# *Dissertation on*

## THE CLINICORADIOLOGICAL PROFILE OF CEREBRAL VENOUS SINUS THROMBOSIS WITH PROGNOSTIC EVALUATION USING MR SEQUENCES

*Submitted in partial fulfilment of requirements for*

**M.D.DEGREE BRANCH VIII RADIODIAGNOSIS**

*of*

**The Tamil Nadu Dr.M.G.R. Medical University Chennai.**


**BARNARD INSTITUTE OF RADIOLOGY**
**MADRAS MEDICAL COLLEGE & RESEARCH INSTITUTE**
**CHENNAI - 600 003**

**SEPTEMBER - 2006**

# CERTIFICATE

This is to certify that this dissertation entitled **"THE CLINICORADIOLOGICAL PROFILE OF CEREBRAL VENOUS SINUS THROMBOSIS WITH PROGNOSTIC EVALUATION USING MR SEQUENCES"** submitted by **Dr.G.SATHYAN,** appearing for Part II M.D. Branch VIII - Radiodiagnosis degree examination in September 2006 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfilment of regulations of The Tamil Nadu Dr.M.G.R. Medical University, Chennai. I forward this to The Tamil Nadu Dr.M.G.R. Medical University, Chennai.

**Dr.T.S. SWAMINATHAN, M.D., DMRD,FICR**
Director,
Barnard Institute of Radiology,
Madras Medical College,
Govt. General Hospital,
Chennai.

**Dr.KALAVATHY PONNIRAIVAN, M.D.,**
Dean
Madras Medical College,
Govt. General Hospital,
Chennai.

# DECLARATION

I solemnly declare that the dissertation entitled **"THE CLINICORADIOLOGICAL PROFILE OF CEREBRAL VENOUS SINUS THROMBOSIS WITH PROGNOSTIC EVALUATION USING MR SEQUENCES"** is done by me at The Madras Medical College and Hospital, during 2004 - 2006 under the guidance and supervision of **Prof.T.S. Swaminathan. M.D., D.M.R.D, F.I.C.R** This dissertation is submitted to The Tamil Nadu Dr.M.G.R. Medical University towards the partial fulfilment of requirements for the award of **M.D. DEGREE (BRANCH VIII) RADIODIAGNOSIS**

Place :

Date :

**Dr.G.SATHYAN**
M.D.RADIODIAGNOSIS
(Postgraduate)
Barnard Institute of Radiology
Madras Medical College, Chennai

# ACKNOWLEDGEMENT

I would like to thank **Prof.Kalavathy Ponniraivan, M.D.,** Dean, Madras Medical College for giving me permission to conduct the study in this institution.

I would like to express my sincere and heartfelt gratitude to my teacher and guide **Prof.T.S.Swaminathan, M.D., D.M.R.D., F.I.C.R.,** Director, Barnard Institute of Radiology, Madras Medical College for having encouraged me to take up this study. But for his guiding spirit, perseverance and wisdom this study would not have been possible.

I wish to thank my H.O.D and other professors, **Prof.V.Chandrasekar, M.D.,D.M.R.D.,** H.O.D., Barnard Institute of Radiology, Madras Medical College, **Prof.P.Kuppuswamy, M.D., D.M.R.D., Prof. N.Kulasekaran, M.D., D.M.R.D, Prof. A.P. Annadurai, M.D., D.M.R.D.** for their support, valuable criticisms and encouragement.

I am greatly indebted to my assistant professors **Dr.Umapathy D.M.R.D., Dr.S.Sundareswaran, D.M.R.D., Dr.Nesam Manivannan D.M.R.D., Dr.Ravi M.D.** and **Dr.Babupeter M.D.,** for their valuable suggestions and support.

I wish to thank all my fellow post graduates for their untiring help and encouragement.

Last but not the least; I thank all my patients for their co-operation, without whom, this study would not have been possible.

# CONTENTS

# INTRODUCTION

Acute stroke is one of the major cause of morbidity and mortality. It can be arterial or venous in origin. Venous infarction, compared to arterial stroke, is less common, potentially treatable and so has a good prognosis.

Cerebral venous thrombosis is not an uncommon disease. The presenting signs and symptoms of cerebral venous sinus thrombosis is so vague and diverse that it is referred to as `The Great Masquerador of diseases'.

Without CT or MRI many cases would be missed since the clinician is unsuspected of this disease. With the widespread availability of CT and MRI, and the awareness of this pathology among general physicians and obstetricians, the rate of diagnosis of this disease has increased manifold during the last two decades.

Many a times cerebral venous thrombosis is unsuspected by the treating clinician that the disease is first diagnosed by the radiologists.

It is important on the part of the radiologist to promptly diagnose cerebral venous thrombosis since with early treatment the pathology is almost completely reversible and so has a very good prognosis.

Catheter angiography is the gold standard for diagnosing venous thrombosis. But the preferred modalities are CT and MRI since they are noninvasive, cheap and with little risk to the patient.

Conventional CT, with and without contrast, is a good first investigation for patients with any CNS signs and symptoms. More than 80%

of Cerebral venous thrombosis can be diagnosed with this. It is also useful in ruling out intracranial tumors and infections.

MRI and MRV are more sensitive than CT in diagnosing cerebral venous thrombosis. It can also map the extent of thrombosis and parenchymal lesions. With certain criteria MRI can also give a prognostic valuation of the disease.

# CEREBRAL VENOUS SINUS THROMBOSIS - A SYNOPSIS

## ANATOMY

The cerebral venous system consists of

(i) Superficial cortical veins.

(ii) Deep medullary and subependymal veins

(iii) Dural venous sinuses

### Dural Venous Sinuses

The dural venous sinuses are interlinked large venous channels of the brain. They drain the veins of brain through superficial cortical veins and deep veins. They ultimately drain via the internal jugular veins. The sinuses are endothelium lined venous channels formed between the superficial and deep layers of the duramater **(*Gray's Anatomy*)**.

The superior sagittal sinus is an unpaired, midline, sagittaly oriented sinus. It lies at the base of falx cerebri. It drains the cortical veins of cerebral convexity. It extends from crista galli anteriorly to the venous confluence posteriorly, where it joins the straight sinus and the two lateral sinuses.

The lateral sinuses are paired sinuses one on either side. It consists of two parts, the transverse and sigmoid sinuses. The transverse sinus lies at the base of tentorium from the venous confluence upto the sigmoid sinus.

The sigmoid sinus lies at the posterolateral wall of the petrous temporal bone, continuous with the transverse sinus and exiting the jugular foramen as internal jugular vein.

The inferior sagittal sinus lies at the free margin of falx cerebri. It drains into the straight sinus. Since it is thin, it in inconsistently imaged.

The Great cerebral vein of Galen is formed by the basal veins of Rosenthal and the internal cerebral veins. It lies below the splenium of corpus callosum. Along with inferior sagittal sinus it forms the straight sinus.

The straight sinus lies at the confluence of falx cerebri and tentorium cerebelli. It courses posteroinferiorly and reaches the confluence of sinuses.

The cavernous sinuses are paired sinuses on either side of pituitary fossa. It is multiseptated and interconnected venous sinus. It receives the superior and inferior ophthalmic veins. It is closely related to ICA which courses within it. It is also associated with cranial nerves, III, IV, V1, V2 and VI which lie along its wall.

The superior and inferior petrosal sinuses are small inconsistently imaged sinuses.

**Superficial Cortical Veins**

The superficial cortical veins lie on the convexity of cerebral hemispheres. They are very variable in position and distribution. Most of them run upward and join the Superior sagittal sinus.

Much of them are unnamed except.

(i) Vein of Labbe which runs from the sylvian fissure to the transverse sinus.

(ii) Vein of Trollard which runs from the sylvian fissure to the superior sagittal sinus.

(iii) Superficial middle cerebral vein, which runs along the sylvian fissure.

In most angiograms one or other of these veins are visualized not all.

**Deep cerebral veins**

The deep cerebral veins consist of

(i) Medullary veins in the depth of cerebral hemispheres.

(ii) Subependymal veins lying near the lateral ventricles receiving the medullary veins.

(iii) Basal veins draining the temporal lobes which arises above the sella courses posteriorly round the midbrain

(iv) Internal cerebral veins formed by thalamostriate and septal veins. They lie one on either side of midline. They commence just behind the foramen of Monro pass in the roof of 3rd ventricle lying in the telachoroidea.

**Normal Anatomic variations**

Like the variations of anatomy of lower limb veins, the cerebral veins also show many normal variations.

The common variations are

(i) Asymmetry of transverse sinus with right sided dominance being most frequent.

(ii) Agenesis of part or full of transverse sinus.

(iii) Hypoplastic rostral end of superior sagittal sinus.

(iv) Hypoplastic inferior sagittal sinus.

# CEREBRAL VENOUS THROMBOSIS

## CAUSES

Any pathology that alters the Virchow's triads of flow, vessel wall and fluid blood physiology can cause thrombosis ***(J.Van Gijn)***. Stroke in young adults accounts 30% of all cases. Among this 10% to 20% are due to CVT. ***(Nagaraja D.Sarma GR).***

The various causes are ***(Karthikeyan, Vijay et al).***

1. Idiopathic
2. Pregnancy & Puerperium
3. Local infections like mastoiditis.
4. Dehydration
5. Oral Contraceptive Pills
6. Local tumor extension eg. Meningioma.
7. Systemic infection.
8. Systemic tumor syndrome.
9. Trauma
10. Coagulopathies
    - Antiphospholipid syndrome
    - Antithrombin III deficiency
    - Factor V Leiden mutation
    - Protein C/S deficiency
    - PNH

10. Inflammatory bowel disease
11. Behcet's syndrome
12. Nephrotic syndrome

In many cases of CVT, no obvious cause is detected and remains idiopathic in origin. Pregnancy and puerperium are associated with hormonal changes that are procoagulant in nature. Also during delivery dehydration may occur resulting in hemoconcentration.

Local infections like mastoiditis cause inflammation of the vessel wall and predisposes to thrombosis. Dehydration caused by diarrhea is an important cause for CVT in our settings. Dehydration cause hemoconcentration and thus sluggish flow predisposing to thrombosis.

Tumors like meningioma cause thrombosis by multiple factors. Large tumors cause pressure effect over adjacent sinus and cause flow disturbance. Also tumors may alter vessel wall by infiltration and cause thrombosis.

Systemic tumor syndrome and septicemia results in hypercoagulable state and endothelial damage resulting in thrombosis. Nephrotic syndrome results in loss of anticoagulant protein in urine and increased procoagulant proteins in serum resulting in thrombosis.

## CLINICAL FEATURES

Cerebral venous thrombosis is protean in its presentation making it very difficult to diagnose unless a high degree of suspicion is present. The presentation can be acute subacute, or chronic.

The various signs and symptoms at presentation are ***(Appenzeller et al)***.

1. Headache - Most common, present in > 80% of cases
2. Seizures - Present in 35% - 40% of cases. It can be focal or generalised.
3. Focal neurological signs - 30% - 35%
4. Altered sensorium - 30%
5. Vomitting.

The headache associated may be classical thunderclap headache or the more common nonspecific in nature. Headache may be caused by venous congestion, raised ICT, neuronal irritation, or SAH.

The seizure activity can be focal or generalised. It is caused by increased neuronal excitability due to edema, hemorrhage, infarct or raised ICT.

The focal neurologic deficits can be hemiparesis, paraparesis, monoparesis, dysphasia, blindness, cranial nerve palsy etc. Intermittent fluctuating hemiparesis is classical of CVT. Since the superior sagittal sinus thrombosis leads to bilateral parasagittal infarcts, spastic paraplegia may

occur. Involvement of cavernous sinus results in palsy of cranial nerves III, IV, V & VI.

The severity of altered sensorium lies between confusion to coma. It implies deeper parenchymal involvement. Since sudden confusion state which is unexplained by any pathology may be the only presenting symptom, CVT may be mistaken for psychiatric disorder.

Cavernous sinus thrombosis results in painful ophthalmoplegia, chemosis and proptosis. Cerebral venous sinus thrombosis can extend retrogradely into the cortical veins causing venous infarcts.

The signs and symptoms of cerebral venous thrombosis may mimic arterial stroke, intracranial tumors, intracranial infection, subarachnoid hemorrhage, or psychiatric disorders.

**Mortality and Morbidity**

The mortality rate ranges from 10% to 15%. The mortality is usually in the acute stage. The cause being involvement of brainstem, deep parenchyma, transtentorial herniation due to mass effect.

The residual deficit rate lies between 6% to 20%. The focal neurological deficits at admission may or may not be reversible. Simple edema and venous congestion, compression due to mass effect may reverse with treatment. Venous infarcts, parenchymal hematoma and secondary ischemic necrosis results in permanent neuronal damage and deficits. Depending on the region of brain involvement the deficits can be hemiparesis, monoparesis, paraparesis, blindness, cranial nerve palsy or dysphasia.

## DIAGNOSIS

Since the diagnosis of cerebral venous thrombosis is often missed by an unsuspecting clinician, radiological diagnosis remains an important part in its management.

The various modes of investigations are

1. CT - Nonenhanced and contrast enhanced.
2. MRI - T1WI, T2WI, FLAIR, GRE, MRV, DWI.
3. Catheter Angiogram.
4. Others.

## COMPUTED TOMOGRAPHY

The first investigation to be undergone by a patient with CNS signs and symptoms is CT. CT helps in diagnosing > 80% of cases of CVT. CT plays an important role in ruling out intracranial abscess or tumors.

The various signs of CVT in CT are.

1. Hyperdense triangle sign - Showing the thrombus within the superior sagittal sinus in NECT.
2. Cord sign - Dense cord like thrombosed cortical veins.
3. Empty Delta sign - In CECT the nonenhancing thrombus within the superior sagittal sinus is surrounded by enhancing collaterals. This is seen as an empty delta.

4. Parenchymal hemorrhage - Due to venous congestion and venous infarction parenchymal hemorrhage occurs. The typical location is subcortical region.

5. Parenchymal hypodensity - This is due to venous congestion edema or infarction. The edema is highly reversible whereas the infarct is not. The infarct is classically of non arterial territory, and involves bilateral convexity or bilateral basal ganglia / thalami.

6. Increased tentorial enhancement due to venous congestion.

False positive empty delta sign is seen in high splitting tentorium, subdural hematoma, subdural empyema.

NECT is an highly sensitive tool for intracranial hemorrhage which is a common finding in CVT.

Recently, using MDCT, venogram of intracranial venous system can be acquired ***(Karthikeyan, Vijay et al)***. Contrast enhancement with scanning in the venous phase is done. CT venogram is then reconstructed using MIP and MPR projection. This is highly sensitive and can be comparable to MRV. But the use of CT venogram is not used widely at present.

**CATHETER ANGIOGRAM**

Catheter angiogram can be done either conventionally or using DSA technique. The venous phase of angiogram is taken. Aortic arch injection of

contrast is done to simultaneously opacify all the four vessels of cerebral circulation. This avoids unopacified blood mimicking a clot within the sinus.

The thrombosed sinus appears as empty channel devoid of contrast surrounded by collaterals. Thrombosed cortical veins are seen as intraluminal filling defects and in delayed phase as cord of contrast collection. Since CT & MRI are sensitive and specific in CVT need of catheter angiogram is obviated ***(Laffile, Bourkobaz et al).***

**MRI AND MRV**

T1WI, T2WI, FLAIR, T2*WI MRV & DWI are used in diagnosis of sinus thrombosis and the parenchymal changes. FSE and GRE sequences are used. These sequences can directly visualise the thrombus within the venous sinus and are also sensitive in detecting the parenchymal lesions of edema, hemorrhage and infarcts.

**Thrombosis in Sinuses**

2D TOF MRA technique is used for venogram of cerebral sinuses. Time of flight phenomenon of flow related enhancement in used. Inflow of unsaturated spins in blood gives more signal when compared to the partially saturated stationary protons. Using post processing venograms are obtained. 2D TOF MRA is sensitive to slow flow. Coronal and oblique section may be taken.

The thrombus within the sinus can be imaged with T1 & T2 weighted images or with MRV. In T1 & T2 weighted images the normal venous sinus

shows flow void due to moving blood. In thrombosed sinuses the flow void is absent with varying signal depending on the age of thrombus and the sequences used.

In acute stages (First 3 to 5 days) the thrombus appears isointense in T1 & T2. During the subacute phase (5 to 30 days) the thrombus appears hyperintense in T1 & T2 images. It should be noted that recanalisation occurs in later stages which alters the signals from thrombus and a flow void may be present.

In 2D TOF MRV the slow flowing venous blood is imaged and canbe inspected as source image and MIP images. Absent signal with frayed appearance is a feature of thrombosis. Hypoplastic venous sinus, in plane effect, slow flow are some artefactual conditions for false positive finding. So a careful analysis of source images and T1, T2 sequences is mandatory. So also thrombus may have hyperintense signals in MRV mimicking flow. Use of contrast and evaluation of T1,T2 images clarify these queries.

The parenchymal edema appears hyperintense in T2WI and FLAIR images. Venous infarcts appear hyperintense in T2 WI and FLAIR images. It is differentiated from edema by DWI which shows hyperintense regions. Venous infarcts often show hemorrhage.

**Hemorrhage**

Hemorrhage can be intraparenchymal or in subarachnoid space. Intraparenchymal hemorrhage appearance in T1 and T2 images vary with the stage of hemoglobin breakdown.

Five stages of evolution of hematoma is recognised using MRI characters on T1 & T2 weighted images ***(Bradley WG et al Gomori et al).***

| | | | | |
|---|---|---|---|---|
| They are | 1. | Hyperacute | - | < 24 hrs |
| | 2. | Acute | - | 1 to 3 days |
| | 3. | Early subacute | - | 3 to 7 days |
| | 4. | Late subacute | - | 7 to 14 days |
| | 5. | Chronic | - | > 14 days |

In the hyperacute stage there is intracellular oxyhemoglobin. In the acute stage oxygen dissociate forming deoxyhemoglobin. During the subacute stage deoxyhemoglobin is oxidatively denatured to methemoglobin which is intracellular in early subacute and extracellular in late subacute due to RBC lysis. During the chronic phase heme is digested by the macrophages and converted to hemosiderin and ferritin.

The progression of hemoglobin catabolism within the hematoma starts in the periphery and extends into the centre. So at a point of time the hematoma consists of hemoglobin stages of varying ages. But the hematoma should be aged taking into account the most advanced stage of hemoglobin catabolism.

The signal intensities of the hematoma depends on age of the hematoma, weightage (T1 or T2) sequence used (FSE or GRE) & field strength.

The common pattern of signal intensities are

| Sl.No | Stages | T1 | T2 |
|---|---|---|---|
| 1. | Hyperacute (oxyhemoglobin) | ISO | Hyper |
| 2. | Acute (Deoxyhemoglobin) | ISO/Hypo | Hypo |
| 3. | Early Subacute (Intracellular methemoglobin | Hyper | Hypo |
| 4. | Late Subacute (Extracellular methemoglobin) | Hyper | Hyper |
| 5. | Chronic (Ferritin, hemosiderin) | Hypo | Hypo |

GRE sequences are more sensitive to hemorrhage ***(Luxia Liang et al)***. Since the 180° refocusing pulse of spin echo sequence is replaced by gradient pulses, the susceptibility is increased. Hemoglobin, because of its susceptibility effect causes signal loss of adjacent protons and appears hypointense.

FLAIR sequence is highly sensitive for subarachnoid hemorrhage ***(Rohit Bakshi et al)***. FLAIR is basically T2 image with suppression of free water protons. So the CSF is suppressed and hypointense in nature. This results in increased visibility of blood in subarachnoid space.

**OTHER INVESTIGATIONS**

Quantitative analysis of D- dimer in blood is done. This is a breakdown product of the thrombus. It is increased in the serum when there is venous thrombosis. A measurement of > 500 ng / ml is highly suggestive of CVT. It has a sensitivity of 83% and specificity of 90% ***(Lalive PH, Demoorloose et al)*** . It is a useful screening modality.

**TREATMENT**

The management of CVT consists of symptomatic treatment and definitive treatment of the thrombus.

It consists of
(i) Antiepileptics
(ii) Treatment of raised ICT
(iii) Antithrombotics
(iv) Treatment of primary cause.

Seizure is a poor prognostic factor. It results in secondary ischemia and brain damage. So control of seizures using antiepileptics both oral and parenteral remain one of the mainstay in CVT treatment.

CVT results in venous congestion, parenchymal edema, hemorrhage and infarcts. These results in raised ICT. This can result in transtentorial herniation of brain parenchyma and brainstem compression leading to sudden death. So prompt treatment of raised ICT prevents much of the mortality associated with CVT. Treatment consists of medical and surgical methods. Medical management includes IV mannitol. Surgical treatment includes burrhole decompression of cranium, hemicraniectomy, surgical debridement ***(Jam Stom et al)***.

Heparin remains the firstline treatment of CVT ***(Marie Germaine et al)***. Recent studies show its safety even in hemorrhagic lesions. It is followed by oral anticoagulants for atleast 12 months.

Local thrombolysis is reserved for those who donot respond well to the above treatment. Local urokinase infusion into the thrombosed sinus results in lysis of thrombus. It can be done through internal jugular or femoral vein routes. rtPA is a good alternative for urokinase. Complication include increased intracranial bleed and pelvic bleed from the puncture site.

Mechanical disruption of clot can be done using various mechanical devices. In case of deep vein thrombosis and cerebellar infarction ventricular drainage is used to relieve hydrocephalus.

# AIM OF THE STUDY

1. To evaluate the findings of cerebral venous sinus thrombosis using T1W, T2W, FLAIR, diffusion weighted images, and MR venogram.

2. To evaluate the prognosis by using restriction of diffusion in diffusion weighted images.

3. To evaluate the prognosis using T1WI, T2WI, FLAIR and MR Venogram.

# REVIEW OF LITERATURE

***1. Roettger C., Trittmache S., Gerriels T. Kops, Stolz E.***

15 patients with proven CVT and parenchymal lesions were followed up for 12 months. The one month follow up MR shows correlation of volume of lesion with recanalisation whereas 12 months followup didnot show this correlation of lesion volume and early recanalisation.

This implies that early lesions consist of both edematous and infarcted regions. Edema recovers early with recanalisation whereas the infarcts persist even after 12 months.

***2. Wassay M., Azeemuddin M.***

Diffusion weighted images are extremely sensitive in differentiating cytotoxic edema from vasogenic edema. Cytotoxic edema shows restricted diffusion appearing hyperintense in DWI.

***3. Mullins ME, Grants PE, Wang B Gonzalez RG, Schoefer PW***

13 patients were evaluated of which 10 showed parenchymal hemorrhagic lesions. Seven lesions showed increased diffusion (ie) vasogenic edema that resolved.

Three lesion had decreased diffusion but resolved in follow up images. Four lesions which were diffusion restricted persisted in follow up scans.

This implies all lesions with increased diffusion and a significant number of lesions with restricted diffusion are reversible with treatment.

***4. Imada, Ikowa, Kavamoto et al***

A case report of 37 yr old male patient with CVT and deep grey matter hyperintense lesion. The lesion showed no restriction on DWI.

With conservative management the follow-up MRI showed reversal of the lesion and complete recovery. MRI with DWI predicts reversibility of lesion when there is no diffusion restriction.

***5. Sarma D., Farb RI et al***

A case report of extensive CVT and parenchymal lesions with restricted diffusion in DWI.

On follow up MRI there was complete reversal of the lesion implying some lesions with restricted diffusion are also completely reversible with treatment.

***6. Milan, Garcia, Vila N. et al***

A case report of CVT with deep parenchymal lesions. The lesions showed no restriction in DWI.

The lesions completely recovered in followup scan implying the reversibility on vasogenic edema.

***7. Wassay M. Bakshi et al***

The authors reviewed 3 cases with CVT. Two patients showed hyperitensity in DWI implying cytotoxic edema and one showed hypointensity showing vasogenic edema.

***8. Favrole, Guichard, Crossland I et al***

28 patients with CVT were evaluated for diffusion within the clots. It was observed that restricted diffusion within the clot is a predictor for poor recanalisation in followup scans.

***9. Ducreax, Oppenheinm C et al***

Seven patients with CVT and T2 hyperintensities were evaluated with DWI. Five patients had hypointensity in DWI and recovered completely two had restricted diffusion of which one recovered and one had sequelae.

This study shows lesions with normal diffusion are reversible. Lesions with diffusion restriction are reversible in some and persist in some patients.

***10. Appenzeller S et al***

Twenty four patients with CVT presented with following symptoms

- Headache - 75%
- Vomitting - 33%
- Altered Sensorium - 21%

Probable causes were

- Pregnancy / Puerperium - 25%
- OCP - 17%
- Trauma - 8%

This study also showed CT to have a false negative rate of 37%

### *11. Oumurciuc R et al*

This study with 17 patients with isolated headache as presenting symptom showed lateral sinus involvement being most common.

### *12. Canhao P; Ferro JM et al*

A multinational prospective study including 624 patients with CVT and evaluation of the causes for death in the acute phase.

Mortality rate was 3.4% within 1 month. Causes of death were transtentorial herniation and multiple lesions. Independent predictors of death were coma, altered sensorium, deep CVT, right sided hemorrhage, and posterior fossa lesions.

### *13. Stolz, Rahimi, Gerrits et al*

The authors analysed 79 patients with CVT for causes of death.

The significantly related factors are age, neurologic deficit at presentation, more than 2 seizures even with treatment, venous infarct, hemorrhagic lesions.

***14. Karthikeyan D. S.Vijay T Kumar, Ikanth.***

Their observation showed the presenting symptoms as 75% with focal deficit and headache, 30% - 50% with seizures, 18% - 38% with benign ICT symptoms. Mortality rate of 10% - 80% and morbidity rate of 6% - 20%.

***15. Breteau, Mourier, Vehier et al***

3 years follow up of 55 patients showed 45 patients independent and 10 patients either dead or dependent.

# MATERIALS AND METHODS

Out study is a prospective cohort study of 48 patients carried out at Barnard Institute of Radiology, Madras Medical College, Chennai between Feb 2004 Jan 2006. All the 48 patients were subjected to MRI & MRV. MRI was performed using 1.5 Tesla super conducting SIEMENS MAGNETOM, SYMPHONY using head coil.

## Method

The patient is placed in supine position in the MR gantry with head coil positioned.

## MR Techniques

Multiplanar scout sections obtained for planning the sequences. Whole brain MR from vertex to the foramen magnum including the base of skull are taken using axial coronal and sagittal sections.

The sequences used were.

**T1WI Sagittal**
TR - 500 msec S/G - 5/1.5
TE - 14 msec

**T2WI Axial**
TR - 4000 msec S/G - 5/1.5
TE - 93 msec

**FLAIR Axial**
TR - 9000 msec S/G - 5/1.5
TE - 105 msec

**T2WI Coronal**
TR - 4000 msec S/G - 5/1.5
TE - 93 msec

**DWI Axial**
TR - 4300 msec S/G - 5/1.5
TE - 123 msec
b=0, 500, 1000

**GRE T2* Axial**
TR - 839 msec S/G - 5/1.5
TE - 27 msec

**MRV - 2D TOF**
TR - 30 msec
TE - 6.5 msec

**MRA - 3D TOF**
TR - 41 msec
TE - 6.8 msec

All the patients presented with CNS signs & symptoms some suspicious of CVT and others with vague features.

**Inclusion Criteria**

1. All patients confirmed by MRI & MRV as cerebral venous sinus thrombosis.
2. Males and females are included.
3. All age groups included.

**Exclusion Criteria**

1. Patients with MR incompatible devices or implants.
2. Patients on life support systems.
3. Patients with claustrophobia.

The clinical features at admission were analysed concentrating on.

1. Headache,
2. Seizure - Focal or generalised
3. Altered sensorium, coma,
4. Focal neurological deficit - Hemiparesis paraparesis, monoparesis, CN palsy, blindness, dysphasia.

**MRI Findings**

**1. Hemorrhage** - The age of the parenchymal hemorrhage whether acute, subacute, chronic is noted using T1W and T2W images. The site of hematoma whether superficial or deep is noted. The presence of subarachnoid hemorrhage is noted using FLAIR images.

**2. Thrombosis within Sinuses** - Using 2D TOF MRV, T1W and T2W images the thrombosis within the superior sagittal, inferior sagittal, straight, lateral sinuses are noted. Cortical vein thrombosis is identified.

**3. T2 hyperintense lesion in superficial and deep cerebral regions.**

T2 hyperintensities are due to prolongation of T2 time due to increased water protons. This increased water molecules can be either in extracellular or intracellular compartment.

Extracellular water is due to interstitial edema of vasogenic origin. Intracellular water is due to failure of membrane Na+ K + ATP ase due to decreased ATP since, in ischemia, hypoxia results in decreased ATP production. Due to pump failure Na+ accumulate intracellularly and along with it water molecules are trapped intracellularly.

T2 WI cannot differentiate these two types of edema.

**4. Diffusion Restriction**

Using DWIs and ADC map the sites of diffusion restriction and their extent are noted

**Time of Flight Effect**

When there is macroscopic proton movement across a slice as in blood flow within a vessel, there is either flow void (SE Sequence) or flow related enhancement (GRE Sequence).

The basic principle to this is that when a partially saturated proton is excited, the signal output is relatively less when compared with excitation of an unsaturated proton. So in a gradient sequence the stationary protons because of repeated excitation are partially saturated, whereas the unsaturated protons that flow fresh from outside the slice when excited gives higher signal. This is known as flow related enhancement.

2D Fourier Transform Gradient Echo TOF Technique in ideally used for MR venogram. Here sequential multiple thin slices of image acquisition is done. This is processed using 2D fourier transformation. 2D FT TOF is sensitive to slow flow. 3D TOF has the disadvantage of saturation of slow flowing blood.

Direct coronal or oblique scan section can be used so that the scan plane is perpendicular to the direction of blood flow. Using post processing MIP technique MR venogram is obtained. Motion compensation gradients, and short echo times reduces signal loss secondary to motion induced phase effect.

# Basic Principles

- Suppression of stationary tissue signal
- Generation of bright Blood flow signal

Image Slice

**Flow**

Saturated Spins

Relaxed Spins

# Bright Inflow Blood Signal

Radio frequency (RF) pulse

Longitudinal Magnetisation Vector

# Background Tissue Suppression

Radio frequency (RF) pulse

Longitudinal Magnetisation Vector

**Diffusion weighted images**

DWI are a set of sequences in which an added gradient is applied to the protons apart from the normal slice selection, frequency and phase encoding gradients.

Water protons normally diffuse randomly in the fluid medium. They can be restricted by macromolecular proteins, membranes of organelles within the cell. Normal size of cell lies between 10 to 20 µm diameter.

In the DWI sequence strong gradient dephasing pulse is first applied. Then after an interval of 40 to 50 msec another rephasing gradient pulse is applied. When the protons are stationary or restricted in diffusion during the interval of 40 msec they would not have diffused much distance and so the dephasing and rephasing pulse cancel each other resulting in good signal output.

When the protons are freely mobile the two gradients are not equally applied since the protons would have travelled across more distance. So there is partial saturation and so signal loss.

The time interval of 40 msec is taken since the proton under normal body conditions would travel a distance of about 10 to 15 µm during this period and this is approximately the size of the human cell.

The gradient strength is increased so that the b values are 0, 500,1000 The b value is directly proportional to the square of gradient strength.

$$b = \gamma^2 \delta^2 G^2 \ (\Delta - \delta/3)$$

At least two sets of images of varying b value are obtained and using mathematical derivation from the signal from each voxel the apparent diffusion coefficient is obtained for each voxel.

This is given shades of gray and plotted as an image giving the ADC map. When the protons are freely mobile there is complete loss of signal represented as hypointense signal in the diffusion sequence with increased ADC depicted as hyperintensity in ADC map.

When the protons are restricted in diffusion there is hyperintensity in higher gradient images with decreased ADC and hypointensity in ADC map.

CT, both NECT and CECT were taken using 5 mm posterior fossa sections and 10mm supratentorial sections.

CT is analysed for.

1. Empty delta sign - This is due to non enhancing thrombus within superior sagittal sinus with surrounding enhancing collaterals in CECT.

2. Hyperdense Triangle sign - caused by dense thrombus within the superior sagittal sinus in NECT.

3. Cord sign of thrombosed cortical veins.

4. Presence of hemorrhage - Hemorrhage appears hyperdense in CT. The hemorrhage is usually intraparenchymal in location with surrounding hypodensity. The hemorrhage can be due to

venous congestion or due to venous infarction. There can also be subarachnoid hemorrhage.

5. Hypodense lesion - Hypodense lesions of parenchyma can be superficial or deep. The hypodense lesions may represent either simple vasogenic edema due to venous congestion or an infarct. CT cannot differentiate the above two. The hypodense lesions are classically bilateral and subcortical in location.

The patients were treated with

1. Supportive care
2. IV fluid
3. Anti edema measures - IV mannitol, IV steroids.
4. Parenteral heparin
5. Oral anticoagulants
6. Antibiotics, Vitamins
7. Surgical decompression

**Follow up**

Followup of all patients at 8 weeks was done. Poor outcome was defined as either death or focal neurological deficit at the end of eight weeks. In our study at the end of 8 weeks 3 patients were dead, all died in the acute phase of the disease. 3 patients had hemiparesis, one had paresis of right upper limb, and two had hemiparesis with dysphasia.

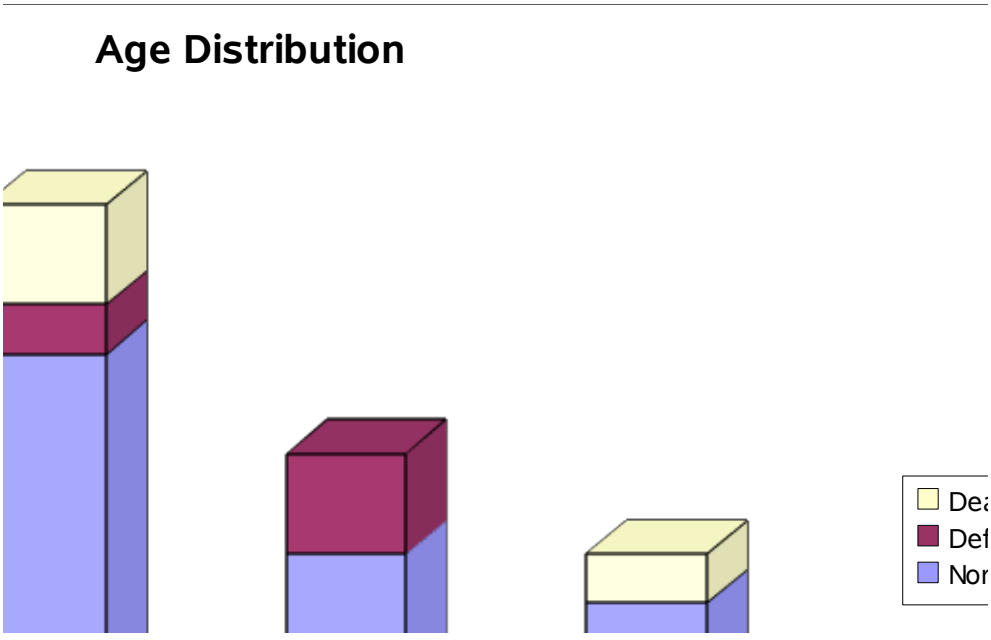
# OBSERVATIONS

## Table 1

## AGE DISTRIBUTION

| | AGE DISTRIBUTION | | | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|
| | < 25 | | 25 - 35 | | 35 - 45 | | > 45 | | | |
| FOLLOWUP | Count | % | Count | % | Count | % | Count | % | Count | % |
| Normal | 12 | 80 | 12 | 80.0 | 8 | 80 | 7 | 87.5 | 39 | 81.25 |
| Deficit | 3 | 20 | 1 | 6.7 | 2 | 20 | | | 6 | 12.5 |
| Death | | | 2 | 13.3 | | | 1 | 12.5 | 3 | 6.25 |
| Total | 15 | 100 | 15 | 100.0 | 10 | 100 | 8 | 100 | 48 | 100 |

There is no significant association between age of the patients and the prognosis.

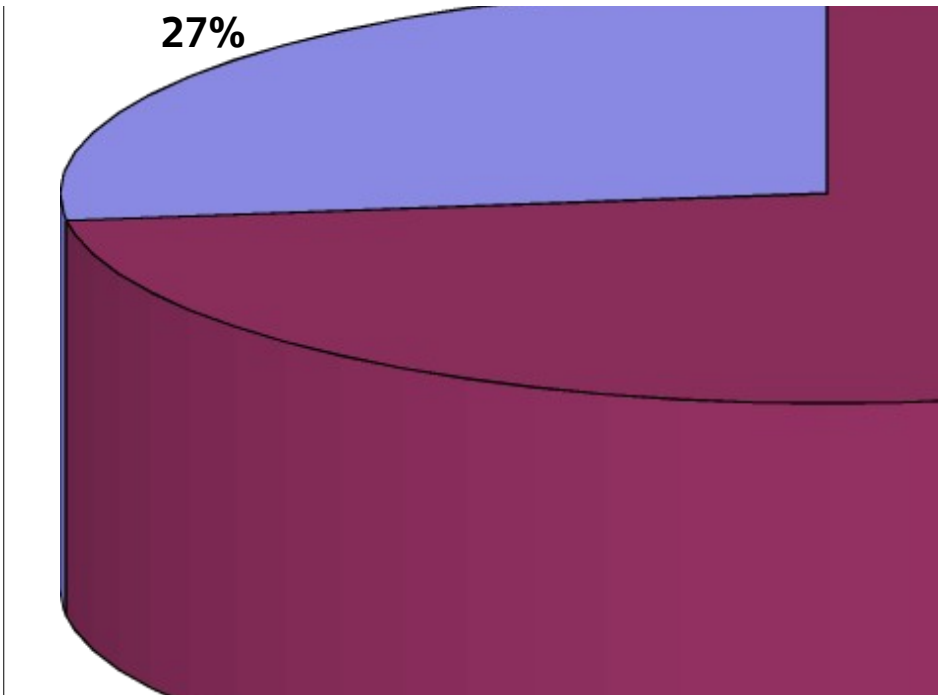


## Table 2

## SEX DISTRIBUTION

| SEX | FOLLOWUP | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|
| | Normal | | Deficit | | Death | | | |
| | Count | % | Count | % | Count | % | Count | % |
| Female | 28 | 71.8 | 5 | 83.3 | 2 | 66.7 | 35 | 72.9 |
| Male | 11 | 28.2 | 1 | 16.7 | 1 | 33.3 | 13 | 27.1 |
| Total | 39 | 100.0 | 6 | 100.0 | 3 | 100.0 | 48 | 100.0 |

There is no significant association between sex of the patient and the prognosis.

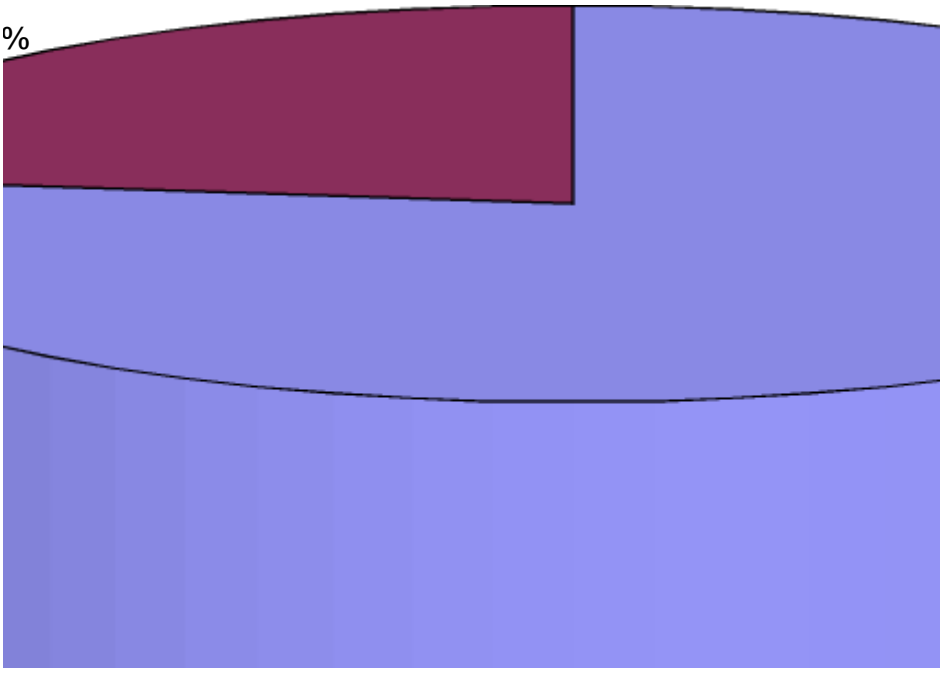


## Table 3

## PUERPERIUM

| FOLLOWUP | PUERPERIUM | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 7 | 87.5 | 21 | 77.8 | 28 | 80.0 |
| Deficit | | | 5 | 18.5 | 5 | 14.3 |
| Death | 1 | 12.5 | 1 | 3.7 | 2 | 5.7 |
| Total | 8 | 100.0 | 27 | 100.0 | 35 | 100.0 |

Out of 35 female patients 27 were puerperal patients. There is no significant association with prognosis.

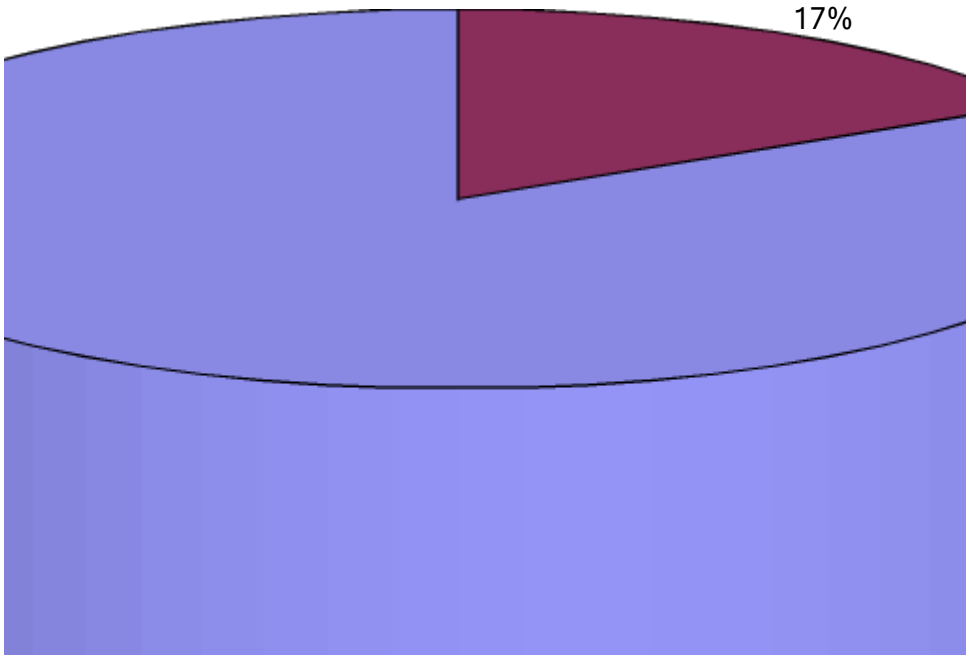


## Table 4

## CLINICAL PRESENTATION - HEADACHE

| FOLLOWUP | HEADACHE | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 8 | 100.0 | 31 | 77.5 | 39 | 81.3 |
| Deficit | | | 6 | 15.0 | 6 | 12.5 |
| Death | | | 3 | 7.5 | 3 | 6.3 |
| Total | 8 | 100.0 | 40 | 100.0 | 48 | 100.0 |

40 (83.3%) patients out of 48 presented with complaints of headache.



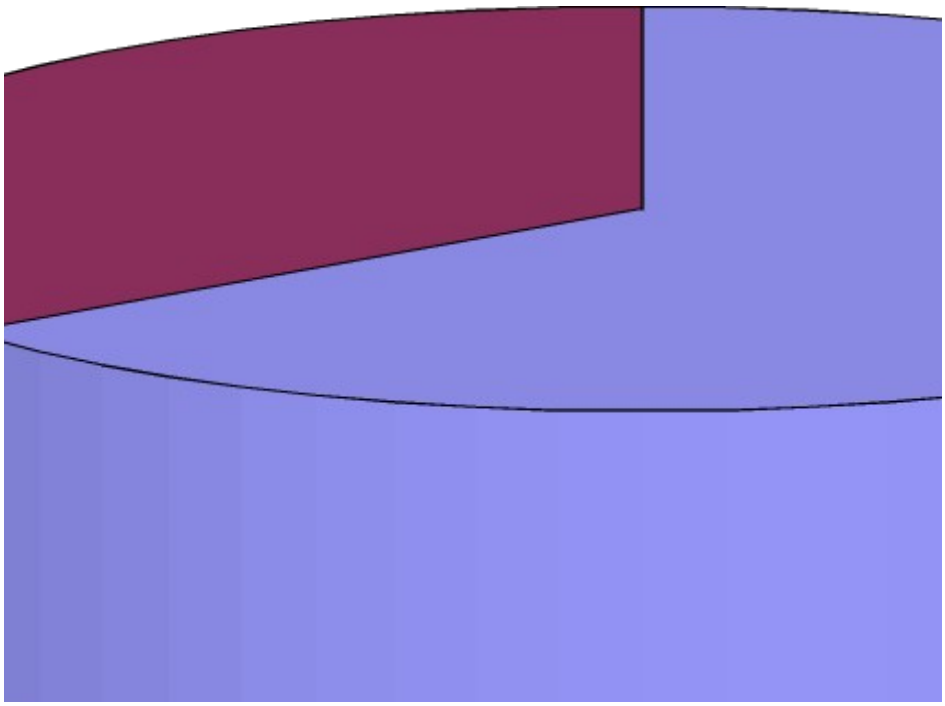
## Table 5

## CLINICAL PRESENTATION - SEIZURE

| FOLLOWUP | SEIZURE | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 27 | 87.1 | 12 | 70.6 | 39 | 81.3 |
| Deficit | 3 | 9.7 | 3 | 17.6 | 6 | 12.5 |
| Death | 1 | 3.2 | 2 | 11.8 | 3 | 6.3 |
| Total | 31 | 100.0 | 17 | 100.0 | 48 | 100.0 |

17 (35.4%) out of 48 patients had seizure at presentation.

There is no statistically significant association between headache or seizure with prognosis ($P > 0.05$).



## Table 6

## CLINICAL PRESENTATION - ALTERED SENSORIUM

| FOLLOWUP | ALTERED SENSORIUM | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 35 | 89.7 | 4 | 44.4 | 39 | 81.3 |
| Deficit | 4 | 10.3 | 2 | 22.2 | 6 | 12.5 |
| Death | | | 3 | 33.3 | 3 | 6.3 |
| Total | 39 | 100.0 | 9 | 100.0 | 48 | 100.0 |

$p < 0.01$ (Chi square test)

Nine (18.7%) out of 48 patients presented with altered sensorium. Out of this 9 patients 5 had poor outcome which is statistically significant ($p < 0.01$).

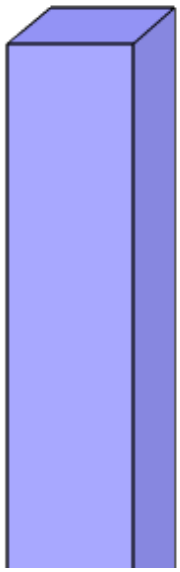


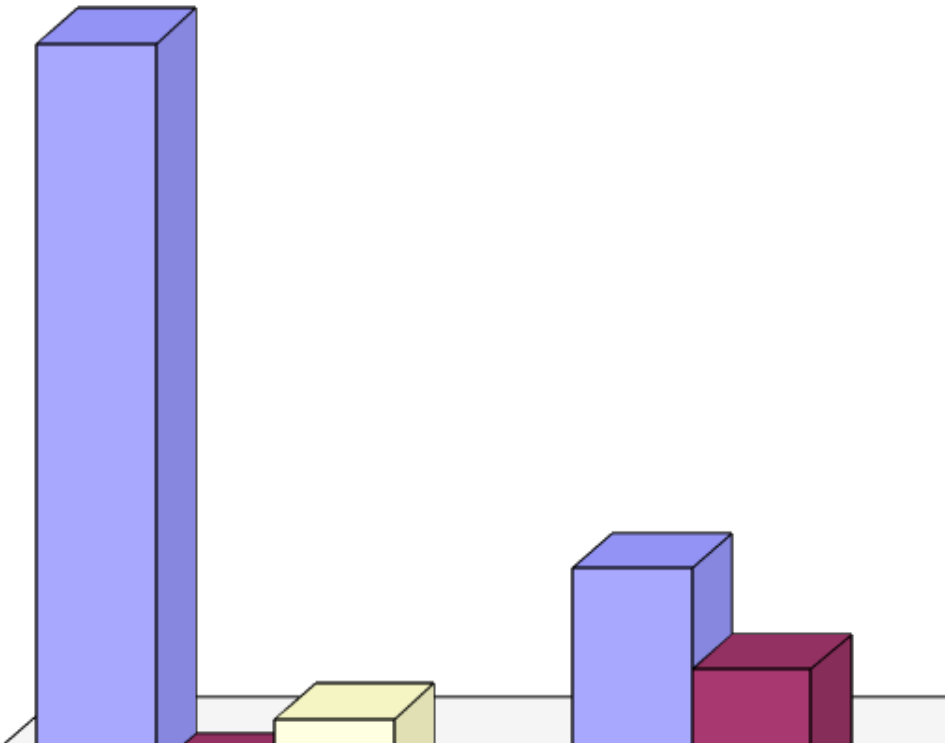
**Table 7**

**CLINICAL PRESENTATION - FOCAL NEUROLOGICAL DEFICIT**

| FOLLOWUP | DEFICIT | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 30 | 88.2 | 9 | 64.3 | 39 | 81.3 |
| Deficit | 1 | 2.9 | 5 | 35.7 | 6 | 12.5 |
| Death | 3 | 8.8 | | | 3 | 6.3 |
| Total | 34 | 100.0 | 14 | 100.0 | 48 | 100.0 |

$p < 0.01$ (Chi square test)

5 out of 9 patients who presented with deficit had poor outcome. Which is statistically significant ($p < 0.01$)



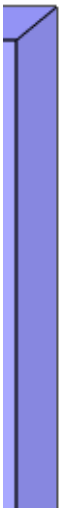
## Table 8

## CT - PARENCHYMAL HEMORRHAGE

| FOLLOWUP | CT- PARENCHYMAL HEMORRHAGE | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 33 | 94.3 | 6 | 46.2 | 39 | 81.3 |
| Deficit | 1 | 2.9 | 5 | 38.5 | 6 | 12.5 |
| Death | 1 | 2.9 | 2 | 15.4 | 3 | 6.3 |
| Total | 35 | 100.0 | 13 | 100.0 | 48 | 100.0 |

$p < 0.01$ (Chi square test)

Presence of hemorrhage in CT is associated with poor outcome. This association is highly significant ($p < 0.01$).



## Table 9

## CT - SSS THROMBOSIS

| FOLLOWUP | CT - SSS THROMBOSIS | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 12 | 100.0 | 27 | 75.0 | 39 | 81.3 |
| Deficit | | | 6 | 16.7 | 6 | 12.5 |
| Death | | | 3 | 8.3 | 3 | 6.3 |
| Total | 12 | 100.0 | 36 | 100.0 | 48 | 100.0 |

## Table 10

## CT - LS THROMBOSIS

| FOLLOWUP | CT - LS THROMBOSIS | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 20 | 83.3 | 19 | 79.2 | 39 | 81.3 |
| Deficit | 3 | 12.5 | 3 | 12.5 | 6 | 12.5 |
| Death | 1 | 4.2 | 2 | 8.3 | 3 | 6.3 |
| Total | 24 | 100.0 | 24 | 100.0 | 48 | 100.0 |

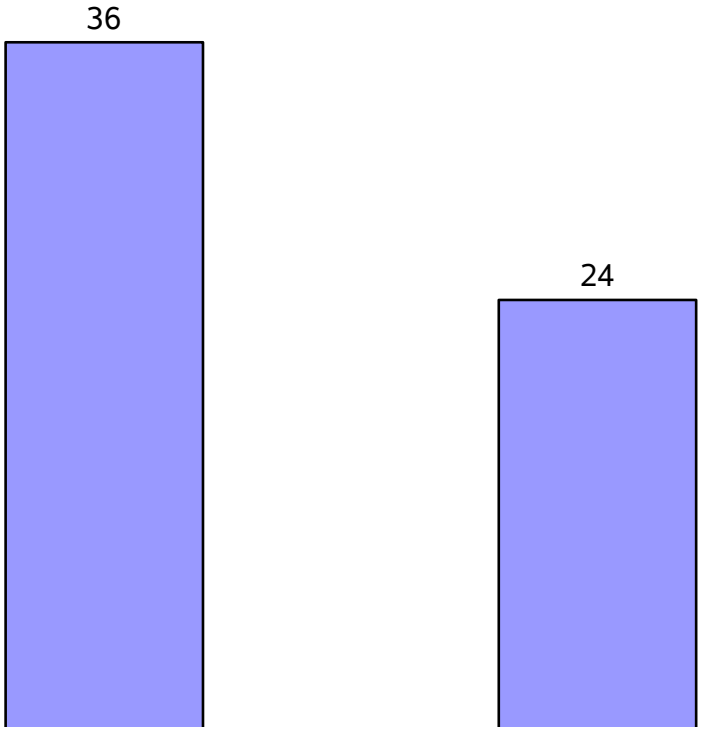
**Table 11**

**CT - STS THROMBOSIS**

| FOLLOWUP | CT - STS THROMBOSIS | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 37 | 84.1 | 2 | 50.0 | 39 | 81.3 |
| Deficit | 5 | 11.4 | 1 | 25.0 | 6 | 12.5 |
| Death | 2 | 4.5 | 1 | 25.0 | 3 | 6.3 |
| Total | 44 | 100.0 | 4 | 100.0 | 48 | 100.0 |

($p > 0.05$ Chi square test)

There is no statistically significant association of thrombosis in CT with outcome ($P > 0.05$).



## Table 12

## CT - HYPODENSITY

| FOLLOWUP | CT - HYPODENSITY | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 36 | 92.3 | 3 | 33.3 | 39 | 81.3 |
| Deficit | 3 | 7.7 | 3 | 33.3 | 6 | 12.5 |
| Death | | | 3 | 33.3 | 3 | 6.3 |
| Total | 39 | 100.0 | 9 | 100.0 | 48 | 100.0 |

$p < 0.01$ (Chi square test)

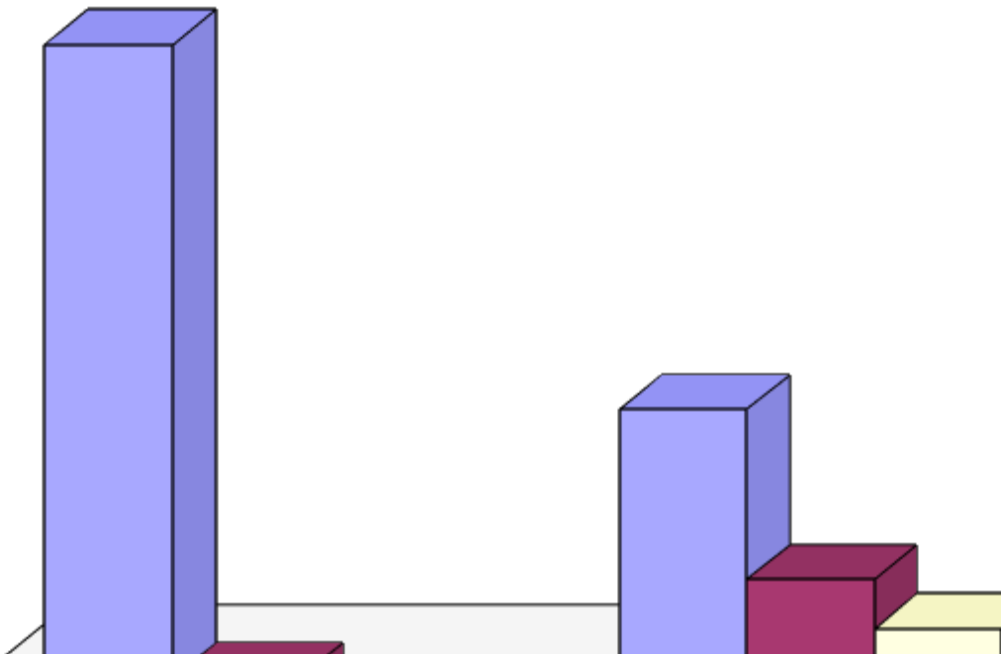
66% of patients with hypodensity had poor outcome when compared to 7.7% of patients without hypodensity which has high statistical significance ($p < 0.001$).

**Table 13**

**MRI HEMORRHAGE**

| FOLLOWUP | MRI HEMORRHAGE | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 27 | 96.4 | 12 | 60.0 | 39 | 81.3 |
| Deficit | 1 | 3.6 | 5 | 25.0 | 6 | 12.5 |
| Death | | | 3 | 15.0 | 3 | 6.3 |
| Total | 28 | 100.0 | 20 | 100.0 | 48 | 100.0 |

20 patients( 41.6%) presented with hemorrhage in MRI 8 patients out of this had poor outcome with high significance ($p < 0.01$)



## Table 14

## MRI - SSS THROMBOSIS

| FOLLOWUP | MRI - SSS THROMBOSIS | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 9 | 100.0 | 30 | 76.9 | 39 | 81.3 |
| Deficit | | | 6 | 15.4 | 6 | 12.5 |
| Death | | | 3 | 7.7 | 3 | 6.3 |
| Total | 9 | 100.0 | 39 | 100.0 | 48 | 100.0 |

## Table 15

## MRI - LS THROMBOSIS

| FOLLOWUP | MRI- LS THROMBOSIS | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 12 | 80.0 | 27 | 81.8 | 39 | 81.3 |
| Deficit | 3 | 20.0 | 3 | 9.1 | 6 | 12.5 |
| Death | | | 3 | 9.1 | 3 | 6.3 |
| Total | 15 | 100.0 | 33 | 100.0 | 48 | 100.0 |

**Table 16**

**MRI - STS THROMBOSIS**

| FOLLOWUP | MRI - STS THROMBOSIS | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 34 | 89.5 | 5 | 50.0 | 39 | 81.3 |
| Deficit | 4 | 10.5 | 2 | 20.0 | 6 | 12.5 |
| Death | | | 3 | 30.0 | 3 | 6.3 |
| Total | 38 | 100.0 | 10 | 100.0 | 48 | 100.0 |

$p < 0.01$ (Chi square test)

81.3% patients presented with SSS thrombosis, 68.7% presented with LS thrombosis. No significant association of SSS, LS thrombosis with poor outcome.

20.8% patients had St.Sinus thrombosis. It is significantly associated with poor outcome ($p < 0.01$)

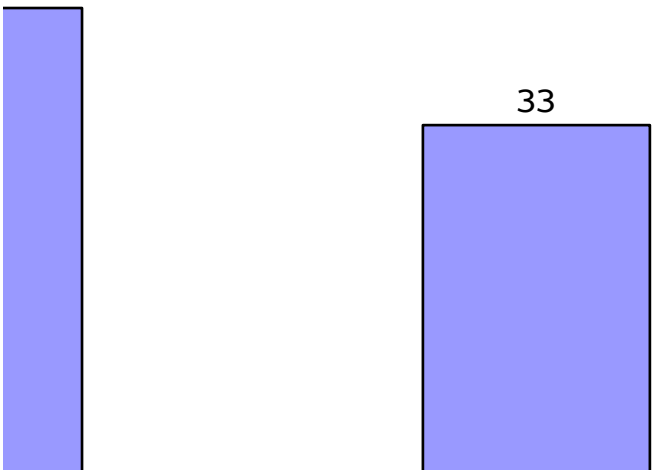


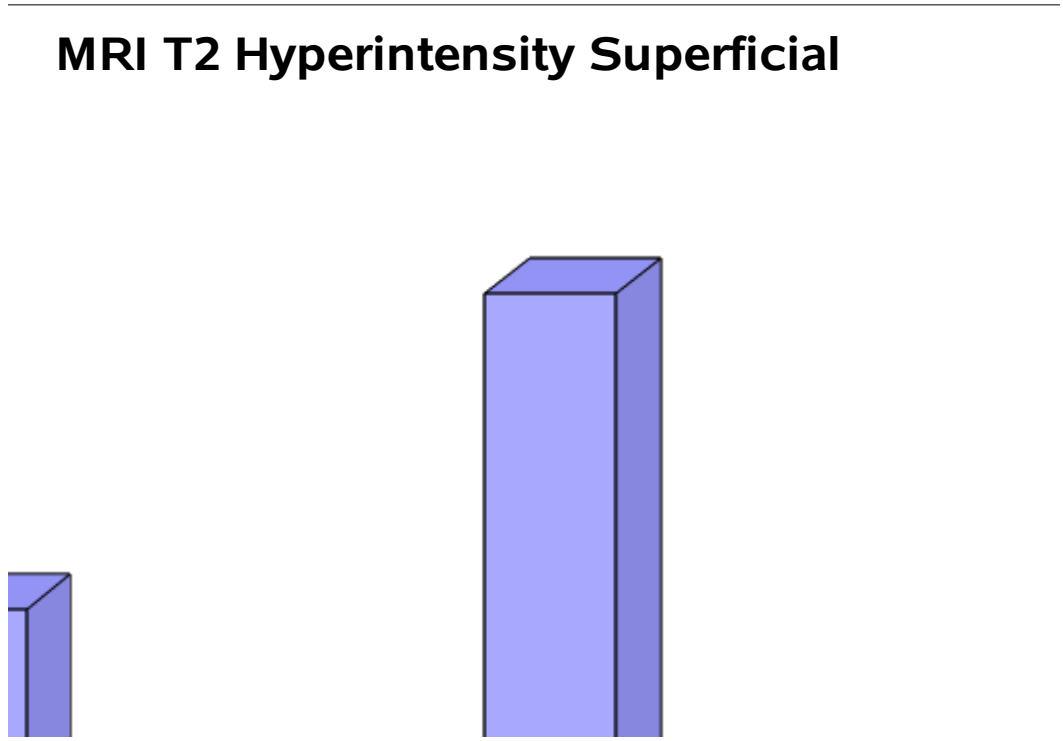
**Table 17**

**MRI - T2 HYPERINTENSITY SUPERFICIAL**

| FOLLOWUP | T2 HYPER - SUPERFICIAL | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 13 | 92.8 | 26 | 76.5 | 39 | 81.3 |
| Deficit | | | 6 | 17.6 | 6 | 12.5 |
| Death | 1 | 7.2 | 2 | 5.9 | 3 | 6.3 |
| Total | 14 | 100.0 | 34 | 100.0 | 48 | 100.0 |

p = 0.24 (Chi square test)

There is no significant association of T2 hyper intensity in superficial parenchyma with poor outcome



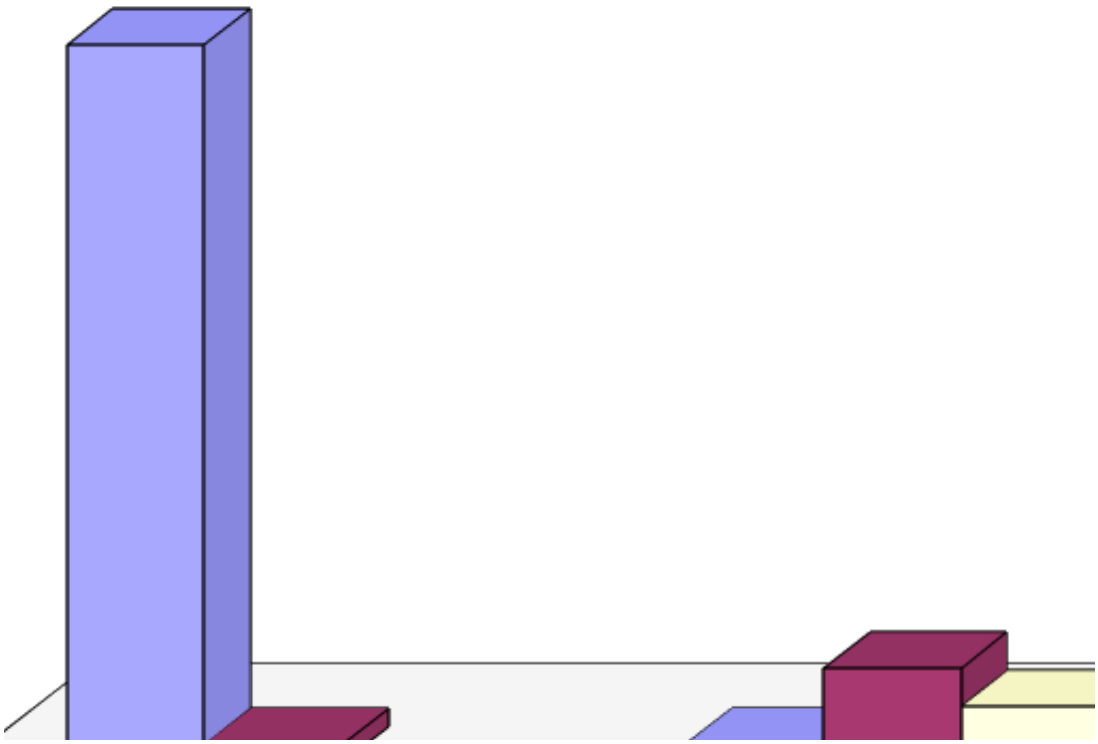
## Table 18

## MRI T2 HYPERINTENSITY DEEP

| FOLLOWUP | T2 HYPER - DEEP | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 38 | 97.4 | 1 | 11.1 | 39 | 81.3 |
| Deficit | 1 | 2.6 | 5 | 55.6 | 6 | 12.5 |
| Death | | | 3 | 33.3 | 3 | 6.3 |
| Total | 39 | 100.0 | 9 | 100.0 | 48 | 100.0 |

$p < 0.001$ (Chi square test)

9 out of 48 patients had deep parenchymal hyperintensity of which 8 had poor outcome which shows a highly significant association ($p < 0.01$).



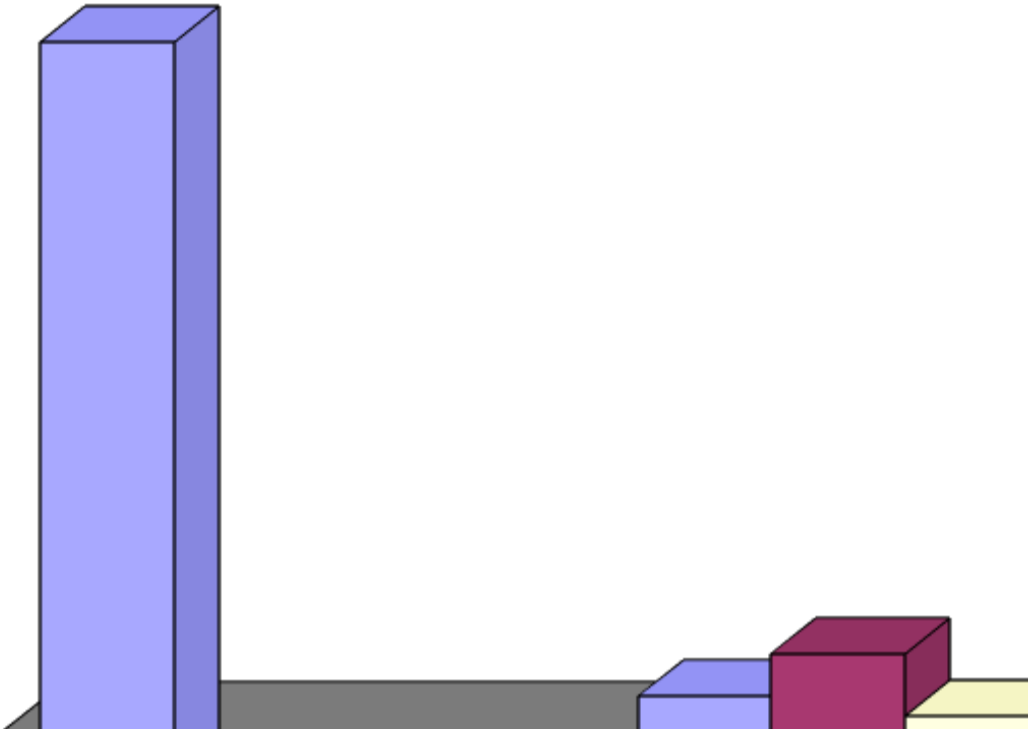
## Table 19

## MRI - DIFFUSION RESTRICTION

| FOLLOWUP | MRI-DIFFUSION RESTRICTION | | | | Total | |
|---|---|---|---|---|---|---|
| | Negative | | Positive | | | |
| | Count | % | Count | % | Count | % |
| Normal | 35 | 100.0 | 4 | 30.8 | 39 | 81.3 |
| Deficit | | | 6 | 46.2 | 6 | 12.5 |
| Death | | | 3 | 23.1 | 3 | 6.3 |
| Total | 35 | 100.0 | 13 | 100.0 | 48 | 100.0 |

$p < 0.001$ (Chi square test)

Follow up is highly associated with MRI diffusion ($p < 0.001$) all 9 patients who had poor outcome had diffusion restriction.



# DISCUSSION

In our study done in Barnard Institute of Radiology Madras Medical College, between Feb 2004 to Jan 2006 48 patients were included. All these patients were selected with MR evidence of cerebral venous sinus thrombosis.

The age range of the affected patients is 18 to 66 with a mean age of 33. There is no statistically significant association of age with poor outcome in our study as against the study of ***Stolz, Rahimi et al*** who showed increasing age to be a poor prognostic indicator.

The Male : Female sex ratio in our study is 1:2.7 that is, males form 27% of the patients. This shows that CVT is not uncommon among males and should be a differential diagnosis in any patient with suspicious CNS signs and symptoms. Sex doesnot play a role in outcome of CVT in our study.

Among the 35 female patients 21 were in puerperal period (ie) 60% of female patients. This proves that puerperium is high risk for CVT. The outcome among puerperal females does not show any significant difference from the nonpregnant female.

In the study of ***Appenzeller et al*** pregnancy, puerperium, OCP are good prognostic indicators.

In our study the percentage of presenting symptoms are

| | | |
|---|---|---|
| Headache | - | 83.3% |
| Seizure | - | 35.4% |

Altered Sensorium - 18.7%

Neurological Deficit - 29.1%

Among these presenting symptoms, altered sensorium and focal neurologic deficit are statistically significant poor prognostic indicators with patients in coma related to death and focal deficit related to deficit at 8 weeks follow up. This is in agreement with the study of ***Appenzeller et al*** and ***Ferro, Canhao et al.***

The number of patients with parenchymal hemorrhage detected by CT is 13 out of which 7 had poor outcome. It is found that this sign is a poor prognostic indicator with $P < 0.01$. According to ***Ferro, Canhao et al*** hemorrhage on admission CT is associated with poor outcome.

The incidence of thrombosis in CT are

Superior Sagittal Sinus - 75%

Lateral Sinus - 50%

Straight Sinus - 8.3%

There is no significant association of thrombosis with the patient outcome.

The presence of parenchymal hypodensity in CT which can be either simple vasogenic edema or a venous infarct was found to be in 9 patients (18.7%) of which 3 died and 3 had deficit. This has a highly significant association with poor outcome with $P < 0.001$.

CT was normal in 9 patients ie 18.7%

41.6% of patients had intraparenchymal hemorrhage detected by MRI. 8 patients out of this had poor outcome. That is 8 out of the 9 poor outcome group had hemorrhage. This is a statistically significant association.

The presence of thrombosis detected by MRI are

Superior Sagital Sinus - 81.3%

Lateral Sinus - 68.7%

Straight Sinus - 20.8%

This corresponded with similar statistics in literature.

The presence of thrombosis in superior sagittal sinus and lateral sinus is not associated with poor outcome. Whereas straight sinus thrombosis is associated significantly with poor outcome.

***Ferro Canhao et al*** predicted a poor outcome with deep cerebral venous thrombosis. Deep Cerebral venous thrombosis is associated with thalamic and deep grey matter and brainstem lesion. So it is associated frequently with altered sensorium and poor Glascow coma scale . This explains the poor outcome of patients with deep cerebral venous thrombosis.

Straight sinus thrombosis is seen in 4 patients in CT whereas in MRI in 10 patients showing the greater pickup rate in MRI.

Detection of deep T2 hyperintense lesions has a poor outcome. In our study 9 out of 48 had deep T2 hyperintense lesions. Out of this 9 patients 8 (88.8%) had poor outcome. In our study of the 9 poor outcome patients 8 (88.8%) had deep T2 hyperintense lesions. So deep hyperintense lesion are sensitive and specific in predicting poor outcome.

Edema can be vasogenic or cytotoxic edema. Cytotoxic edema is due to entry of water into the cells due to decreased ATP. This in course leads to cell death and infarction. So cytotoxic edema is the precursor of infarction. Since intracellular macromolecules and membranous organelles interact and bound with the intracellular water molecules, these protons have restricted diffusion.

This can be detected by using DWI in which separate diffusion gradients of varying strengths are given and the signal loss due to diffusion is measured.

In DWI free protons have decreased signals in higher gradients, whereas bound or restricted protons give higher signals or hyperintense. It is to be remembered that cytotoxic edema is only the precursor of infarction.

In our study group all the 9 patients who had poor outcome had restricted diffusion. Four patients who had restricted diffusion had no residual CNS deficit.

In other words all poor outcome patients had diffusion restriction i.e 100% sensitive in predicting outcome. But 4 patients who had restricted diffusion recovered completely i.e it is less specific in predicting the outcome.

According to ***Mullins et al*** three out of 14 lesions had restricted diffusion but resolved in follow-up and 4 out of 14 had restricted diffusion and persisted.

In our study against the above study 9 out of 30 had restricted diffusion and had poor out come and 4 out of 30 had restricted diffusion and with good outcome.

Reversible DWI restricted lesions are shown in the studies of ***Ducreax, Appenhium et al, Sarma D, Farbe et al.***

# SUMMARY

In our prospective cohort study of 48 patients with cerebral venous thrombosis diagnosed by MRI and MRV it can be summarised as follows.

1. Cerebral venous thrombosis incidence is not uncommon in males.

2. Puerperium is a risk factor for cerebral venous thrombosis.

3. Presence of altered sensorium and focal neurological deficits at admission predicts poor outcome.

4. CT is a good first line investigation for Cerebral venous thrombosis.

5. Presence of intraparenchymal hemorrhage and parenchymal hypodense lesions in CT are associated with poor outcome.

6. MRI features of poor outcome are

   (i) Presence of intraparenchymal hemorrhage

   (ii) Straight sinus thrombosis

   (iii) Presence of deep parenchymal T2 hyperintense lesion.

   (iv) Restriction of diffusion.

# CONCLUSION

In our study of cerebral venous thrombosis it can be concluded that MRI is useful in prognostic evaluation. Diffusion weighted images are good prognostic tools. Restriction of diffusion is a poor prognostic indicator.

MRI features of deep T2 hyperintense lesions, intraparenchymal hemorrhage and straight sinus thrombosis are poor prognostic factors.

Presence of altered sensorium and focal neurological deficit at admission and CT findings of parenchymal hemorrhage and parenchymal hypodense lesions also predict poor outcome.

# BIBLIOGRAPHY


1. Appenzeller, Zeller CB et al
CVT : Influence of risk factors and imaging findings on prognosis.
Clin. Neurol Neurosurgery 2005 Aug 107(5) : 371 - 8.

2. Ameridou, Spiloti et al
Bithalamic infarct - CVT Vs Basilar tip embolism.
Clin. Neurol Neurosurgery 2004 Sep 106 (4) 345 - 7.

3. Biourse, Tong, Newman NJ.
Cerebral Venous Thrombosis
Curr Treat option Neurol : 2003 Sep 5 (5) 409 - 20.

4. Breteau G. Mounier - Vehier et al
CVT - 3 yr clinical outcome in 55 consecutive patients.
J.Neurol 2003 Jan 250 (1) 29 - 35.

5. Bergui, Bradac,
Clinical picture of patients with CVT and pattern of dural sinus involvement.
Cerebrovas dis 2003 16 (3) 211 - 6.

6. Bradley WG et al.,
MR appearance of hemorrhage in brain
Radiology 1993 189 : 15.

7. Cumurciuc R. Crassard et al
Headache as the only neurological symptom of CVT.
J.Neurol Neurosurgery. Psy 2005 Aug 76 (8) : 1043

8. Canbao, Ferro JM et al
Causes and predictors of death in CVT.
Stroke 2005 Aug 36 (8) 1720 - 25.

9. Chu Kang Yoon, Roph K.
DWI in CVT.
Arch Neurol 2001 Oct 58 (10) 1538 - 9

10. Corvol, Oppenheim et al.
DWI in a case of CVT
Stroke 1998 Dec 29 (12) 2649 - 52.

11. Crombe - Haven , Gille
Isolated deep CVT diagnosis on CT / MR.
JBR - BTR 2003 Sep - Oct 86 (5) 257 - 61.

12. Connor, Jarosz
MRI in CVT
Clin Radiol 2002 Jun 57 (6) 449 - 61.

13. Crassard, Brasser,
Cerebral Venous Thrombosis,
J.Neuro ophthol 2004 Jun 24 (2) 156 - 63,

14. Ducreax, Oppenhim et al
DWI in CVT
AJNR 2001 Feb 22(2) 261 - 8.

15. Favrole, Guichard, Crassard et al
DWI of intravascular clot in CVT.
Stroke 2004 Jan 35 (1) 99 - 103.

16. Ferro GM
Cerebral Venous Thrombosis
J. Neuroradiol 2002 Dec 29 (4) 231 - 9.

17. Forbes KP pipe Heisermann
Cytotoxic edema in CVT
AJNR 2001 Mar 22 (3) 450 - 5.

18. Fery, Muro et al
CVT - Combined intrathrombus rtPA and IV heparin
Stroke 1999 Mar 30 (3) 489 - 94.

19. Gomori et al
Intracranial hematoma
Radiology 1985 157 : 87.

20. Hermann, Sporev. Yoursy
Thrombosis of internal cerebral vein with transient unilateral thalamic edema - a case report.
AJNR 2004 Sep 25 (8) 135 1-5.

21. Iurlaro, Beghi, Masetto etal
Headache as clinical marker in CVT. A prospective multicentric study.
Neurol Sci 2004 Oct 25 : Suppl 3 S298 - 99.

22. Imada, Ikawa et al
Usefullness of DWI in diagnosis and treatment of CVT - a case report.
No.Shinkei Geka 2004 Mar 32 (3) 285 - 9.

23. Iankowicz, Drozdowski
CVT - Clinical aspects and consequences
Neurol Neurochir Pol 2003 Sept - Oct 37 (5) 1073 - 84.

24. Jan Stam
Treatment of CVT.
NEJM Apr 2005 152 : 1791 - 98.

25. Kuwahara, Abe et al
DWI in CVT - Reversible parenchymal lesion.
No To Shirkei 2001 Jan 53 (1) 79 - 83.

26. Keller, Flacke, Urbach, Schild HH
DWI in deep CVT.
Stroke 1999 May 30 (5) 1144 - 6.

27. Karthikeyan, Vijay, Kumar, Kanth
CVT - Spectrum of CT Findings
IJRI 2004 14 : 2 : 129 - 137.

28. Lalive, de moerlase, Lovblad et al
Measurement of D - dimer in CVT
Neurology 2003 Oct 28 : 61 (8) 1057 - 60.

29. Loffita, Barkobza et al
Clin Radiology 1997 Sep 52 (9) 671 - 9.

30. Luxia Lioang Yukunori et al
Detection of intracranial hemorrhage with GRE
AJNR 1999. 20 - 1527 - 34.

31. Lovblad, Bassetti, et al
DWI in CVT
Cerebrovas Dis 2001 : 11 (3) 169 - 76.

32. Lovblad, Bassetti et al
DWI in deep CVT
Neuroradiology 2000 Oct 42 (10) 728 - 31

33. Lee Sk, ter Brugge
CVT in adult - Role of imaging & management
Neuroimaging Clin N.Am 2003 Feb 13 (1) 139 - 52.

34. Mullin Me Grant PE, WangB Conzolez
CVT - Parenchymal abnormalities - Assessment with DWI.
AJNR 2004 Nov - Dec 25 (10) 1666 - 75.

35. Millan, Garcia, et al
DWI in CVT
Neurologia 2002 Oct 17(80) 443 - 6.

36. MoodyAR
MR - Direct thrombus imaging
J.Thromb Hemostat 2003 Jul 1 (7) 1403 - 9.

37. Nagaraja Sarma
Treatment of CVT
Neurolog India 2002 June 50(2) 114 - 16.

38. Rottger C. Trittmacher Gerriet T et al
Reversible MR imaging abnormalities following CVT.
AJNR. 2005 Mar 26 (3) 607 - 13.

39. Roy PM Gras E
Images in Clinical medicine, CVT,
N.Engl JM 2003 Oct 30 349 (18) 1730.

40. Renowden S.
Cerebral Venous Thrombosis
Eur. Radiol 2004 Feb 14 (2) 215 - 26.

41. Radier, Schluck et al
Progression of CVT - A retrospective study
Presse Med 2003 May 10 32 (16) 728 - 35.

42. Rohit Bakshi et al
FLAIR in intraventricular hemorrhage
AJNR. 1999 Apr 20; 629 - 36.

43. Stolz E Rahimi Gerrieds, Kraus, Karps M.
CVT : Prognostic factors and long term outcome
Clin Neurol Neurosurg 2005 Feb 107 (2) 99 - 107.

44. Sarma D. Farb Mickuliz, TerBrugge
Reversal of restricted diffusion in CVT - a case report.
Neuroradiology Feb 2004, 46 (2) 118 - 21.

45. Schwarte, Daffershofer et al
Current controversies in diagnosis and Management of CVT
Nervenarzt 2003 Aug 74(8) 639 - 53.

46. Villringer Einhaupl
Dural sinus and CVT
New Horizon 1997 Nov 5 (4) 332 - 41.

47. Wasay, Bakshi et al
DWI in SSS thrombosis
J. Neuroimaging 2002 Jul 12; (3) 267 - 9.

48. Wassay M. Azeemuddin M.
Neuroimaging 2005 Apr 15(2) 118 - 28.

# PROFORMA

Name: Age: I.P.No:

| | |
|---|---|
| > 25 Yrs | |
| 25-35 Yrs | |
| 35-45 Yrs | |
| >45 Yrs | |

Sex : M/F Address:

| | |
|---|---|
| Puerperium | |
| Others | |

**Clinical features**

| | | |
|---|---|---|
| Headache | : | Yes / No |
| Seizure | : | Yes / No<br>Focal / Generalised |
| Altered sensorium | : | Yes / No |
| Coma | : | Yes / No |
| Focal Neurol Deficit | : | Yes / No<br>Hemiparesis / Monoparesis / Paraparesis / Dysphasia / Blindness / CN deficit / |

Others.

**Investigations**

| | | | | |
|---|---|---|---|---|
| CT - | Hemorrhage | | : | Yes / No |
| | Hypodensity | | : | Yes / No |
| | Thrombosis - | SSS | : | Yes / No |
| | | LS | : | Yes / No |
| | | STS | : | Yes / No |
| MRI | Hemorrhage | | : | Yes / No |
| | Thrombosis | SSS | : | Yes / No |
| | | LS | : | Yes / No |
| | | STS | : | Yes / No |
| | | Cort.Vein | : | Yes / No |

| | | | |
|---|---|---|---|
| T2 Hyper intensity | | : | Yes / No |
| | Superficial | : | Yes / No |
| | Deep | : | Yes / No |
| Diffusion Restriction | | : | Yes / No |

**Treatment**

| | | |
|---|---|---|
| Heparin | : | Yes / No |
| Oral Anticoagulants | : | Yes / No |
| Antiepileptic | : | Yes / No |
| Antiedema | : | Yes / No |
| Surgical decompression | : | Yes / No |

**8 Weeks Follow up**

| | | |
|---|---|---|
| Death | : | Yes / No |
| Focal Neurol deficit | : | Yes / No<br>Hemiparesis / Monoparesis / Paraparesis / Dysphasia / Blindness / CN deficit / Others. |

# ANNEXURE

## ABBREVIATION

| | | |
|---|---|---|
| CVT | - | Cerebral Venous Sinus Thrombosis |
| SSS | - | Superior Sagittal Sinus |
| LS | - | Lateral Sinus |
| STS | - | Straight Sinus |
| FSE | - | Fast Spin Echo |
| GRE | - | Gradient Recalled Echo |
| TOF | - | Time of Flight |
| MRV | - | Magnetic Resonance Venogram |
| MRA | - | Magnetic Resonance Angiogram |
| TR | - | Time for Repitition |
| TE | - | Time to Echo |
| T1WI | - | T1 weighted image |
| ICA | - | Internal Carotid Artery |
| FLAIR | - | Fluid Attenuated Inversion Recovery Sequence |
| NECT | - | Non Enhanced Computed Tomography |
| CECT | - | Contrast Enhanced Computed Tomography |
| MDCT | - | Multi Detector Computed Tomography |
| DSA | - | Digital Subtraction Angiography |
| DWI | - | Diffusion Weighted Images |

# KEY TO MASTER CHART

| | | |
|---|---|---|
| Sex | - | 0 Female<br>1 Male |
| Puer | - | Puerperium |
| HA | - | Headache |
| Alt Sen | - | Altered Sensorium |
| Def | - | Focal neurological deficit |
| HGE | - | Hemorrhage |
| SSS T | - | Superior Sagittal Sinus Thrombosis |
| LS T | - | Lateral Sinus Thrombosis |
| STS T | - | Straight Sinus Thrombosis |
| Hypo | - | Hypodensity |
| Cort VT | - | Cortical Vein Thrombosis |
| T2 Super | - | T2 Superficial hyper intensity |
| T2 Deep | - | T2 Deep hyper intensity |
| Diff Res | - | Diffusion restriction<br>0 - Absent<br>1 - Present |
| Followup | - | 0 - Normal<br>1 - Focal neurological deficit<br>2 - Death |