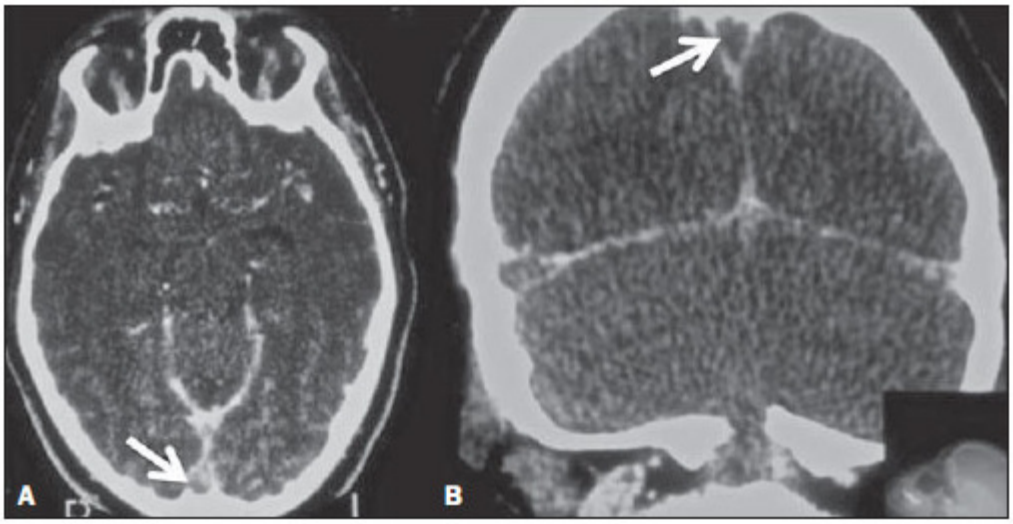


Figure 2: Contrast CT brain image showing EMPTY DELTA SIGN (Superior Sagittal Sinus Thrombosis).

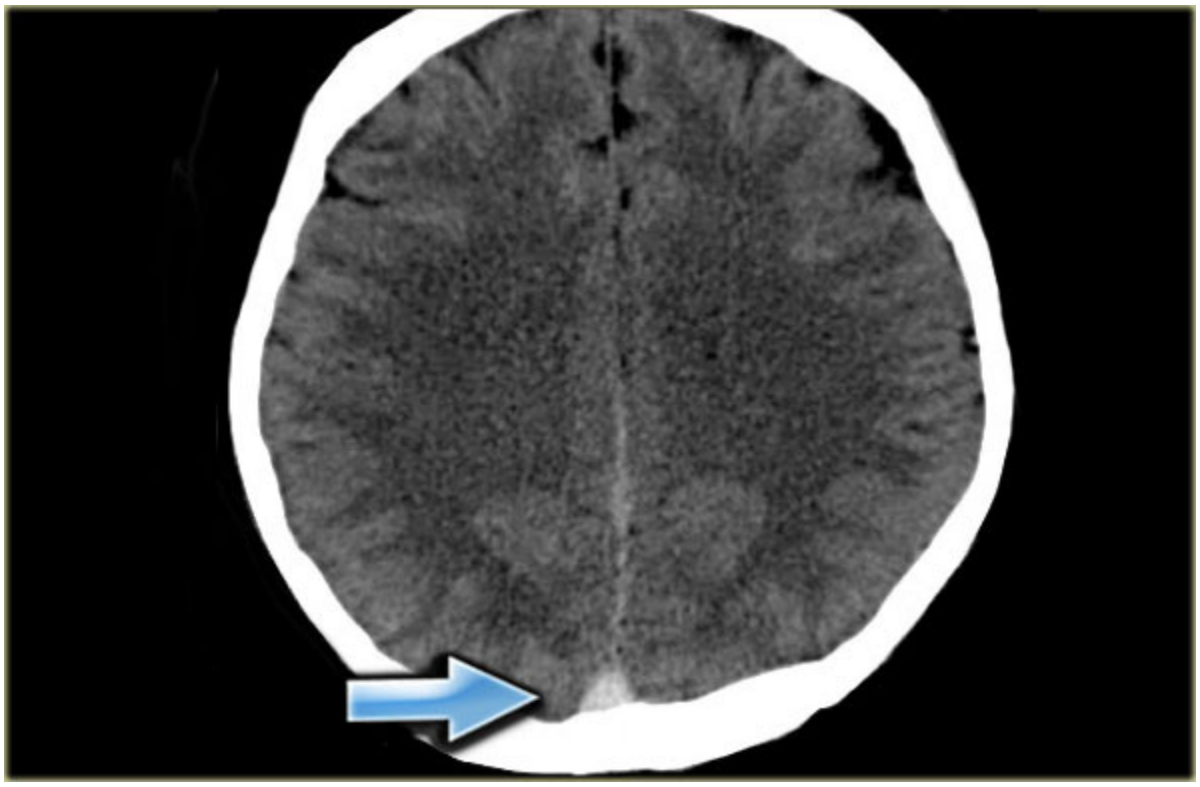


Figure 3: Plain CT Brain showing DENSE TRIANGLE SIGN (SUPERIOR SAGGITAL SINUS THROMBOSIS)

MRI with MRV:

The American Heart Association (AHA) / American Stroke Association (ASA) 2011 scientific statement recommends MR with T2-weighted imaging and MR venography as the imaging test of choice for evaluation of suspected cerebral venous thrombosis [65]. MRI brain with MRV is the most sensitive study for detection of CVT in the acute, subacute and in chronic phases of CVT [1]. In the

acute phase (3 to 5 days), on T1-weighted images the thrombus appears isointense to brain tissue and in T2 –weighted images it appears hypointense. In the subacute phase, thrombus appears hyperintense in both T1 and T2 weighted images. After 2 to 3 weeks findings depend on whether or not the sinus remains occluded or whether it is partly or completely recanalised.

However, MR venography has limited utility in patients with renal impairment.

CT venography:

CT venography is comparable to MR venography for the diagnosis of CVT[68,69]. It provides a rapid and reliable method for detection of CVT especially in patients with contraindications for MRI.

Cerebral arterial angiography:

Cerebral arterial angiography with venous phase imaging is consideres the gold standard investigation for the diagnosis of CVT[24]. But it is invasive and therefore performed when MRV/CT venography is inconclusive [24]. There should be total or partial occlusion of at least one dural sinus on two projections. Often there is also occlusion of cerebral veins, late venous emptying and evidence of venous collateral circulation. A lack of filling of a transverse sinus is not due to congenital

hypoplasia if there is an appropriate sinus groove and jugular foramen on the plain skull X-ray. In obscure subacute encephalopathies, cerebral angiography or MR should always be done to rule out intracranial venous thrombosis before resorting to brain biopsy.

LABORATORY:

Thrombophilia testing:

Because of high frequency of thrombophilia among patients with CVT, screening for hypercoagulable conditions should be performed in patients when needed.

D-dimer:

Although an elevated D-dimer supports the diagnosis of CVT, normal D-dimer level is not sufficient to exclude the diagnosis in patients with compatible clinical presentation [70,71].

EEG: It is abnormal in about 75% of patients but the changes are nonspecific [24].

CSF analysis:

CSF is often abnormal in CVT including raised CSF pressure, elevated protein and pleocytosis [24]. But it is not a routinely recommended investigtion.

Transcranial Doppler ultrasonagraphy and transcranial power or color Doppler imaging with or without the use of contrast agent were reported as potential non invasive techniques for diagnosis of CVT [72], but more studies are needed to determine the true clinical value of these methods.

PROGNOSIS:

Classically the clinical course of CVT is variable and difficult to predict for an individual patient .In general prognosis is less favourable in patients with both extremes of age. In the ISCVT cohort 79% of patients recovered completely [40]. The current case fatality appears to be 10-20 % with further 10-20 % surviving with persistent deficit [36]. Primary cause of death in acute phase of CVT is transtentorial herniation most frequently from large hemorrhagic lesion.

In ISCVT, independent predictors of poor outcome as defined by death or disability at 6 months were age > 37 years, male gender, deep CVT, presence of motor deficit, CNS infections, malignancy, hemorrhage on admission in CT/MRI,GCS score on admission < 9 [36,3].

Predictors of mortality at 30 days in the ISCVT cohort were depressed consciousness, mental status disorders, deep CVT, right hemispheric hemorrhage and posterior fossa lesion [73].

COMPLICATIONS:

Complications with patients who survived acute phase of CVT are further venous thrombotic events, seizures and headache. Headache severe enough to require hospital admission occur in 14% of patients with CVT [3]. In such case MRI with MRV are necessary to exclude recurrent CVT and other intracranial lesions and to document persistent venous occlusion, partial or complete sinus recanalisation or dural sinus stenosis. Seizures occur in upto 11% of patients [40]. Severe visual loss due to intracranial hypertension can occur rarely[3].

Psychological and cognitive complaints are not uncommon among CVT survivors. About half of the survivors of CVT may become depressed or anxious and can manifest minor cognitive or language deficit which may preclude resumption of previous levels of professional activity [74].

Sequelae of CVT include cognitive and motor impairment, seizures, symptomatic persistent intracranial hypertension.

Recurrence of CVT is rare and difficult to document. In the ISCVT cohort, 2.2% of patients had recurrent CVT and 4.3% had other thrombotic complications[40] especially DVT and pulmonary embolism.

In CVT, recanalisation of thrombosed vein and sinuses occurs in about 40 to 90 % of patients, especially with in the first 3 to 4 months [75]. It is useful for patients with CVT to undergo MRI/MRV 3 to 6 months after CVT to document extent of recanalisation. However in adults recanalisation of occluded sinus is in no way related to outcome after CVT.

TREATMENT:

Treatment of CVT includes

1. Antithrombotic treatment,

2. Symptomatic treatment of intracranial hypertension, seizures, headache and visual failure.

3. Etiological treatment of associated conditions and risk factors.

Antithrombotic treatment:

The guidelines for the treatment of CVT was issued by the European federation of neurological societies (EFNS) in 2006 [76]. The EFNS guidelines advice that patients with CVT without contraindications to anticoagulation should be treated either with body weight adjusted subcutaneous low molecular weight heparin or dose adjusted intravenous heparin. On the basis of data from randomized controlled

trails and observational studies anticoagulation is recommended as safe and effective for treatment of CVT with or without intracranial hemorrhage on presentation [65,76] .Hence concomitant intracranial hemorrhage is not a contraindication for anticoagulation therapy.

Aims of antithrombotic treatment are to recanalize the occluded sinus or veins, to prevent further propagation of thrombus, to prevent pulmonary embolism and to treat the underlying prothrombotic state inorder to prevent the recurrence and thrombus formation in other parts of body.

Risk of intracranial hemorrhage is <5% and systemic hemorrhage is <2% following heparin use and such hemorrhages do not influence the outcome[77].

There is no evidence regarding the safety and efficacy of antiplatelet drug therapy in CVT.

Fibrinolysis:

Evidences for the use of either systemic or local thrombolysis in the treatment of CVT are not sufficient[24]. Catheter related fibrinolysis may be considered at experienced centers for patients who have large and extensive CVT or who clinically worsen despite anticoagulation, possibly those without intracranial

hemorrhage. A review conducted on 169 patients with CVT showed substantial clinical benefit on treating those with severe disease manifestations with fibrinolysis [78]. However, 17% of patients developed intracranial hemorrhage after fibrinolysis and clinical deterioration occurred in 5% of patients[78]. Mechanical endovascular disruption of thrombus can also be done.

Surgical interventions:

Surgical thrombectomy is reserved for the rare circumstance in which severe clinical deterioration occurs despite maximal medical therapy [65].

Treatment of intracranial hypertension:

It includes elevating the head end of the bed, antiedema measures with osmotic diuretics, for example: mannitol, hyperventilation therapy to a target Paco2 of 30 to 35 mmhg, decompression surgeries such as craniectomy or hematoma evacuation in patients with impending herniation [79].These measures are associated with improved clinical outcomes and can be life saving. If severe headache persist or if visual acuity is decreasing, repeated lumbar punctures, a lumboperitoneal shunt, stenting of a sinus stenosis or fenestration of the optic nerve sheath can also be done [40].

Treatment of the underlying cause:

Any underlying cause should be treated, for example, lifelong anticoagulation should be given for patients with severe hereditary thrombophilia, patients with OCP associated CVT should never use OCPs again. But pregnancy or puerperium associated CVT is not a contraindication for future pregnancies because the risk of CVT in subsequent pregnancies among those with a history of pregnancy or puerperium associated CVT earlier is considered to be low [3,45,80].

Long term management:

AHA/ASA 2011 scientific statement recommends anticoagulation with oral vitamin K antagonist and a target INR of 2-3 for 3 to 6 months for those with provoked CVT and 6-12 months for those with unprovoked CVT and for those with mild hereditary thrombophilia [65]. An indefinite period of anticoagulation is recommended for those with 2 or more episodes of CVT and for those with 1 episode of CVT along with an associated severe thrombophilia [65].

Seizures:

The risk factors for subsequent early seizures in those with CVT are acute seizures and supratentorial lesions. Prophylactic antiepileptics should be considered in

patients with these risk factors [59]. The long term risk of remote seizures is approximately 11% [3]. The risk factors for remote seizures are acute seizures, motor deficit and supratentorial hemorrhagic lesions. Long term antiepileptics are recommended for patients with these risk factors [40]. Valproate is preferred to phenytoin and carbamazepine because of the lesser interactions with oral anticoagulants compared to the others and also because it can be used intravenously. If valproate is not tolerated, newer antiepileptics like Lamotrigine, Levetiracetam or Topiramate can be used.

MATERIALS AND METHODS

Study design: Cross sectional study.

Study material:

The study was conducted on 45 radiologically confirmed cases of cerebral venous thrombosis admitted in Medicine and Neurology departments of Thanjavur medical college hospital during the period from November 2010 to November 2012.

Inclusion criteria:

1. Patients with confirmed clinical and radiological diagnosis of cerebral venous thrombosis and

2. With age >12 years were included in our study.

Exclusion criteria:

1. Patients whose clinical presentation could be explained by any other neurological disease.
2. Patients without radiological evidence of CVT.
3. Patients with age <12 yrs were excluded from our study.

Methodology:

The study was conducted on 45 patients of cerebral venous thrombosis.

The diagnosis of CVT was based on appropriate clinical findings supported by radiological evidence of CVT. Radiological diagnosis was based on established radiological criteria [11-17].

We obtained Informed consent from all of our patients.

In all the 45 patients detailed history including demographic factors, type and duration of symptoms, onset of symptoms : acute (<48 h), subacute (48 h to <30 days), and chronic (>30 days) [18], features suggestive of etiological factors, personal habits, comorbid illnesses, detailed menstrual and obstetric history in case of females was taken. All the patients were subjected to detailed clinical examination including general, neurological and other systems examination. Assessment of consciousness level, Glascow coma scale[19] score at the time of admission were also recorded in all patients. In GCS, the grading of severity is done as follows, severe (GCS score of ≤ 8), moderate (GCS score of 9-12), and mild (GCS score of 13-15) [19].

Neuroimaging in the form of CT brain and MRI brain with MRA and MRV was done in all patients. Details like presence of cerebral edema, haemorrhagic infarct, non

haemorrhagic infarct, presence of direct signs like cord sign, dense delta sign etc., occurrence of focal or diffuse subarachnoid haemorrhage in CT brain were recorded. In MRI with MRV, type and number of sinuses involved, involvement of cortical veins, internal jugular vein extension, and laterality of the sinuses involved were noted.

Investigations like complete blood count, erythrocyte sedimentation rate (ESR), blood urea, blood sugar, serum creatinine, serum electrolytes, lipid profile, X-ray chest, Electrocardiogram, Elisa for Human Immunodeficiency Virus (HIV),VDRL, coagulation profile including bleeding time, clotting time, prothrombin time, activated partial thromboplastin time were done in all patients. Males with haemoglobin level of <13 g/dl and females with haemoglobin level of <12 g/dl were considered to have anemia in our study [18].

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

Outcome at the end of the hospital stay was recorded in all patients. The modified Rankin score was used for outcome assessment [20]. The modified Rankin

Scale (mRS) is commonly used for expressing the degree of disability of post stroke patients as well as those with other neurological disability. Hence it is widely used in the analysis of outcome in stroke clinical trials. mRS was actually introduced by Dr. John Rankin of Stobhill Hospital Glasgow, Scotland [21] in 1957 and was first modified by Prof. C. Warlow's group at Western General Hospital in Edinburgh [22]. The currently used modified Rankin Scale was given by van Swieten, et al., in 1988[23].

The scoring is done from 0 to 6 as follows,

0 - No symptoms.

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

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

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

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

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

6 - Dead.

In our study, good outcome was defined as modified Rankin Scale score of 0 to 2 and poor outcome was defined as modified Rankin Scale score of >2 [5]. Factors associated with good and poor outcome were also analysed in our study.

These demographic, clinical, laboratory, neuroimaging, outcome data were recorded and analysed using a standard proforma. Statistical analysis was done using SPSS software. Statistical analysis used descriptive, univariate and multivariate methods. Continuous variables were presented as mean, median and ± SD. Categorical variables were expressed as proportions and Fischer's test was used to study the association in proportions. We estimated the relative risk and the resulting 95% Confidence Interval to study associations. P value of equal to or less than 0.05 was considered statistically significant.

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

OBSERVATION AND RESULTS

Our study was conducted in 45 patients of cerebral venous thrombosis. We analysed the demographic characters, onset and type of symptoms, etiology and clinical features of the disease in these patients. All of them were subjected to imaging modalities like CT brain and MRI brain with MRA and MRV and we analysed the imaging characters of the disease in these 45 patients. We analysed the in hospital mortality rate and morbidity rate at the time of discharge. We also analysed the factors associated with in hospital mortality, poor outcome defined as mRS score of >2 and good outcome defined as mRS score of ≤2 and we observed the following.

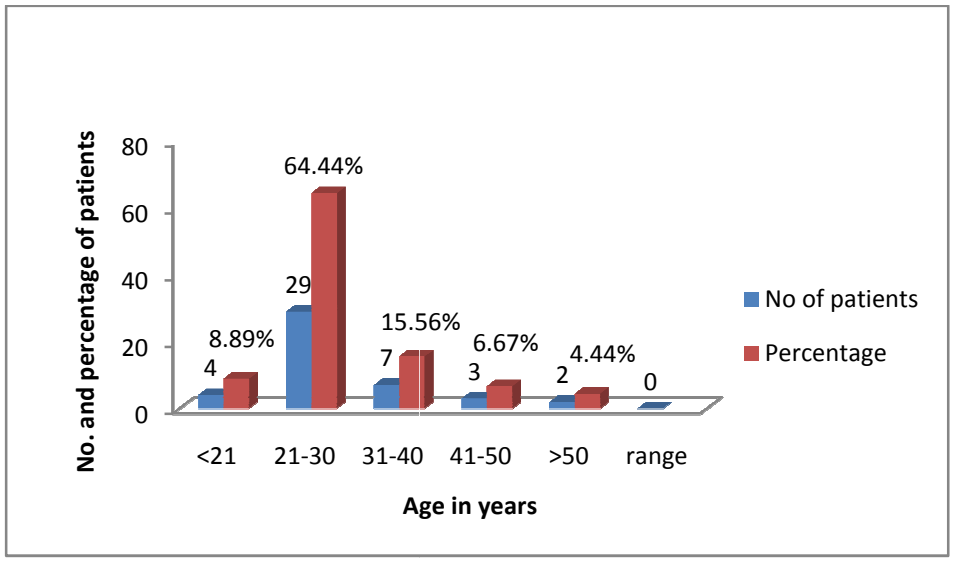
Age distribution:

Age range of our patients was 17 to 65 years. Of the 45 patients, 64% patients were in the age group of 21 to 30 years.

Table 1: Showing Age Distribution

Age range (in years)	No. of patients	Percentage
<21	4	8.89
21-30	29	64.44
31-40	7	15.56
41-50	3	6.67
>50	2	4.44

Figure1: Showing Age Distribution



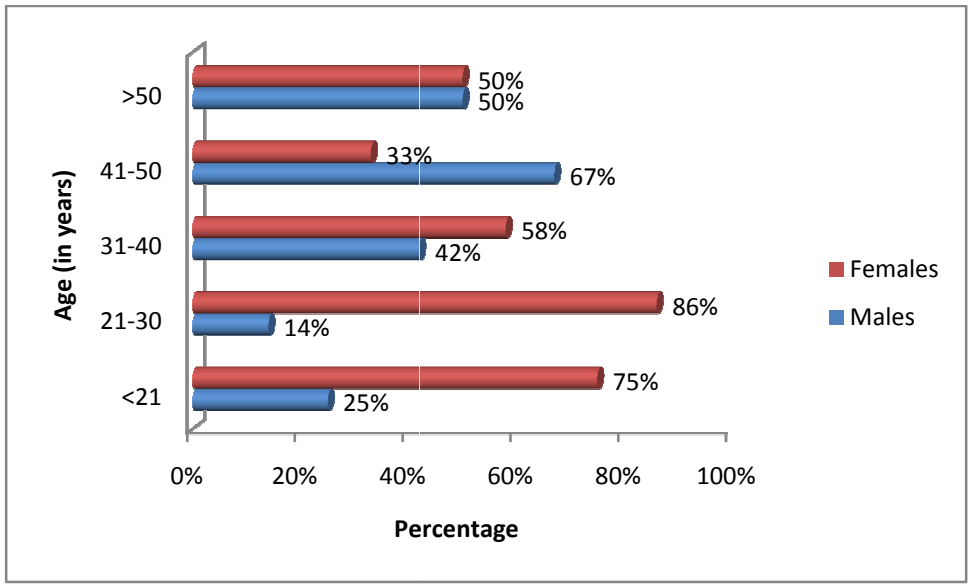
Mean age of our patients was 29.29 years and the median was 26 years.

Distribution of sex in different age groups was also analysed and it showed that females formed the majority of the young patients in our study.

Table 2: Showing sex distribution in different age groups

Age (in years)	Total no. of patients	Males(No. of patients and percentage)	Females(No. of patients and percentage)
<21	4	1(25%)	3(75%)
21-30	29	4(14%)	25(86%)
31-40	7	3(42%)	4(58%)
41-50	3	2(67%)	1(33%)
>50	2	1(50%)	1(50%)

Figure 2: Showing sex distribution in different age groups



Peak age incidence was between 21 to 30 years with major contribution from females (86%). The mean age of females was lower (27.71yrs) than that of males (34.18 yrs).

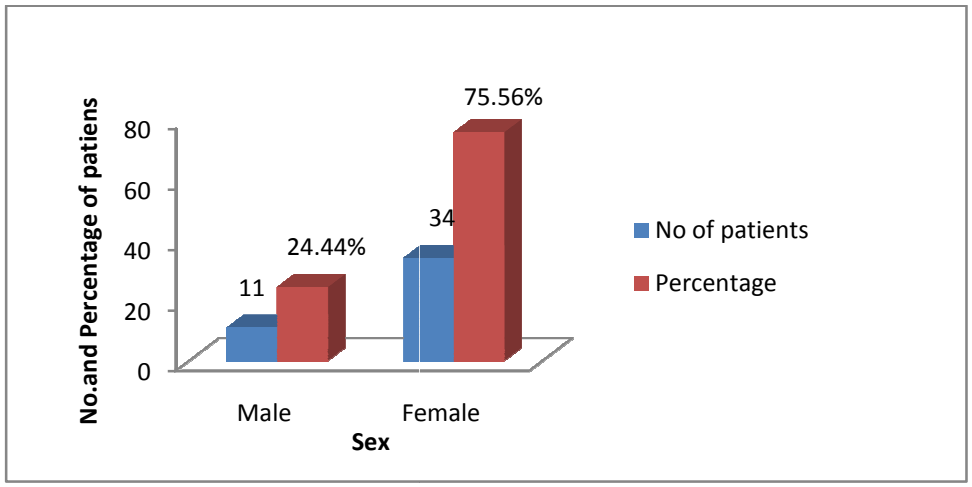
Sex distribution:

Table 3: Showing sex distribution

Sex	No. of patients	Percentage
Male	11	24.44
Female	34	75.56

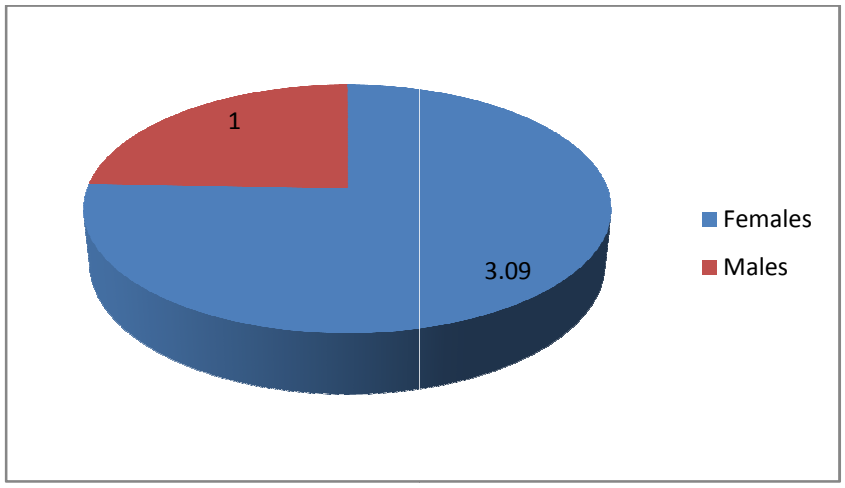
In our study, 75.56 % of patients were females.

Figure 3: Showing sex distribution



Male female ratio was M:F=1:3.09.

Figure 4: Showing Male female ratio



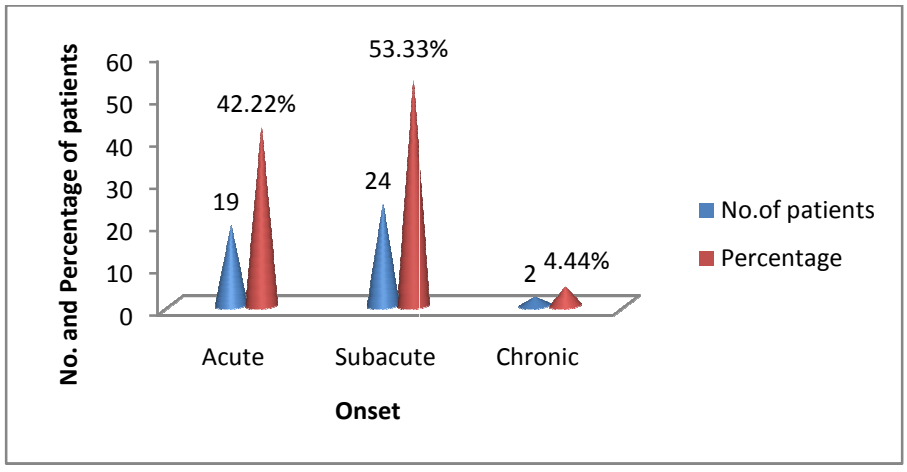
Onset of symptoms:

Onset of the symptoms was analysed as acute (≤2 days), subacute (3 to 30 days) and chronic (≥30 days). Our observations were as follows,

Table4: Showing onset of symptoms

Onset	No. of patients	Percentage
Acute	19	42.22
Subacute	24	53.33
Chronic	2	4.44

Figure 5: Showing onset of symptoms



About 53.33% of patients had subacute onset of symptoms. The median and mean duration of the presenting illness was 3 days and 6 days respectively.

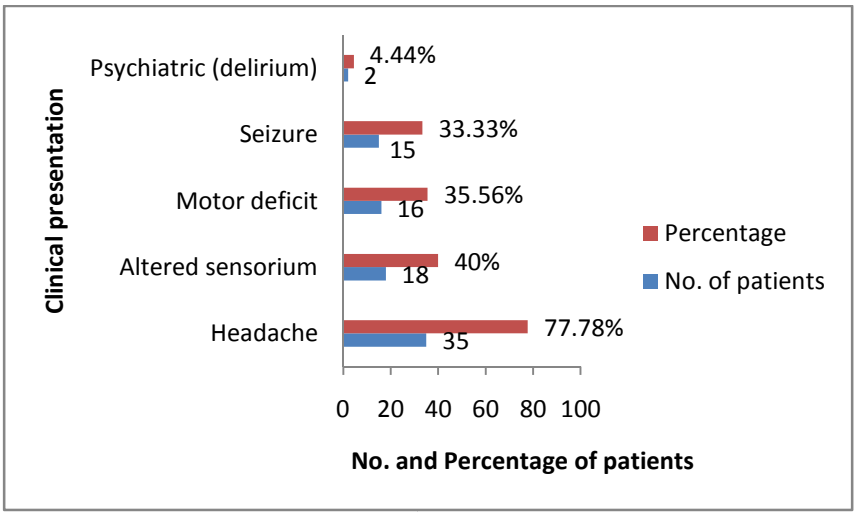
PRESENTING ILLNESS:

The most common presenting symptom was headache (77.78%) followed by altered sensorium (40%), motor deficit (35.56%) and seizure (33.33%). 2(4.44%) patients presented with delirium.

Table 5: Showing presenting symptoms

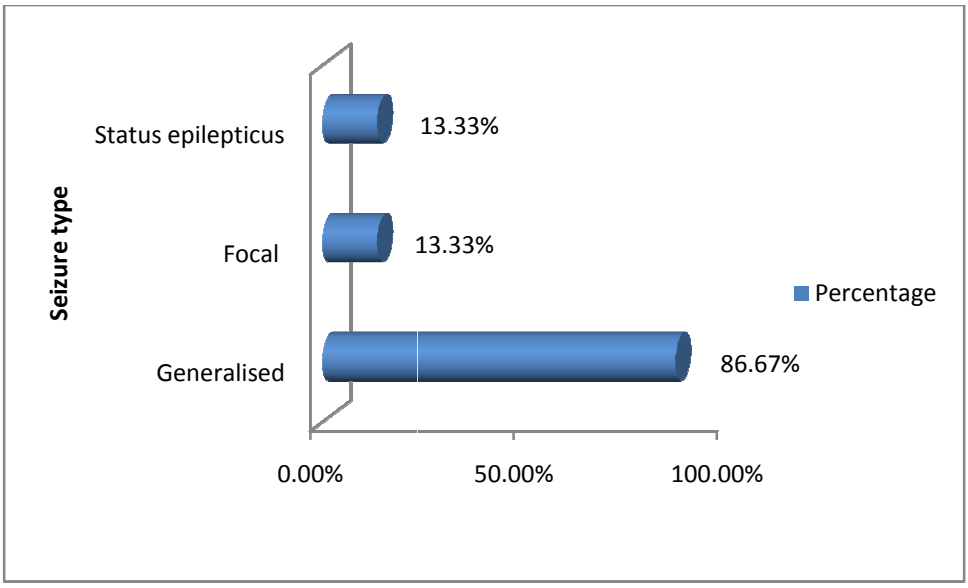
Presenting symptoms	No. of patients	Percentage
Headache	35	77.78
Altered sensorium	18	40
Motor deficit	16	35.56
Seizure	15	33.33
Delirium	2	4.44

Figure 6: Showing Presenting symptoms



Among the 15 patients who presented with seizure, 13 patients (86.67%) presented with generalized seizure. Focal seizure occured in just 2 (13.33%) patients and 2(13.33%) patients presented with status epilepticus.

Figure 7: Showing seizure type



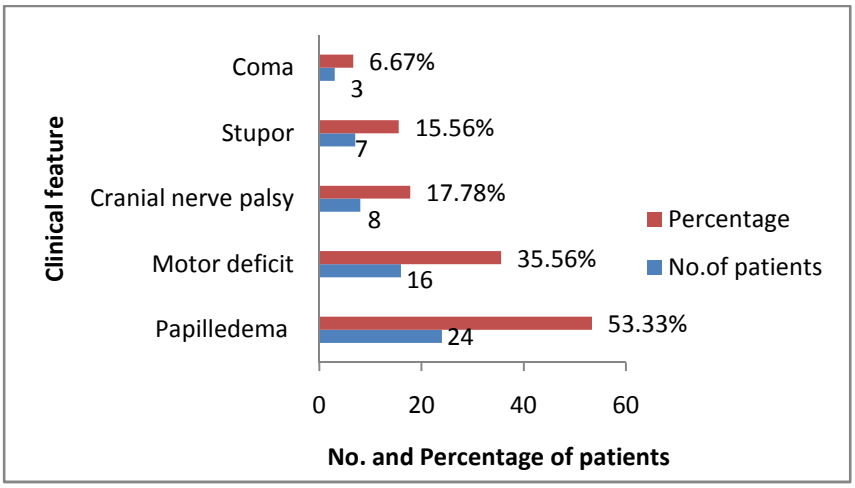
Clinical features:

Papilledema was the most common clinical finding occurring in 24(53.33%) of patients. Focal motor deficit most commonly hemiplegia (11 patients) occurred in 16 (35.56%) patients. 8(17.78%) patients had cranial nerve palsy. Most common nerve involved was the 6^{th} nerve. One patient who had a chronic onset presented with bilateral secondary optic atrophy (secondary to long standing papilledema).7 (15.56%) patients were stuporous and 3(6.67%) patients were comatosed.

Table 6: Showing clinical features

Clinical feature	No.of patients	Percentage
Papilledema	24	53.33
Motor deficit	16	35.56
Cranial nerve palsy	8	17.78
Stupor	7	15.56
Coma	3	6.67

Figure 8: Showing clinical features



None of our patients had sensory deficit, cerebellar signs or features of autonomic system involvement.

Signs of meningeal irritation occurred in 2 of our patients.

Majority of our patients had mixed features of the 3 classical syndromes namely Isolated Hypertension syndrome, focal syndrome and encephalopathy. However Isolated Hypertension Syndrome (IHS), consisting of headache with or without vomiting, papilledema, and visual troubles occurred in 13(28.88%) patients. All the 13 patients had headache; papilledema occurred in 9 of them and 4 had cranial nerve palsy, most common being the 6thcranial nerve palsy.

Etiology:

Most common etiological factor noted in our study was puerperium.

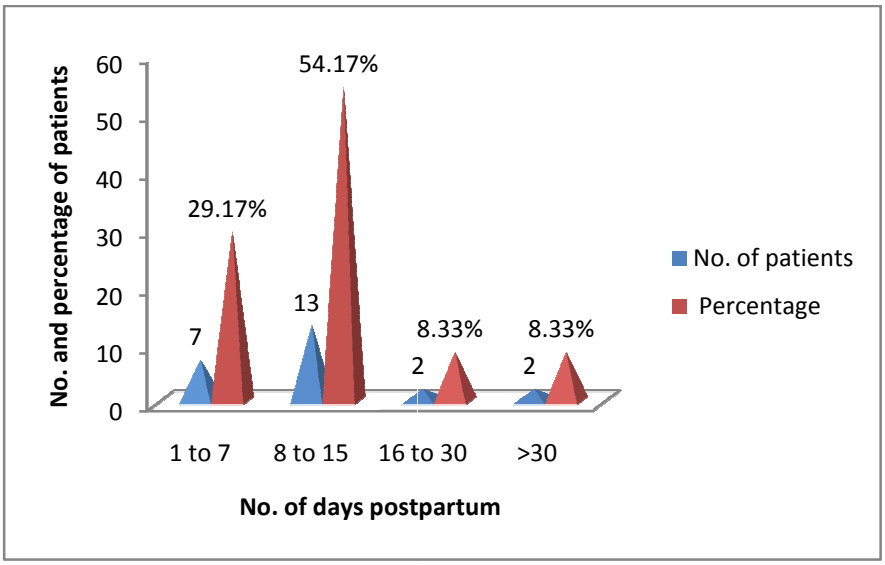
Among the 45 patients, about 24 females (53.33%) were in puerperium. They formed 70.59% of the total 34 females included in our study.The mean age of these females with puerperal CVT was lower (26.32yrs) than that of other females (32.4yrs).

Among these 24 patients, 20(83.34%) patients had symptom onset within 2 weeks postpartum especially between 8 to 15 days postpartum.

Table 7: Showing No. of days of onset of symptoms postpartum

No. of days Postpartum	No. of patients	Percentage
1 to 7	7	29.17
8 to 15	13	54.17
16 to 30	2	8.33
>30	2	8.33

Figure 9: Showing No. of days of onset of symptoms postpartum



Among the 24 patients, 10(41.67%) patients were of second gravida, 8(33.33%) were Primigravida and 6(25%) were of third gravida.

Among the 24 patients 6(25%) were post LSCS patients and none of these females had PIH in the antepartum period.

Dehydration, one of the important causes of CVT was noted in 3 of our patients who were post operative, post LSCS and alcoholic each.

One of our patient was a case of systemic lupus erythematosis. She was a 17 year old female who presented with acute onset headache and generalized seizure. She presented in status epilepticus. She had superior sagittal sinus thrombosis

and she tested positive for antiphospholipid antibody syndrome. She also had associated autoimmune thyroiditis. She had good recovery.

Another patient tested positive for antiphospholipid antibody test and negative for dsDNA. She was a 22 year old female who presented with acute onset headache and papilledema with history of recurrent abortions. Her CT brain was normal but MRI with MRV showed superior sagittal sinus thrombosis and she recovered well.

One patient had hyperhomocystenemia. He was a 21 year old male who presented with subacute onset of headache, vomiting, papilloedema and 6^{th}cranial nerve palsy. He had non haemorrhagic infarct in CT brain and thrombosis of left transverse sinus, straight and sigmoid sinus without involvement of SSS.

2 patients had CVT associated with meningitis. One patient was a 26 year old female, 3^{rd} gravida, who presented 20 days post LSCS with fever, headache, stupor and monoplegia. She had haemorrhagic infarct in CT brain and thrombosis of left side transverse sinus in MRI with MRV. The other was a 20 year old female who presented with subacute onset fever, headache, altered sensorium and SSS thrombosis.

Another patient, a known case of Polycythemia vera presented with Superior sagittal sinus thrombosis. He was a 29 year old male diagnosed earlier to have polycythemia vera and on irregular follow up, presented with subacute onset headache and delirium and haemorrhagic infarct in CT brain secondary to SSS thrombosis.

One of our patients presented 4 days post partum with headache and generalized seizure, along with bleeding tendencies. She was a 30 year old female, who had developed intrauterine death of foetus at 30 weeks antepartum; foetus had been delivered by induction of labour 4 days prior to landing in our hospital. She was found to have disseminated intravascular coagulation. She had haemorrhagic infarct in CT brain and MRI with MRV showed superior sagittal sinus thrombosis.

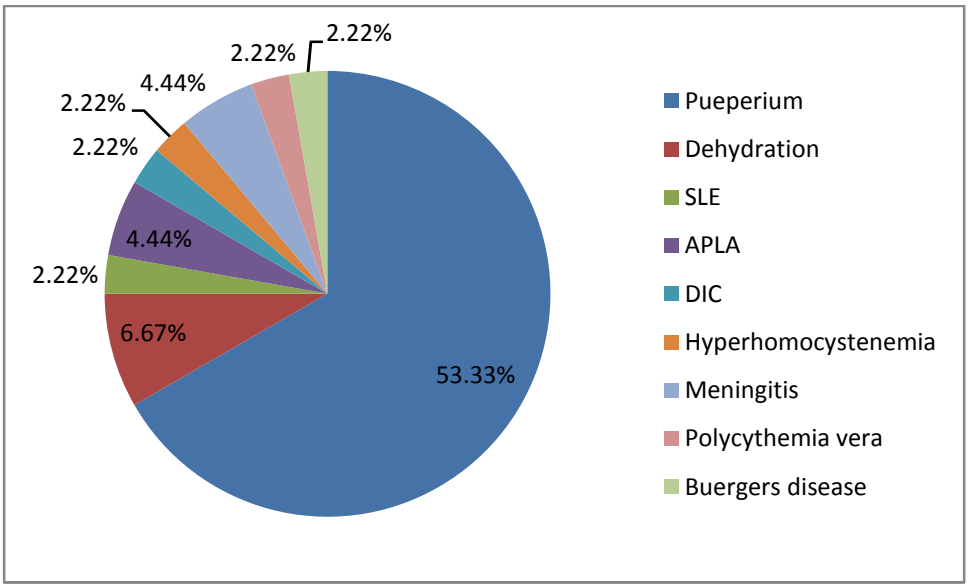
One of our patient was a known case of Bueger's disease, on anticoagulant therapy. He was 37 year old male, chronic smoker, who had undergone amputation of right great toe 5 years back. He presented with 15 days history of headache, right hemiperesis and visual disturbances. He had bilateral secondary optic atrophy, and bilaterally weak popliteal, posterior tibial and dorsalis pedis pulses. His CT brain showed hypodensity in the left caudate nucleus, frontal and temporal region.MRI brain showed thrombosis of SSS, left transverse, sigmoid

sinus with subarachnoid haemorrhage in left fronto temporo parietal region and lacunar infarct in left lentiform and caudate nucleus.MRV showed thrombosis of all major dural venous sinuses except straight sinus with multiple venous collaterals and multiple lacunar infarcts in left basal ganglia,left deep periventricular white matter.

Table 8: Showing Etiology

Etiology	No. of patients	Percentage
Pueperium	24	53.33
Dehydration	3	6.67
SLE	1	2.22
APLA	2	4.44
DIC	1	2.22
Hyperhomocystenemia	1	2.22
Meningitis	2	4.44
Polycythemia vera	1	2.22
Bueger's disease	1	2.22

Figure 10: Showing Etiology



RADIOIMAGING

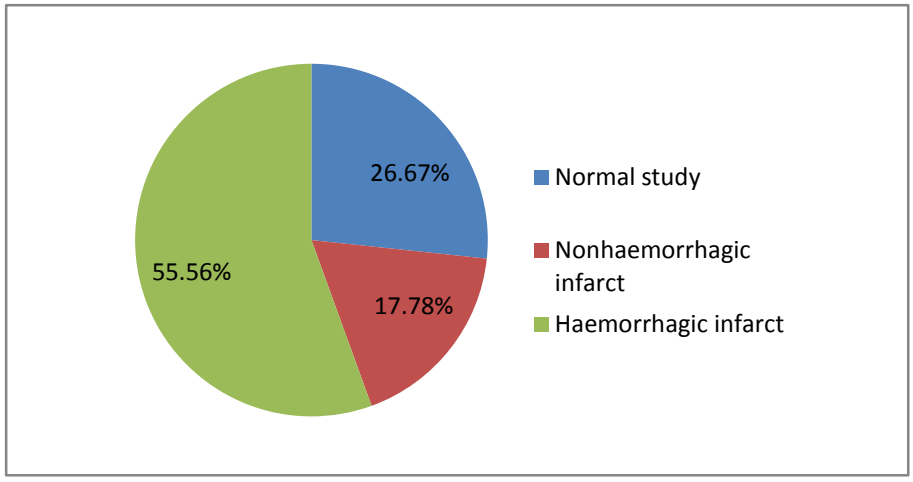
All the 45 patients included in our study were subjected to both CT brain and MRI brain with MRV.

CT brain was done as an initial investigation and about 26.67% of our patients had normal study. Haemorrhagic infarct, characteristic of venous infarct occurred in 55.56% of patients and non haemorrhagic infarct occurred in 17.78% of patients.

Table 9: Showing features in CT brain

CT Brain	No. of patients	Percentage
Normal study	12	26.67
Nonhaemorrhagic infarct	8	17.78
Haemorrhagic infarct	25	55.56

Figure 11: Showing features in CT brain



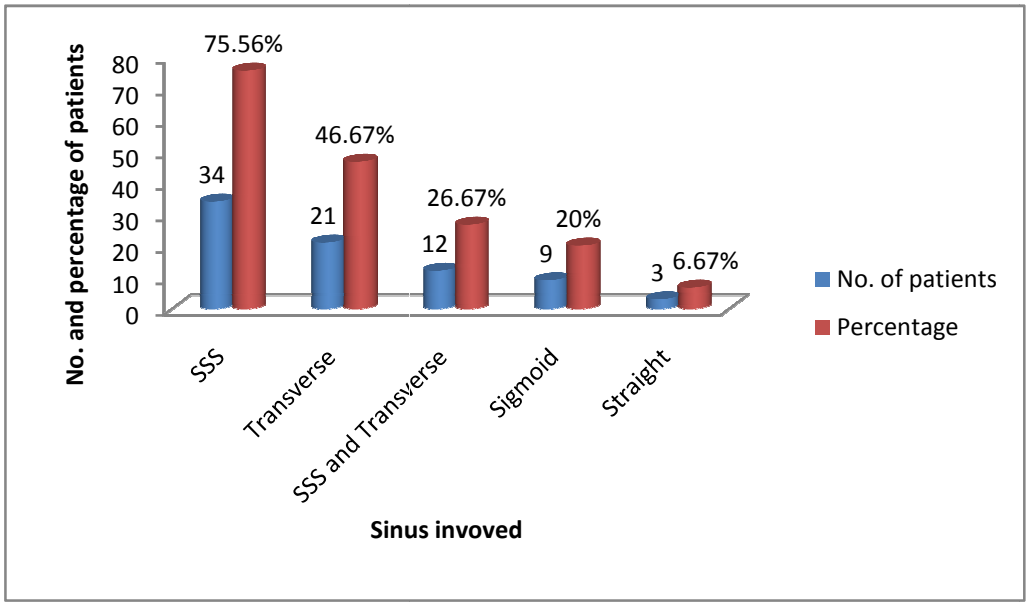
Subarachnoid haemorrhage was noted in 2(4.44%) of our patients. Direct signs were observed in just 11.11% of our patients most commonly the dense delta sign suggesting superior sagittal sinus thrombosis.

MRI with MRV showed that among the individual sinuses, superior sagittal sinus (SSS) was the most common sinus involved (75.56% of patients), followed by the transverse sinus (46.67% of patients). Less commonly involved were the sigmoid and the straight sinus. 37.78% of patients showed involvement of more than 1 sinus, most commonly involvement of both SSS and transverse sinus (26.67% of patients).

Table 10: Showing involvement of sinuses by MRI with MRV

Sinus involved	No. of patients	Percentage
Superior sagittal sinus(SSS)	34	7.56
Transverse	21	46.67
SSS and Transverse	12	26.67
Sigmoid	9	20.00
Straight	3	6.67

Figure 12: Showing involvement of sinuses by MRI with MRV



Also 5 (11.11 %) patients showed involvement of more than 2 sinuses of which 2(4.44%) had involvement of all 4 sinuses.

Among the patients with transverse sinus involvement, right side transverse sinus was more commonly involved (11 patients) than the left (8 patients) and 2 patients had bilateral transverse sinus involvement.

Among the 45 patients, 7 patients (15.56%) had extension of the thrombus into the internal jugular vein; 8 patients (17.78%) had associated involvement of cortical veins of which 2(4.44%) had extensive bilateral cortical veins thrombosis.

None of our patients had isolated cortical vein thrombosis or deep vein thrombosis.

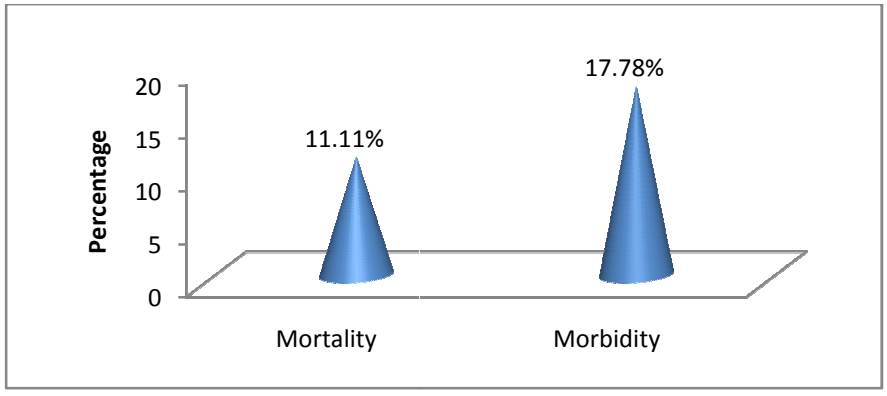
OUTCOME:

5 patients died during the in hospital stay. 8 patients had residual neurological deficit at the time of discharge. Hence the mortality rate in our study was 11.11% and morbidity rate was 17.78%.

Table 11: Showing Mortality and Morbidity

Outcome	No. of patients	Percentage
Mortality	5	11.11
Morbidity	8	17.78

Figure 13: Showing Mortality and Morbidity



Among the 5 deaths, 1 (20%) was a male and 4 (80%) were females. One of these 4 female deaths was a post partum death.

Factors associated with in hospital mortality in our study were analysed.

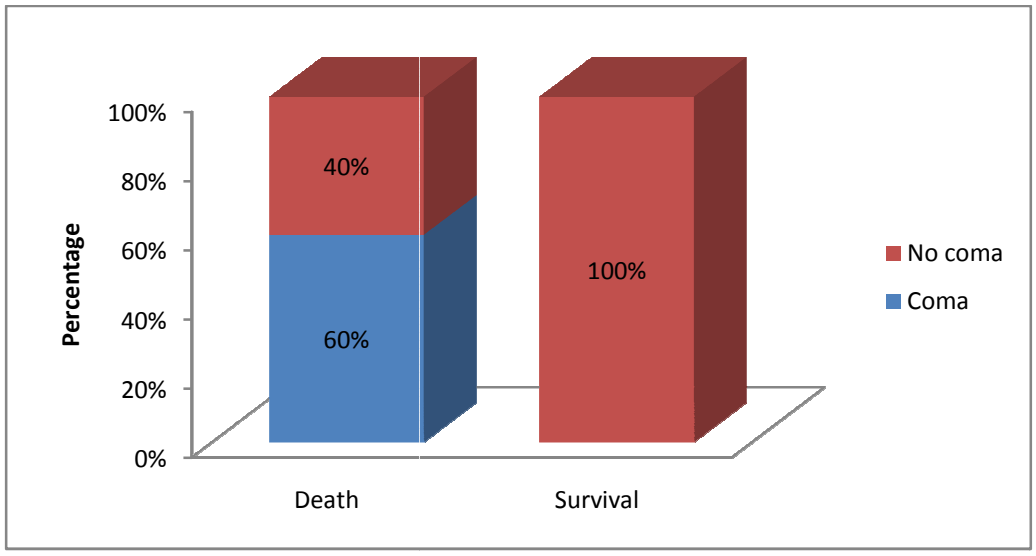
Coma at the time of admission was shown to be significantly associated with in hospital mortality. (p=0.0007; considered extremely significant).

Table 12: Showing comparison of coma with survival and death

	Coma(No. of patients and Percentage)	No coma(No. of patients and Percentage)
Death	3(60)	2(40)
Survival	0	40(100)

By Fischer's test p=0.0007

Figure 14: Showing Percentage of coma in survival and death



The association of age with in hospital mortality was assessed. The median and mean age of patients who died was greater (median=36yrs,mean=38.8yrs) than those who survived(median=26yrsmean=28.1yrs).

Also age ≥35 years was significantly associated with in hospital mortality.

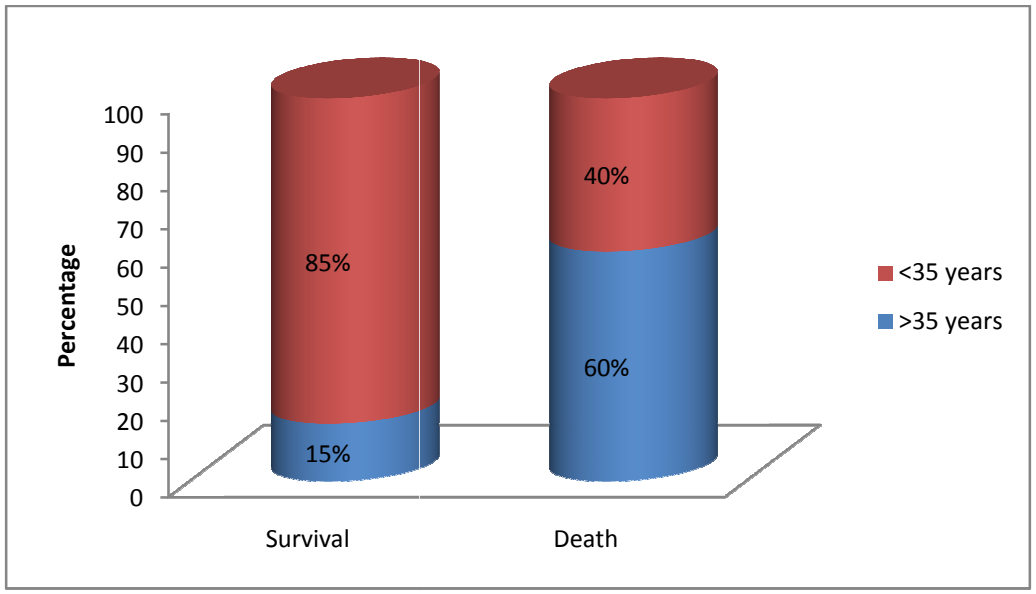
Table 13: Showing comparison of age with death and survival

	Age ≥35 years(No. and percentage of patients)	Age <35 years(No. and percentage of patients)
Survival	6(15)	34(85)
Death	3(60)	2(40)

By Fischer's test p=0.0471, considered significant.

Relative risk=4.000, 95% Confidence Interval: 1.431 to 11.183 (using the approximation of Katz)

Figure 15: Showing age distribution in death and survival



Association of GCS with in hospital mortality was studied.

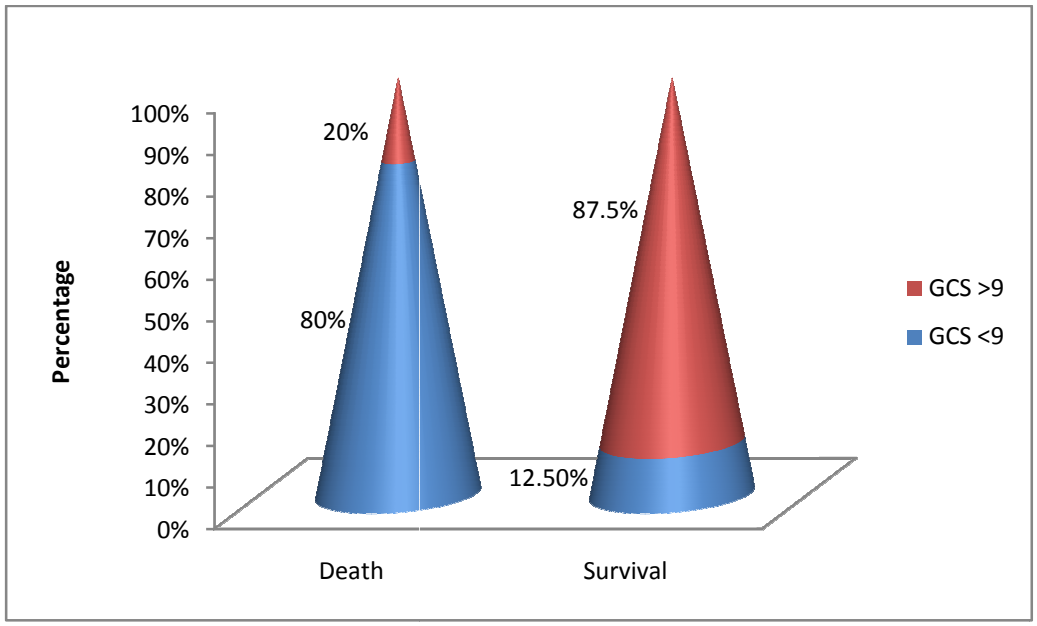
Table 14: Showing comparison of GCS with death and survival

	GCS <9 (No. and percentage of patients)	GCS≥9 (No. and percentage of patients)
Death	4(80)	1(20)
Survival	5(12.5)	35(87.5)

By Fischer's test P value is 0.0038, considered very significant.

Relative risk = 6.400, 95% Confidence Interval: 2.525 to 16.220 (using the approximation of Katz.)

Figure 16: Showing GCS score in death and survival



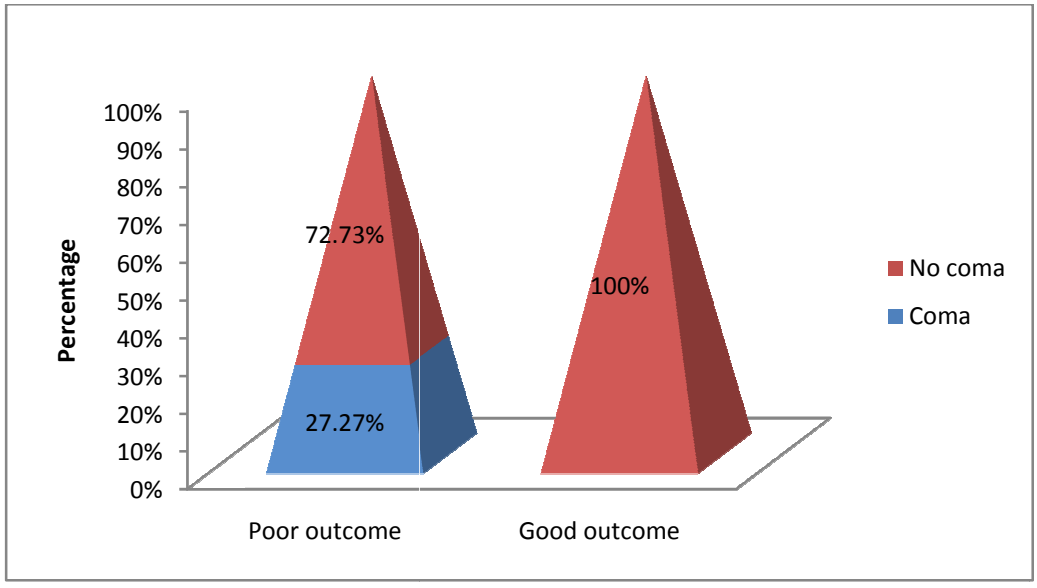
Poor outcome defined as modified Rankin Scale score of >2 was seen in 13 patients. Factors associated with poor outcome were analysed in our study. We found that coma at the time of admission was significantly associated with poor outcome.

Table 15: Showing comparison of coma with poor outcome

	Coma(No. and percentage of patients)	No coma(No. and Percentage of patients)
Poor outcome	3(27.27)	8(72.73)
Good outcome	0	34(100)

By Fischer's test, p=0.0116, considered significant.

Figure 17: Showing percentage of coma in good and poor outcome



Presence of motor deficit was also significantly associated with poor outcome as shown below.

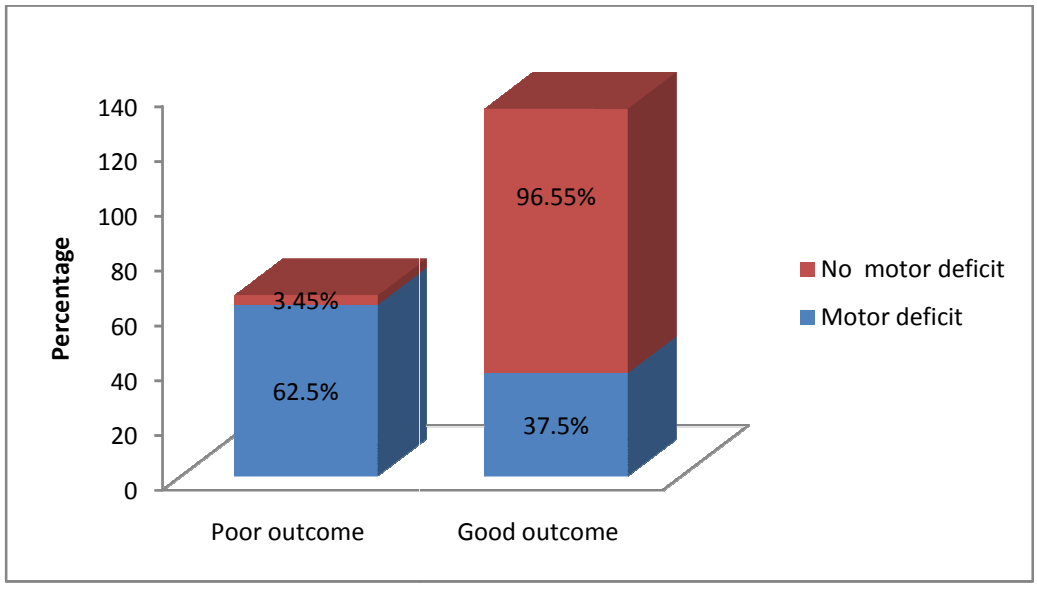
Table 16: Showing comparison of motor deficit with poor outcome

	Motor deficit (No.and Percentage of patients)	No motor deficit(No. and Percentage of patients)
Poor outcome	10(62.5)	1(3.45)
Good outcome	6(37.5)	28(96.55)

By Fischer's test, p=0.0001, considered extremely significant

Relative risk = 5.152, 95% Confidence Interval: 2.433 to 10.905 (using the approximation of Katz).

Figure 18: Showing percentage of motor deficit in good and poor outcome



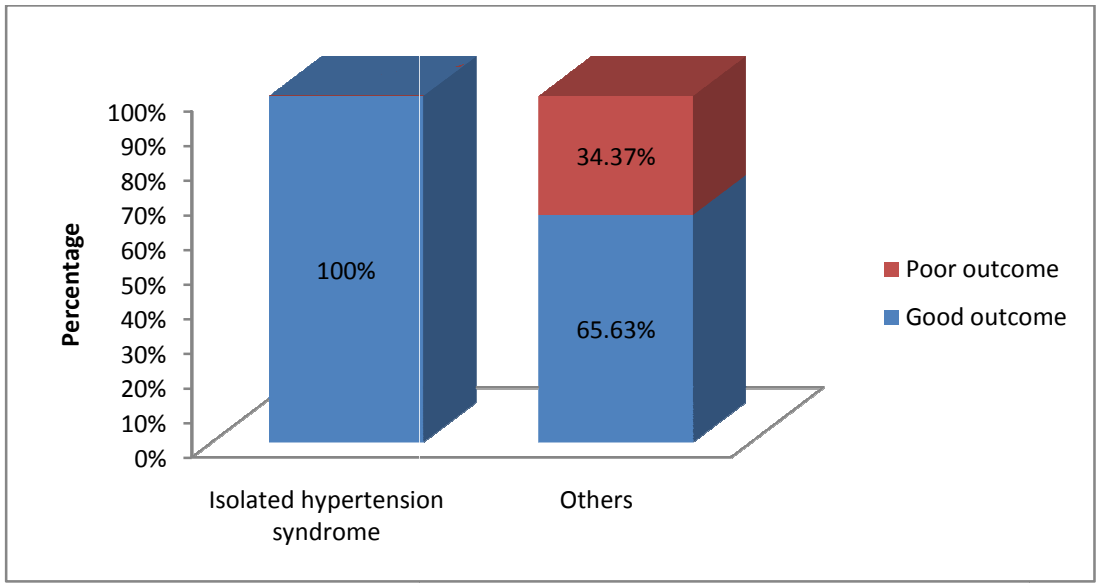
Presentation as Isolated intracranial hypertension syndrome was significantly associated with good outcome defined as mRS scale score of ≤2.

Table 17: Showing comparison of Isolated intracranial hypertension syndrome with good and poor outcome.

	Isolated intracranial hypertension syndrome(No. and Percentage of Patients)	Others(No. and Percentage of Patients)
Good outcome	13(100)	21(65.63)
Poor outcome	0	11(34.37)

By Fischer's test p=0.0195, considered significant.

Figure 19: Showing outcome in Isolated in Isolated intracranial hypertension syndrome.



DISCUSSION

The epidemiological factors, clinical presentation, etiological factors of CVT are highly variable. It is considered to be more common in South Asia and in the Middle East [9,10]. It is a disease of children and young adults [1,2,3].

In the largest clinical series, the International Study on Cerebral vein and dural sinus thrombosis (ISCVT) , the median age of patients with CVT was 37 years [3] and in a study conducted in Pakistan by Khealani et al [5], median age was 35 years compared to the median age in our study of 26 years. The mean age in our study was 29.29 years, lower than that reported by Khealani et al, Christo et al [81]in a Brazilian study and Narayan et al [18]in their study done in Hyderabad, India.

Hence a lower mean and median age was observed in our study compared to most other studies [5,18,81]. This could be because 64% of our patients were between the age range of 21-30 years of which 86% were females.

Hence the mean age of females (27.71years) in our study was lower than that observed by Khealani et al[5]. We also observed that the mean age of females in our study was lower than that of males (34.18years) similar to the observations of Khealani et al[5].

Though CVT can occur at any time from infancy to old age, most reported modern cases have been in adult women in association with puerperium[33]. It is more common in females than in males[3].

In our study, females formed the majority (75.56%) of the total 45 patients similar to that observed in most other studies [5,81,82] except that of Narayan et al[18] who observed a male predominance (53.7%).

ISCVT reported a female male ratio of 2.9:1 [3], a close approximate to our observation of 3.09:1. In contrary, Narayan et al observed a higher male female ratio of 1.17:1 [18].

The onset of symptoms was analysed as Acute (<48 hrs), Subacute (>48hrs to 30 days) or Chronic (>30 days) in our study [18]. According to literature, in ≥50% of the patients, the onset is subacute [40]. In our study, 53.33% of patients manifested subacutely, followed by a lesser percentage of 42.22% manifesting acutely and just (4.44%) of patients manifesting chronically similar to the observations of Narayan et al[18].

The median duration of presenting illness was 3 days in our study, lower than the observation of 7 days by Khealani et al [5] but very much closer to the observation of 4 days by ISCVT[3].

The mean duration of symptoms in our study it was 6 days as opposed to16.1 days observed by Narayan et al[18].

As for as the presenting symptoms are concerned, upto 90% of patients with CVT complain of headache which is the most frequent symptom and often the initial one[40]. By ISCVT, head ache was the only symptom in 9% of patients with CVT[40].

In our study headache was the most common symptom (77.78%) similar to the observations of most other studies [5,18,82,83].

Headache was observed as a sole symptom in 28.88% of our patients closer to that observed by Cristo et al [81] of about 33.3% whereas by ISCVT only 9% of patients manifested with sole headache [40].

Other common symptoms noted in our study were altered sensorium (40%), focal neurological deficit and seizures similar to other studies [5,82,83].

Next to headache, most common symptom observed in our study was altered sensorium, observed in about 40% of patients closer to 37% observed by Khealani et al[5].

Seizures are more frequent in CVT than in other stroke types [40]. About 30 to 40% of patients present with seizures, either focal or generalized or with status

epilepticus[58,59]. Seizures was observed in 33.33% of our patients however seizure incidence was slighlty higher in other studies [5,82,18] . Similar to other studies [5,81,82], generalized seizure was more commonly observed than focal seizure in our study and status epilepticus was observed in just 4.44% patients.

Focal neurological deficit, most commonly hemiplegia was observed in 35.56% of our patients closer to the observations of khealani et al[5] and Ashjazadeh et al[82].

Comparison of the common clinical presentations in our study with other studies is shown in the table below.

Symptoms	In our study	Khealani et al[5]	Ashjazadeh et al[82]	Dutch-European study[83]
Headache	77.78%	81%	93.54%	95%
Altered sensorium	40%	37%	25%	39%
Motor deficit	35.56%	45%	35.48%	43%
Seizures	33.33%	39%	36.4%	47%

Papilloedema occurs in about 50% of patients with CVT[24] .Most common clinical finding observed in our study was papilledema similar to other studies[82,83]. Other

common findings were hemiplegia and cranialnerve palsy. Most common nerve involved was the 6 th cranial nerve.

Coma was observed in 6.67% of our patients much lower than that reported in other studies[5,83] . Most of our patients presented with a combination of the three classical syndromes of presentation in CVT namely, Isolated intracranial hypertension syndrome, Focal syndrome, and Encephalopathy. However Isolated intracranial hypertension syndrome was observed in 28.88% of our patients compared to 18.2 % by Narayan et al [18]. Just 2 (4.44%) of our patients presented delirium.

Pregnancy and puerperium are considered an important risk factor related to CVT in women [3,32,45] . Studies from the west report <15% of their patients to be in the postpartum state [3,84]. Douglas et al [84,85] reported that frequency of peripartum CVT is 8.9 t0 11.6 cases per 100000 deliveries. ISCVT reported obstetric CVT in only 20% of cases compared to studies from Mexico and India which report a much higher frequency [3,6,32].

In our study 53.33% of patients were in postpartum state. Similar higher incidence of puerperal CVT was reported by the Pakistanian study [5].

Though peripartum CVT is especially common in less developed world countries with high pregnancy rates [46] there is a difference among different developing countries[5]. For example, Daif et al [29] from Saudi Arabia reported only 1 out of 40 patients being in postpartum state and a very lower incidence of peripartum CVT was also reported in the Iranian study by Ashjazadeh et al [82] and the study done in Hyderabad, India by Narayan et al [18].However in our study puerperium was the leading cause of CVT.

Douglas et al [84] reported by multivariate analysis that caesarean section and hypertension was significantly associated with peripartum and post partum CVT. The authors of the study suggest that the small increased risk with pregnancy induced hypertension might be due to a higher rate of caesarian section in women with hypertension and that there may be a cumulative effect of resistance to activated protein C during pregnancy together with decreased protein C levels following surgery [86,87] . However such an association of caesarean section or pregnancy induced hypertension was not seen in our study.

By literature, most common risk factor among women is use of oral contraceptive pills [40]. Various studies have demonstrated the increased risk of CVT in patients using oral contraceptives and thrombophilia, particularly in the presence of

prothrombin gene mutation, factor V Leiden mutation and hyperhomocystenemia. Use of oral contraceptive pill has been an important risk factor for CVT in the west, with a reported incidence of 40 to 45% [81]. The Iranian study by Ashjazadeh et al [82] also reported a higher incidence of OCP induced CVT among their patients, who used them for a prolonged duration to avoid menstruation during religious ceremonies like Ramadan fasting or Hajj .

Whereas in our study use of OCP was not reported even in a single case. Similar lower incidence was reported by other Pakistanian and Indian studies [5,18]. The reason could be due to lack of awareness of contraceptive methods among people visiting our hospital, who are mostly from rural areas in and around Thanjavur.

Infective causes of CVT are rare nowadays, being responsible for only 6 to 12 % of patients with CVT[3,42]. However in developing countries, systemic and nervous system infections may remain an important cause of CVT (18%) [5].

In our study, CVT secondary to meningitis was reported in 4.44% of cases in concordance with most of the recent studies [3,82] .Whereas in a Pakistanian study systemic and CNS infection was a leading risk factor, being observed in 18% of patients [5].

A prothrombotic condition was identified in 34% of patients in ISCVT cohort, being genetically determined in 22% patients [3]. Most common prothromotic conditions are prothrombin gene mutation, factor V leiden mutation and anticardiolipin /antiphospholipid antibody syndrome.Less common are protein C, protein S deficiency and antithrombin III deficiency. Christo et al [81] and wysokinska et al [88] reported the incidence of thrombophilia as 10 to 13%. However the studies from Iran and Pakistan reported an incidence of just 4 to 5 %[5,82].

Narayan et al reported 7.2% to be Anticardiolipin antibody positive and 12.3% as Protein C/S positive. In our study anticardiolipin antibody positivity was noted in 4.44% of patients.

Hyperhomocystenemia is a known risk factor for venous thrombosis. It results from low socioeconomic conditions secondary to deficient nutritional status, low plasma folate and vitamin B12 levels, which are associated with an increased risk of CVT in some developing countries [89,90].Hyperhomocystenemia was reported in 18.2% of cases by Narayan et al.[18] as opposed to 4.5% by ISCVT [3] and 9% by khealani et al [5]. In our study just 2.22 % of patients had hyperhomocystenemia.

Anemia and alcoholism were reported as major risk factors by Narayan et al [18].In our study anemia was seen in 20% of patients and 13.33% of patients were alcoholics but the causative nature of these risk factors were not proven in our study.

Imaging with CT is the most frequently done initial investigation in the diagnosis of CVT. Indirect signs are more common than direct signs which comprise direct visualization of thrombus.

In our study, indirect signs were more frequently observed. Haemorrhagic infarct was the most common finding being noted in 55.56% of patients similar to the observations of most of the studies[5,18]. Direct signs were observed in just 11.11% of our patients most commonly the dense delta sign suggesting superior sagittal sinus thrombosis.

The Superior sagittal sinus and the lateral sinuses are the most commonly affected sinuses in CVT, followed by the straight sinus and the cavernous sinus [36,37].

Most common sinus involved in our study was superior sagittal sinus (75.56%) in concordance with most of the studies in literature [18,24,33,82]. By contrast certain

studies [81,88]reported a higher incidence of transverse sinus involvement than SSS involvement.

Involvement of single sinus was seen in 62.22% of patients closer to 50% reported by khealani et al [5].

Involvement of ≥2 sinuses was seen in 37.78% of patients in our study as against 50% and 66% involvement by christo et al [81]and wysokinska et al [88] respectively.

By ISCVT [4] mortality occurred in 8.3% of their patients and most studies report a mortality rate of 10 to 20% [7,36,82]. In our study mortality rate was 11.11%.

Higher mortality than ISCVT could be due to tertiary care nature of our study centre.

According to Bousser2000 [7] and Girot et al 2007 [36], 10-20% of patients survive with persistent deficits after CVT. In our study morbidity rate was 17.78%, lower than that reported in most other studies [3,5,81] the reason for which is unclear.Total recovery was seen in 71.11% of our patients compared to 79% by ISCVT [3]and 71.2% by Narayan et al [18].

On assessing the predictors of good outcome (defined as mRS scale score of ≤2) we observed that patients who presented with isolated intracranial hypertension syndrome had good outcome (p=0.0195) similar to the data in literature [24].

In ISCVT, independent predictors of poor outcome as defined by death or disability at 6 months were age > 37 years, male gender, deep CVT, presence of motor deficit, CNS infections, malignancy, hemorrhage on admission in CT/MRI, GCS score on admission < 9 [36,3].

Predictors of mortality at 30 days in the ISCVT cohort were depressed consciousness, mental status disorders, deep CVT, right hemispheric hemorrhage and posterior fossa lesion [73].

Predictors of in hospital mortality in our study were age ≥35 years (p=0.0471), coma at the time of admission (p=0.0007) and GCS <9 (p= 0.0038) and predictors of poor outcome (defined as mRS scale score of >2) in our study were coma at the time of admission (p=0.0116) and motor deficit (p=0.0001) similar to observations made by Ashjazadeh et al [82], khealani et al [5] and ISCVT [3] except that the presence of haemorrhagic infarct in initial CT brain was not associated with poor outcome in our study as opposed to above mentioned studies [3,5,82].

SUMMARY

The summary of the observations made in our study are as follows,

According to our study,

1. CVT is more common in young females between the age group of 20 to 30 years.
2. CVT is 3 times more common in females than males.
3. Majority of patients present subacutely between 3 to 30 days, with a mean duration of 6 days.
4. Headache is the most common symptom and papilledema is the most common sign.
5. Headache may be the isolated symptom in 28% of patients.
6. Other common symptoms are altered sensorium, focal motor deficit, generalized seizures and delirium.
7. Presentation with isolated intracranial hypertension is more common than isolated focal syndrome and isolated encephalopathy.
8. Puerperium is the leading cause of CVT in our study with majority presenting within the first 2 weeks postpartum.

9. Oral contraceptive usage is a rare cause of CVT in our setting.

10. Most common finding in CT Brain is haemorrhagic infarct.

11. CT Brain can be normal in about 26 to 27 % of patients.

12. Subarachnoid haemorrhage can occur in as few as <5% of cases.

13. Superior sagittal sinus and right transverse sinus are the most common sinuses involved in CVT.

14. Isolated cortical vein thrombosis and deep vein thrombosis are rare.

15. Isolated intracranial hypertension syndrome is associated with good outcome.

16. The mortality rate of CVT is about 11% and the morbidity rate is about 17%.

17. Hence in general, the prognosis is good in CVT, if promptly diagnosed and treated, except when the patient is ≥35 years of age or presents with GCS score of <9 or is comatosed at presentation.

LIMITATION OF THE STUDY:

An important limitation of our study is small sample size. We could have got more impressive results if the sample size had been large.

CONCLUSION

Cerebral venous thrombosis, due to its wide spectrum of clinical presentation might be confused with other pathologies and hence the diagnosis may get easily missed or delayed. The clinical picture can vary from headache refractory to analgesics to coma. Since headache is the most common symptom, CVT should be suspected whenever a young adult presents with symptoms and signs of raised intracranial tension with or without other neurological symptoms. Since presentation with headache as a sole symptom is not uncommon, CVT should be a differential diagnosis of significant headache in young adults even in the absence of other signs and symptoms and examination of the fundus to rule out papilledema might serve as an important tool in arriving at a diagnosis when suspected.

CVT might be the underlying cause when a patient presents with diffuse encephalopathy, focal deficit, seizures, psychiatric symptoms or migraine. Hence CVT should be suspected when a young adult presents with stroke especially in the absence of vascular risk factors.

There is a definite variation in the risk factor profile of CVT from that of the west. Oral contraceptive use is not a major risk factor in our setting, whereas

peripartum CVT is the leading risk factor in our setting thus enforcing the importance of suspecting CVT in every peripartum female with neurological symptoms and also educating these females about the symptoms of the disease and the importance of reporting early to hospitals once symptoms appear.

CVT should always be suspected whenever imaging of the brain shows haemorrhagic infarct especially in non arterial territories. Since one may get misguided by a normal CT brain study, it is better to do more sensitive investigations like MRI and MRV whenever possible to confirm the diagnosis of CVT.

Though the outcome of CVT is in general good if promptly diagnosed and treated, the predictors of poor outcome and death may help us to provide extra vigilance in case of at risk patients.

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MASTER CHART

s.No	Name	Age(in years)	Sex	Acute onset	Subacute onset	Chronic onset	Duration of presenting illness	Headache	Altered sensorium	Seizure	Focal seizure	Generalised seizure	Status epilepticus	Motor deficit	Psyhiatric manifestations	Fever	Visual symptoms	Diabetes	Hypertension	Smoking	Alcoholism	Conscious	Drowsy	Stupor	Coma	Glascow coma scale
1	Subramani	45	M	N	Y	N	7	Y	Y	N	N	N	N	Y	N	N	N	Y	Y	Y	Y	N	N	Y	N	8\15
2	Gnanasekaran	30	M	N	Y	N	4	Y	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	15/15
3	Senthil kumar	33	M	N	Y	N	3	Y	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	15/15
4	Venkatesan	19	M	N	Y	N	10	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	N	15/15
5	Vijayalakshmi	40	F	Y	N	N	2	Y	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	5\15
6	Dhanalakshmi	21	F	Y	N	N	1	Y	N	Y	Y	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	15\15
7	Shantha	65	F	Y	N	N	1	N	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	8\15
8	Arokiamary	45	F	N	Y	N	4	Y	N	Y	Y	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	15/15
9	Panneer selvam	37	M	N	Y	N	15	Y	N	N	N	N	N	Y	N	N	Y	N	N	Y	N	Y	N	N	N	14/15
10	Dharmalingam	55	M	N	N	Y	32	Y	N	Y	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	15/15
11	Sudharani	24	F	N	Y	N	3	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	15/15
12	Vishwalingam	41	M	N	N	Y	33	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	15/15
13	Radhika	29	F	Y	N	N	2	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	15/15
14	Rajalakshmi	24	F	Y	N	N	1	N	Y	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	8\15
15	Mary	24	F	N	Y	N	15	Y	N	Y	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	14/15
16	Mariyammal	28	F	N	Y	N	7	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	15/15
17	Anbarasi	25	F	Y	N	N	1	N	Y	Y	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	15/15
18	Arokiamary	40	F	Y	N	N	1	N	N	Y	N	Y	N	Y	N	N	N	N	N	N	N	Y	N	N	N	15\15
19	Indumathy	26	F	Y	N	N	2	Y	Y	Y	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	14\15
20	Punitha	30	F	N	Y	N	3	Y	N	Y	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	14\15
21	Mahendran	36	M	Y	N	N	2	N	Y	N	N	N	N	Y	N	N	N	N	N	N	Y	N	N	N	Y	7\15
22	Sudha	22	F	Y	N	N	2	Y	N	Y	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	14\15
23	Uma	24	F	Y	N	N	1	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	14\15

s.No	Name	Age(in years)	Sex	Acute onset	Subacute onset	Chronic onset	Duration of presenting illness	Headache	Altered sensorium	Seizure	Focal seizure	Generalised seizure	Status epilepticus	Motor deficit	Psyhiatric manifestations	Fever	Visual symptoms	Diabetes	Hypertension	Smoking	Alcoholism	Conscious	Drowsy	Stupor	Coma	Glascow coma scale
24	Hema	27	F	Y	N	N	2	Y	Y	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	Y	N	8\15
25	Raja	30	M	N	Y	N	14	Y	N	N	N	N	N	N	N	N	Y	N	N	Y	Y	Y	N	N	N	15\15
26	Devi	26	F	Y	N	N	2	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	14\15
27	Khajabeevi	17	F	Y	N	N	1	Y	Y	Y	N	Y	Y	N	N	Y	N	N	N	N	N	N	Y	N	N	13/15
28	Jaya	26	F	N	Y	N	7	Y	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	8\15
29	Annakili	28	F	Y	N	N	1	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	15/15
30	Suganthi	24	F	N	Y	N	5	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	15/15
31	Vishalam	20	F	N	Y	N	3	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	Y	N	N	N	15\15
32	Rajeshwari	35	F	N	Y	N	10	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	15/15
33	Chithra	24	F	N	Y	N	6	Y	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	14\15
34	Sakunthala	20	F	N	Y	N	10	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	15/15
35	Rajesh	21	M	N	Y	N	7	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N	N	15/15
36	Vijaya	27	F	Y	N	N	1	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	15/15
37	Radha	22	F	N	Y	N	10	N	N	Y	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	15\15
38	Lakshmi	24	F	N	Y	N	4	Y	Y	N	n	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	8\15
39	Kanmani	32	F	N	Y	N	12	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	15/15
40	Devaki	26	F	Y	N	N	2	Y	Y	Y	N	Y	N	Y	N	Y	N	N	N	N	N	N	N	N	Y	7\15
41	Maheshwari	26	F	N	N	N	7	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	15/15
42	Muruganand	29	M	N	Y	N	10	Y	N	N	N	N	N	N	Y	N	N	N	N	Y	Y	N	Y	N	N	15\15
43	Farida banu	22	F	Y	N	N	2	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	14\15
44	Latha	23	F	Y	N	N	1	N	N	Y	N	Y	N	Y	N	N	N	N	N	N	N	N	N	Y	N	8\15
45	Kumari	26	F	N	Y	N	5	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	15/15

MASTER CHART

s.No	Name	Age(in years)	Sex	Speech disturbances	Cranial nerve palsy	Type of cranial nerve palsy	Motor deficit	Monoplegia	Hemiplegia	Quadriplegia	Papilledema	Secondary optic atrophy	Meningeal signs	Isolated intracranial hypertens	Puerperium	No. of days Postpartum	Post LSCS	Gravida	Hyperhomocysteinemia	Polycythemia vera	CTD	APLA	Migraine	Thyroid diseases	Recent surgery	Pallor
1	Subramani	45	M	N	N		Y	N	Y	N	Y	N	N	N	N		N		N	N	N	N	N	N	N	N
2	Gnanasekaran	30	M	N	N		N	N	N	N	Y	N	N	Y	N		N		N	N	N	N	N	N	N	N
3	Senthil kumar	33	M	N	N		N	N	N	N	Y	N	N	Y	N		N		N	N	N	N	N	N	N	N
4	Venkatesan	19	M	N	Y	6	N	N	N	N	Y	N	N	Y	N		N		N	N	N	N	N	N	N	N
5	Vijayalakshmi	40	F	N	Y	3	Y	N	N	Y	Y	N	N	N	N		N		N	N	N	N	N	N	N	N
6	Dhanalakshmi	21	F	N	N		Y	N	Y	N	Y	N	N	N	y	8	N	1	N	N	N	N	Y	N	N	Y
7	Shantha	65	F	N	Y	7	Y	N	Y	N	Y	N	N	N	N		N		N	N	N	N	N	N	Y	N
8	Arokiamary	45	F	N	N		Y	N	Y	N	N	N	N	N	N		N		N	N	N	N	N	N	N	Y
9	Panneer selvam	37	M	Y	Y	2	Y	N	Y	N	N	Y	N	N	N		N		N	N	N	N	N	N	N	N
10	Dharmalingam	55	M	N	N		N	N	N	N	Y	N	N	Y	N		N		N	N	N	N	N	N	N	Y
11	Sudharani	24	F	N	N		N	N	N	N	Y	N	N	Y	Y	40	N	1	N	N	N	N	N	N	N	N
12	Vishwalingam	41	M	N	Y	5,8	N	N	N	N	Y	N	N	Y	N		N		N	N	N	N	N	N	N	N
13	Radhika	29	F	N	N		Y	N	Y	N	N	N	N	N	Y	32	N	2	N	N	N	N	N	N	N	N
14	Rajalakshmi	24	F	N	N		N	N	N	N	N	N	N	N	Y	1	Y	2	N	N	N	N	N	N	N	N
15	Mary	24	F	N	N		N	N	N	N	N	N	N	N	Y	14	Y	2	N	N	N	N	Y	N	N	Y
16	Mariyammal	28	F	N	N		N	N	N	N	Y	N	N	N	Y	7	Y	3	N	N	N	N	N	N	N	Y
17	Anbarasi	25	F	N	N		N	N	N	N	N	N	N	N	Y	8	N	2	N	N	N	N	N	N	N	N
18	Arokiamary	40	F	N	N		Y	N	Y	N	N	N	N	N	N		N		N	N	N	N	N	N	N	N
19	Indumathy	26	F	N	N		N	N	N	N	N	N	N	N	Y	12	N	1	N	N	N	N	N	N	N	N
20	Punitha	30	F	N	N		N	N	N	N	N	N	N	N	Y	6	Y	3	N	N	N	N	N	N	N	N
21	Mahendran	36	M	N	N		Y	N	Y	N	N	N	N	N	N		N		N	N	N	N	N	N	N	N
22	Sudha	22	F	N	N		N	N	N	N	N	N	N	N	Y	6	N	1	N	N	N	N	N	N	N	Y
23	Uma	24	F	N	N		N	N	N	N	Y	N	N	N	Y	10	N	1	N	N	N	N	N	N	N	N

s.No	Name	Age(in years)	Sex	Speech disturbances	Cranial nerve palsy	Type of cranial nerve palsy	Motor deficit	Monoplegia	Hemiplegia	Quadriplegia	Papilledema	Secondary optic atrophy	Meningeal signs	Isolated intracranial hypertens	Puerperium	No. of days Postpartum	Post LSCS	Gravida	Hyperhomocysteinemia	Polycythemia vera	CTD	APLA	Migraine	Thyroid diseases	Recent surgery	Pallor
24	Hema	27	F	N	N		N	N	N	N	Y	N	N	N	Y	20	N	2	N	N	N	N	N	N	N	N
25	Raja	30	M	N	Y	6	N	N	N	N	Y	N	N	Y	N		N		N	N	N	N	N	N	N	N
26	Devi	26	F	N	N		N	N	N	N	N	N	N	N	N		N		N	N	N	N	N	N	N	N
27	Khajabeevi	17	F	N	N		N	N	N	N	N	N	N	N	N		N		N	N	Y	N	N	Y	N	Y
28	Jaya	26	F	N	N		Y	Y	N	N	Y	N	Y	N	Y	20	Y	3	N	N	N	N	N	N	N	N
29	Annakili	28	F	N	N		Y	N	Y	N	N	N	N	N	Y	6	N	3	N	N	N	N	N	N	N	Y
30	Suganthi	24	F	N	N		N	N	N	N	N	N	N	Y	Y	7	N	1	N	N	N	N	N	N	N	N
31	Vishalam	20	F	N	N		N	N	N	N	N	N	N	N	Y	15	Y	1	N	N	N	N	N	N	N	Y
32	Rajeshwari	35	F	N	N		N	N	N	N	N	N	N	Y	Y	10	N	2	N	N	N	N	N	N	N	N
33	Chithra	24	F	N	N		Y	Y	N	N	Y	N	N	N	Y	8	N	3	N	N	N	N	N	N	N	N
34	Sakunthala	20	F	N	N		N	N	N	N	N	N	Y	Y	N		N		N	N	N	N	N	N	N	N
35	Rajesh	21	M	N	Y	6	N	N	N	N	Y	N	N	Y	N		N		Y	N	N	N	N	N	N	N
36	Vijaya	27	F	N	N		Y	N	Y	N	N	N	N	N	Y	8	N	2	N	N	N	N	N	N	N	Y
37	Radha	22	F	N	N		N	N	N	N	N	N	N	N	Y	10	N	1	N	N	N	N	N	N	N	N
38	Lakshmi	24	F	N	N		Y	Y	N	N	Y	N	N	N	Y	8	N	3	N	N	N	N	N	N	N	N
39	Kanmani	32	F	N	N		N	N	N	N	N	N	N	N	Y	6	N	2	N	N	N	N	N	N	N	N
40	Devaki	26	F	N	N		Y	N	N	Y	Y	N	N	N	Y	12	N	2	N	N	N	N	N	N	N	N
41	Maheshwari	26	F	N	N		N	N	N	N	Y	N	N	Y	N		N		N	N	N	N	N	N	N	N
42	Muruganand	29	M	N	N		N	N	N	N	Y	N	N	N	N		N		N	Y	N	N	N	N	N	N
43	Farida banu	22	F	N	Y	6	N	N	N	N	Y	N	N	N	N		N		N	N	N	Y	N	N	N	N
44	Latha	23	F	N	N		Y	N	Y	N	Y	N	N	N	N		N		N	N	N	N	N	N	N	N
45	Kumari	26	F	N	N		N	N	N	N	Y	N	N	Y	Y	10	N	2	N	N	N	N	N	N	N	N

MASTER CHART

s.No	Name	Age(in years)	Sex	Dehydration	Normal study CT brain	Nonhaemorrhagic infarct CT b	Haemorrhagic infarct CT brain	Direct signs CT brain	Subarachnoid haemorrhage	Superior sagittal sinus	Transverse sinus	Side of transverse sinus	Straight sinus	Sigmoid sinus	IJV extension	Cortical veins	Bilateral disease	Multiple sinuses(≥2)	>2 sinuses	Death	Residual deficit	Poor outcome
1	Subramani	45	M	N	N	Y	N	N	N	Y	N		N	N	N	N	N	N	N	N	Y	Y
2	Gnanasekaran	30	M	N	N	N	Y	Y	N	Y	Y	R	N	N	N	N	N	Y	N	N	N	N
3	Senthil kumar	33	M	N	Y	N	N	N	N	Y	Y	R	N	N	N	N	N	Y	N	N	N	N
4	Venkatesan	19	M	N	Y	N	N	N	N	N	Y	R	N	Y	Y	N	N	Y	N	N	N	N
5	Vijayalakshmi	40	F	N	N	N	Y	N	N	Y	Y	L	N	N	N	N	N	Y	N	Y	N	Y
6	Dhanalakshmi	21	F	Y	N	N	Y	N	N	Y	N		Y	N	N	Y	Y	N	N	N	Y	Y
7	Shantha	65	F	Y	N	N	Y	N	N	N	Y	L	N	Y	Y	Y	Y	Y	N	Y	N	Y
8	Arokiamary	45	F	N	N	Y	N	Y	N	Y	N		N	N	N	N	N	N	N	N	N	N
9	Panneer selvam	37	M	Y	N	Y	N	N	Y	Y	Y	L	N	Y	N	N	N	Y	Y	N	N	Y
10	Dharmalingam	55	M	N	Y	N	N	N	N	Y	Y	R	N	N	N	N	N	Y	N	N	N	N
11	Sudharani	24	F	N	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
12	Vishwalingam	41	M	N	Y	N	N	N	N	N	N		N	Y	N	N	N	N	N	N	Y	N
13	Radhika	29	F	N	N	Y	N	N	N	N	N		N	Y	N	N	N	N	N	N	N	Y
14	Rajalakshmi	24	F	N	Y	N	N	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
15	Mary	24	F	Y	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
16	Mariyammal	28	F	N	N	N	Y	N	N	Y	Y	R	N	N	N	N	N	Y	N	N	N	N
17	Anbarasi	25	F	N	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
18	Arokiamary	40	F	N	Y	N	N	Y	N	Y	Y	R	N	N	N	N	N	Y	N	N	N	N
19	Indumathy	26	F	N	N	N	Y	N	N	Y	N		N	N	N	Y	N	N	N	N	N	N
20	Punitha	30	F	N	N	N	Y	N	N	Y	N		N	N	N	Y	N	N	N	N	N	N
21	Mahendran	36	M	Y	N	N	Y	N	N	Y	Y	B/L	Y	Y	Y	N	Y	Y	Y	Y	N	Y
22	Sudha	22	F	N	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
23	Uma	24	F	Y	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	N	N

s.No	Name	Age(in years)	Sex	Dehydration	Normal study CT brain	Nonhaemorrhagic infarct CT b	Haemorrhagic infarct CT brain	Direct signs CT brain	Subarachnoid haemorrhage	Superior sagittal sinus	Transverse sinus	Side of transverse sinus	Straight sinus	Sigmoid sinus	IJV extension	Cortical veins	Bilateral disease	Multiple sinuses(≥2)	>2 sinuses	Death	Residual deficit	Poor outcome
24	Hema	27	F	Y	Y	N	N	N	N	Y	N		N	N	N	N	N	N	N	Y	N	Y
25	Raja	30	M	Y	N	Y	N	N	N	Y	Y	R	N	N	N	N	N	N	N	N	N	N
26	Devi	26	F	N	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
27	Khajabeevi	17	F	Y	N	Y	N	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
28	Jaya	26	F	N	N	N	Y	Y	N	N	Y	L	N	N	N	N	N	N	N	N	Y	N
29	Annakili	28	F	N	Y	N	N	N	N	N	Y	L	N	N	N	Y	N	N	N	N	Y	Y
30	Suganthi	24	F	N	N	N	Y	N	Y	Y	N		Y	N	N	N	N	Y	Y	N	N	N
31	Vishalam	20	F	N	N	N	Y	N	N	N	Y	L	N	N	N	N	N	N	N	N	N	N
32	Rajeshwari	35	F	N	y	N	N	N	N	N	Y	R	N	Y	Y	N	N	Y	N	N	N	N
33	Chithra	24	F	N	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	Y	N
34	Sakunthala	20	F	N	Y	N	N	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
35	Rajesh	21	M	N	N	Y	N	N	N	N	Y	L	N	Y	Y	N	N	Y	N	N	N	N
36	Vijaya	27	F	N	Y	N	N	N	N	N	Y		N	N	N	Y	N	N	N	N	Y	Y
37	Radha	22	F	N	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	N	N
38	Lakshmi	24	F	N	N	N	Y	N	N	Y	N		N	N	N	N	N	N	N	N	Y	N
39	Kanmani	32	F	N	N	N	Y	N	N	N	Y	R	N	Y	Y	N	N	Y	N	N	N	N
40	Devaki	26	F	Y	N	N	Y	Y	N	Y	Y	B/L	N	N	Y	Y	Y	Y	N	Y	N	Y
41	Maheshwari	26	F	N	N	N	Y	N	N	y	Y	R	N	N	N	N	N	Y	Y	N	N	N
42	Muruganand	29	M	Y	N	Y	N	N	N	y	N		N	N	N	N	N	N	N	N	N	N
43	Farida banu	22	F	N	Y	N	N	N	N	y	N		N	N	N	N	N	N	N	N	N	N
44	Latha	23	F	Y	N	N	Y	N	N	Y	N		n	n	N	N	N	N	N	N	N	N
45	Kumari	26	F	N	N	N	Y	N	N	Y	Y	R	n	n	N	Y	N	Y	Y	N	N	N

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA-613004

(Affiliated to the T.N Dr.MGR Medical University, Chennai)

ETHICAL COMMITTEE

CERTIFICATE

Name of the Candidate : Dr.R.DEVAN

Course : M.D. (GENERAL MEDICINE)

Period of Study : NOVEMBER 2010 – NOVEMBER 2012

College : THANJAVUR MEDICAL COLLEGE

Dissertation Topic : STUDY ON CLINICAL PROFILE OF CEREBRAL VENOUS THROMBOSIS .

The Ethical committee, Thanjavur Medical College has decided to inform that your Dissertation Topic is accepted and you are permitted to proceed with the above study.

Thanjavur

Date :

THANJAVUR MEDICAL COLLEGE * THANJAVUR-4 *

Secretary

Ethical Committee

KEY TO PROFORMA

CT- Computed Tomography

MRI- Magnetic Resonance Imaging

MRA- Magnetic Resonance Angiography

MRV- Magnetic Resonance Venography

mRS - modified RANKIN SCALE

KEY TO MASTER CHART

M- Male

F- Female

N- No

Y- Yes

R- Right

L- Left

B/L- Bilateral

CTD- Connective Tissue Disorder

APLA- Antiphospholipid Antibodies

CT- Computed Tomography

IJV- Internal Jugular Vein

LSCS- Lower Segment Caesarean Section

PROFORMA

CLINICAL PROFILE OF CEREBRAL VENOUS THROMBOSIS

NAME : AGE : SEX:

OCCUPATION : INCOME: In patient No.:

DATE OF ADMISSION : DATE OF DISCHARGE :

HISTORY:

NEUROLOGICAL SYMPTOMS:

	Yes /No	**Duration**
Head Ache		
Type:		
Loss of Consciousness:		
Altered Sensorium :		
Convulsions :		
Number of Episodes :		
Duration:		
Focal:		
Generalised:		
Status Epilepticus:		
Post - Ictal state:		

Limb weakness :	**Upper limb**	**Lower limb**
	Right	
	Left	

Speech Disturbance:

Neuropsychiatric manifestations:

Visual symptoms:

h/s/o cranial nerve involvement:

h/o sensory disturbances:

h/o bladder/ bowel disturbances:

h/o ataxia:

h/s/o autonomic dysfunction:

ONSET OF SYMPTOMS: **Yes/No**

ACUTE (<2 days):

SUBACUTE (3 days to 30 days):

CHRONIC (>30 days):

NON- NEUROLOGICAL SYMPTOMS:

Yes /No

- ❖ Fever
- ❖ Nasal discharge
- ❖ Ear Ache / Ear Discharge
- ❖ Diarrhea/ vomiting

SYMPTOMS SUGGESTIVE OF EIOLOGY:

RHEMATOLOGICAL SYMPTOMS

Yes /No

- ❖ Arthralgia
- ❖ Oral ulcer
- ❖ Skin rash
- ❖ Alopecia
- ❖ Photosensitivity
- ❖ Raynaud's Phenomenon
- ❖ Others (Specify)

HISTORY SUGGESTIVE OF THROMBOPHILIA Yes/No

H/O prior Deep Vein Thrombosis/ Pulmonary embolism

H/O prior Cerebral Venous Thrombosis:

H/O recurrent abortion (in females):

DRUG HISTORY Yes/No

- Oral Contraceptive Pill:
- Anticoagulants:
- Others:

MENSTURAL HISTORY :

OBSTETRIC HISTORY

- GRAVIDA :
- PARA:
- History of Pregnancy induced hypertension / Eclampsia :
- Normal vaginal delivery :
- Lower Segment Caesarean Section:
- Time of onset of symptoms since delivery :

PAST HISTORY : Yes/No

- Diabetes mellitus :
- Systemic hypertension :
- Coronary artery disease:
- Valvular Heart Disease:
- Tuberculosis:
- Seizure Disorder :
- Migraine :

- Thyroid Disorder (hyper / hypo):
- Others (Specify):
- h/o recent surgery:

FAMILY HISTORY **Yes/No**

- Diabetes mellitus :
- Systemic hypertension:
- Tuberculosis:
- Connective tissue disorder:
- h/o Deep Vein Thrombosis:
- h/o Cerebral Venous Thrombosis:

PERSONAL HISTORY **Yes/No**

- Smoking
- Alcohol
- Drug addictions / abuse

EXAMINATION:

GENERAL **Yes/No**

Febrile

Pallor

Icterus

Cyanosis

Clubbing

Edema

Lymphadenopathy

Sign of dehydration

Pulse rate:

Blood Pressure:

Signs suggestive of Connective Tissue Diseases (if any):

CENTRAL NERVOUS SYSTEM EXAMINATION

Higher functions

Consciousness :

Normal :

Drowsy :

Stupor :

Coma :

Glasgow Coma Scale :

Orientation :

Speech :

Memory :

Other higher function Abnormality (if any):

FUNDUS :

CRANIAL NERVES	Yes/No
I	
II	
III	
IV	
VI	
V	
VII	
VIII	
IX	
X	
XI	
XII	

SPINOMOTOR SYSTEM **Right** **Left**

Bulk

Tone - Upper Limb

- Lower Limb

Power - Upper Limb

- Lower Limb

Reflexes

Superficial Reflexes : Corneal

Conjunctival

Abdomen

Plantar

Deep tendon Reflexes

	Right	**Left**
Upper Limb		
Lower Limb		

Gait:

Sensory system:

	Yes/No
Cerebellar involvement :	
Involuntary movements:	
Autonomic involvement :	
Meningeal signs:	

OTHER SYSTEMS	**Yes/No**
Cardiovascular system:	
Respiratory system:	
Abdomen:	

DIAGNOSIS :

INVESTIGATIONS

Complete blood count:

Random blood sugar:

Renal function test:

Electrolytes:

Lipid profile:

Chest X-ray:

Electrocardiogram:

Echocardiogram:

Ultrasound abdomen:

Doppler lower limb:

Veneral Disease Research Laboratory:

ELISA for Human Immunodeficiency Virus:

Bleeding Time:

Clotting Time:

Activated partial thromboplastin time:

Prothrombin Time:

International normalised ratio:

OTHERS (as required) :

CT- BRAIN: **Yes/No**

Hemorrhagic infarct:

Non hemorrhagic infarct:

Cerebral edema:

Enhancement of falx and tentorium:

Sub-arachnoid hemorrhage:

Cord sign:

Dense triangle sign:

Empty delta sign:

MRI brain WITH MRA & MRV:

	Yes/No
Superior sagittal sinus:	
Transverse sinus: Right/Left	
Straight sinus:	
Sigmoid sinus:	
Deep cerebral veins thrombosis:	
Internal Jugular Vein extension:	
Cortical veins thrombosis:	
Unilateral / Bilateral disease:	

OUTCOME:	**Yes/No**
Death:	
Disability:	
mRS score:	

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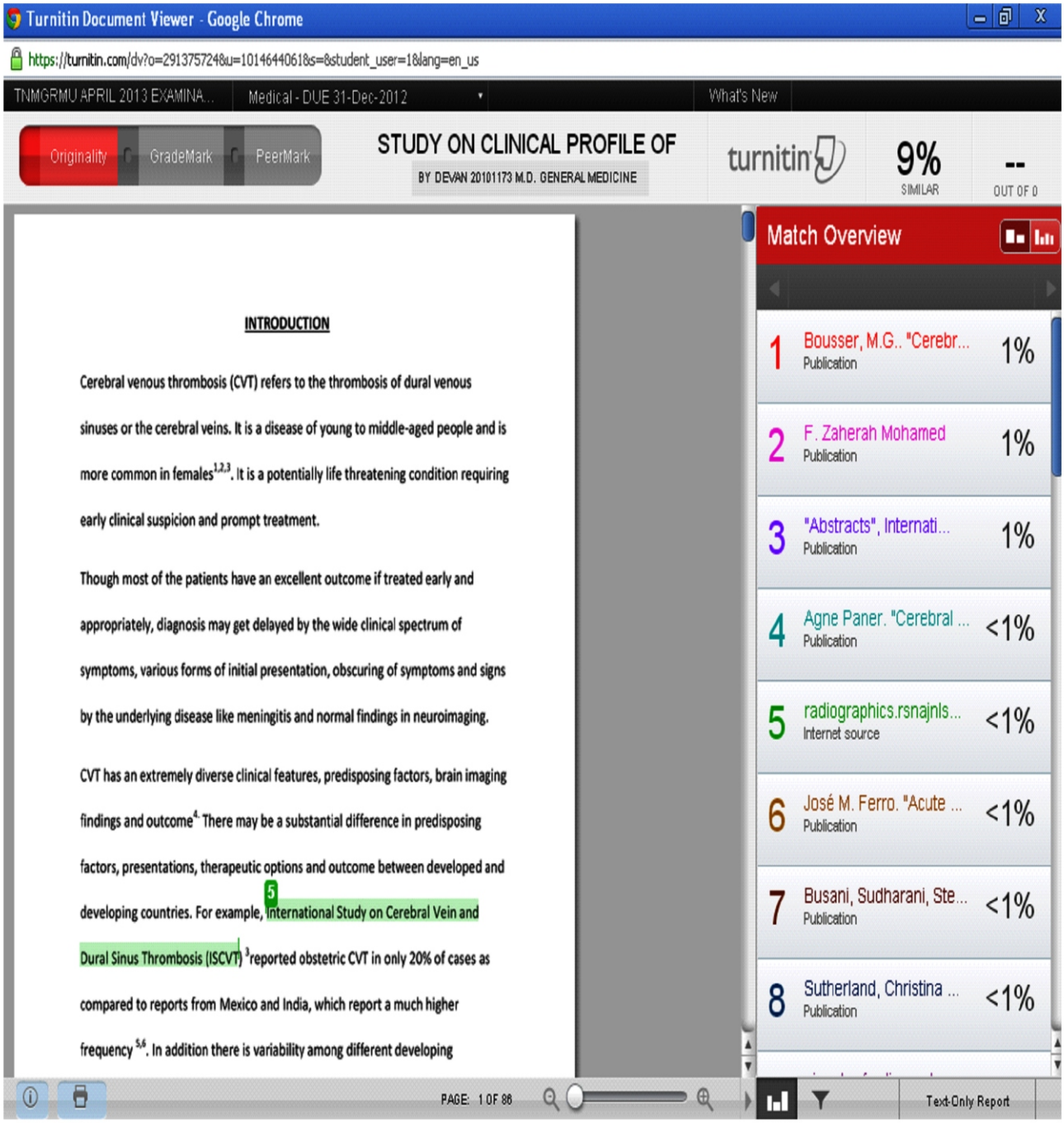
INTRODUCTION Cerebral venous thrombosis (CVT) refers to the thrombosis of dural venous sinuses or the cerebral veins. It is a disease of young to middle-aged people and is more common in females 1,2,3 . It is a potentially life threatening condition requiring early clinical suspicion and prompt treatment. Though most of the patients have an excellent outcome if treated early and appropriately, diagnosis may get delayed by the wide clinical spectrum of symptoms, various forms of initial presentation, obscuring of symptoms and signs by the underlying disease like meningitis and normal findings in neuroimaging. CVT has an extremely diverse clinical features, predisposing factors, brain imaging...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INTRODUCTION
Cerebral venous thrombosis (CVT) refers to the thrombosis of dural venous sinuses or the cerebral veins. It is a disease of young to middle-aged people and is more common in females[1,2,3]. It is a potentially life threatening condition requiring early clinical suspicion and prompt treatment.
Though most of the patients have an excellent outcome if treated early and appropriately, diagnosis may get delayed by the wide clinical spectrum of symptoms, various forms of initial presentation, obscuring of symptoms and signs by the underlying disease like meningitis and normal findings in neuroimaging.
CVT has an extremely diverse clinical features, predisposing factors, brain imaging findings and outcome[4]. There may be a substantial difference in predisposing factors, presentations, therapeutic options and outcome between developed and developing countries. For example, International Study on Cerebral Vein and Dural Sinus Thrombosis (ISCVT) [5] reported obstetric CVT in only 20% of cases as compared to reports from Mexico and India, which report a much higher frequency [5,6]. In addition there is variability among different developing
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