A STUDY OF CEREBROSPINAL FLUID FLOW DYNAMICS IN PATIENTS WITH CEREBRAL VENOUS THROMBOSIS.

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CERTIFICATE

This is to certify that this Dissertation entitled, **"A STUDY OF CEREBROSPINAL FLUID FLOW DYNAMICS IN PATIENTS WITH CEREBRAL VENOUS THROMBOSIS"** is a bonafide record of work done by **Dr.K.BHARANI** under our guidance and supervision in the Institute of Neurology, Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai, submitted as partial fulfillment for the requirements of D.M. Degree examination Branch I NEUROLOGY, AUGUST 2013, under the Tamil Nadu Dr. M.G.R. Medical University, Chennai.

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DECLARATION

I solemnly declare that this dissertation titled **"A STUDY OF CEREBROSPINAL FLUID FLOW DYNAMICS IN PATIENTS WITH CEREBRAL VENOUS THROMBOSIS"** is done by me in the Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of **Prof. Dr. R.LAKSHMI NARASIMHAN , M.D., D.M.,** Professor of Neurology, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfillment of the university requirements for the award of the degree of D.M. Neurology.

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INSTITUTIONAL ETHICS COMMITTEE
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Telephone No : 044 25305301
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CERTIFICATE OF APPROVAL

To
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Dear Dr. K. Bharani

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "CSF flow dynamics in cerebral venous thrombosis" No.02042012.

The following members of Ethics Committee were present in the meeting held on 19.04.2012 conducted at Madras Medical College, Chennai -3.

1.	Dr. S.K. Rajan. M.D.,FRCP.,DSc	-- Chairperson
2.	Prof. Pregna B. Dolia MD Director , Institute of Biochemistry, MMC, Ch-3	-- Member Secretary
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10.	Tmt. Arnold Soulina MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

INTRODUCTION

Thrombosis of cerebral veins is a distinct cerebrovascular disorder that most often affects young adults and children. Cerebral venous thrombosis is far more common than previously assumed. The spectrum of its clinical presentation is extremely wide and course is highly variable[1].

There are three intracranial compartments namely the brain tissue, blood and CSF that if any pathology involves these will result in intracranial hypertension. Thrombosis of a cerebral venous sinus results in impediment of the sinus blood outflow. This causes increase in CSF pressure and increase in intracerebral venous blood pressure causing brain edema.

With time, probably due to establishment of collateral venous circulation there is resolution of brain edema, cerebral sinus compression and resolution of CSF pressure.

An occlusion of superior saggital sinus may to some degree, obstruct resorption of CSF by the arachnoid villi, leading to increased CSF pressure. But studies done so far find only small disturbance in CSF absorption process with only a minor contribution to intracranial pressure elevation[1, 2].

Hence we propose to study the CSF flow dynamics by bolus saline infusion method and analyse its relation to the various sites of venous sinus thrombosis proven radiologically by MRV.

AIMS AND OBJECTIVES

1. To study the cerebrospinal fluid opening pressure and outflow resistance in patients with cerebral venous sinus thrombosis.
2. To analyse the correlation between Intra Cerebral Pressure and the site of venous sinus thrombosis.
3. To analyse the correlation between clinical symptoms and outcome with the site of sinus thrombosis.

REVIEW OF LITERATURE

Cerebral venous sinus thrombosis is an uncommon condition but over past few years has been diagnosed more often due to greater awareness and availability of modern neuroimaging techniques. It is a challenging condition due to its variability of presentation[1].

All age groups are affected with more common occurrence in females because of pregnancy, oral contraceptive use, puerperium etc. Septic involvement of sinus causing thrombosis was the most common cause previously affecting mainly cavernous sinus and transverse sinus. Over past few decades incidence of aseptic sinus thrombosis has increased with predominant involvement of superior sagittal sinus[2,3].

ETIOLOGY

Predisposing factors may not be identified in upto 20% of patients with cerebral venous thrombosis. A general distinction can be made as infective and noninfective causes. Among the noninfective causes, connective tissue disorders, inflammatory diseases and malignancies are more frequent[4].

Prothrombotic conditions such as protein c and protein S deficiency, Factor V Leiden, prothrombin gene mutation and antithrombin III deficiency may account for 10-20% of cases of venous sinus thrombosis. The co-existence of other predisposing factors with above mentioned Prothrombotic conditions increases the risk of

developing sinus thrombosis. In women, it occurs more frequently during puerperium than pregnancy[4,5].

VENOUS ANATOMY

Cerebral venous system can be divided as

1. Superficial system
2. Deep system

1. Superficial cerebral venous system

This comprises of

1. Mediodorsal group draining into superior sagittal sinus and straight sinus.
2. Laterodorsal group draining into the transverse sinus.
3. Anterior group draining into the cavernous sinus.

The middle cerebral vein is connected to superior sagittal sinus by vein of Trolard and by vein of Labbe to transverse sinus[3].

2. Deep cerebral venous system

The choroidal vein, septal vein and thalamostriate vein join together to form the internal cerebral vein. The internal cerebral veins on either side along with the basal veins of Rosenthal join to form the great vein of Galen, that drains into the straight sinus.

When compared to the superficial venous system,deep system is rather constant[3].

As the cerebral venous sinuses lack valves, blood flow is possible in all directions resulting in spread of infections. They lack tunica muscularis which allows them to remain dilated and have the huge capacity to compensate an occlusion.

They receive blood also from meningeal, emissary and diploic veins resulting in frequent spread of infection from the catchment area.

The direction of blood draining into the superior sagittal sinus is against the blood flow in the sinus, causing turbulence that is further aggravated by the fibrous septa. This fact explains the greater prevalence of superior sagittal sinus thrombosis.

The dural sinus especially the superior sagittal sinus contain most of the arachnoid villi and granulations, the blocking of which will lead to raised intracranial pressure[6].

PATHO-PHYSIOLOGY

There are three intracranial compartments namely brain tissue, CSF and blood-that if pathologically altered can result in raised intracranial pressure[7].

Primary thrombosis of a cerebral sinus causing impediment of the sinus blood outflow will increase the sagittal sinus pressure. The result is

increase of intracerebral venous blood pressure and volume, increase of CSF pressure. Finally there is impediment of cerebral function.

With the establishment of collateral circulation over time, there is resolution of brain edema and reduction of CSF pressure, resulting either in normalization of cerebral function or chronic stage with persistently elevated CSF pressure. Also, the disturbance of CSF absorption at the level of arachnoid villi and granulations can cause decrease of conductance or otherwise an increase of outflow resistance[8].

Pathophysiologically, there are important differences between venous and arterial thrombosis. Venous sinus thrombosis is a continuous process in which a balance between thrombolytic and prothrombotic factors are disturbed leading to extension of thrombosis over time. This slow extension of thrombus with resulting good collateralization of vessels explain the gradual onset of symptoms[8,9].

There must be a large area of transiently and reversibly disturbed cerebral tissue resulting in complete reversibility of their neurological deficit. The elevated venous and capillary pressure can result in hemorrhagic infarction in around 10- 50% of cases[10].

CLINICAL FEATURES

The onset may be acute, subacute or insidious. There are several different clinical constellations, 18-38% present with symptoms resembling benign intracranial hypertension with headache, visual

disturbances and papilloedema; 75% of the cases present with headache and focal neurological deficit; Upto 30-50% of cases may present with seizures and Todd's paralysis. There is strong overlap between all these groups and cases may switch over from one to the other in the course of their illness[6].

DIAGNOSIS

Computed tomography can be normal in 10-20% of patients with proven cerebral venous thrombosis. MRI combined with MRV is the diagnostic modality of choice. However, there are certain drawbacks of this technique which in some cases may make cerebral angiography necessary[11].

One of the commonly encountered problems is the hypoplasia of the anterior aspect of superior sagittal sinus, a normal variant, that can mimic thrombosis. Also, contrast enhancement along the edge of the thrombus can be mistaken for normal contrast material accumulating within a sinus[11,12].

CEREBROSPINAL FLUID DYNAMICS PHYSIOLOGY OF CSF PATHWAYS

A good knowledge of CSF dynamics is needed for understanding the intracranial-intraspinal changes under pathological conditions. CSF dynamics is a fascinating , but less explored field in neurosciences. It

includes all factors concerning CSF formation, circulation and absorption, CSF flow rate, direction and pressure etc[13],..

CSF FORMATION

The main production site of CSF is the choroid plexus in the lateral,third and fourth ventricles. The formation probably takes place in two way,

a. Ultrafiltration of plasma across the endothelial capillary wall,
b. Active transport of sodium and bicarbonate by the choroidal epithelium.

The CSF formation rate is about 0.35ml/min, 20ml/hr at a total of 500ml/day. The movements of the cilia of the ependyma, the respiratory and arterial pulsations, postural changes and the pressure gradient between the ventricular system and the venous side of the sinuses result in a flow of CSF[14].

REGULATION OF ICP/CSF FORMATION

The balance between CSF formation and absorption in holding ICP constant is given by the davson equation:

ICP = FrRout+Pss = Er Rout + Pss

Where Rout is the resistance to CSF outflow, Pss is sagittal sinus pressure, Er is the CSF elimination rate and Fr is the CSF formation rate[14,15].

ICP is normally maintained by the resistance factor of CSF outflow. Distension of spinal meninges, compression of venous vascular structures and venting of CSF constitute the major mechanisms which protect the brain from elevations of ICP. Regulation of CSF volume by means of outflow resistant factor, especially the venous part is the major mechanism for protection of the brain against lethal increases of ICP[16].

The correlation between an increasing volume and the resulting pressure is known as the PV relationship and Marmarou et al introduced the PV index (PVI). They measured the response of CSF pressure to a bolus injection and calculated both compliance and resistance to CSF flow.

$$R_{out} = tPo/PVI[\ \log Pt\ (Pp-Po)/Pp(Pt-Po)$$

Where Pp is the peak pressure, Po is opening pressure, Pt is instantaneous pressure at time 't' on the recovery slope and t is the elapsed time from the instant of injection to the point at which Pt is determined[16,17].

CSF ABSORPTION

CSF is principally absorbed through the villi valves penetrating the sinuses, to some extent in the villi along the spinal roots, and an unknown proportion is absorbed into the brain itself along the brain capillaries, through the ependyma and via the choroid plexus. There is a linear relationship between CSF absorption and ICP[14].

The two main pathways of CSF absorption in the villi are either valve or vacuolar mechanisms. The pressure in the CSF space is higher than the pressure in the sagittal sinus, which is higher than in the torcular area of the sinuses and higher than the pressure in the jugular vein. CSF pressure (ICP) is related to dural sinus pressure by the formula:

Pss = 0.36 ICP + 36 mm H_2O[15,16.]

RESISTANCE TO CSF OUTFLOW

CSF outflow resistance (Rout) is the reciprocal of conductance and reflects the CSF absorption at the arachnoid villi.

The aim is to measure the outflow rate of CSF from the CSF compartment.

The three methods in clinical practice are

1. Infusion test

a. Constant pressure servo controlled infusion method

b. Constant infusion method

c. Constant infusion and constant pressure method

All these techniques are based on identical principles of:

a) Injecting or infusing artificial CSF intrathecally at either a constant rate or constant pressure.

b) Plotting the flow against ICP levels.

The slope of the regression curve is an expression of conductance to CSF outflow (Cout) and the reciprocal is resistance to outflow (Rout). The calculation of Rout implies a constant rate of CSF production and constant CSF and cerebral blood volumes, irrespective of the increases in ICP during the study[18].

All methods give a possibility of CSF leakage,which most often will result in too low Rout values. The interpretation of the pressure increase during the constant rate of lumbar infusion is difficult and unreliable, and the constant pressure servo controlled infusion method is time consuming.

The closed set up of some of the infusion methods makes spontaneous ICP fluctuations unavoidable, while the open system of the lumboventricular perfusion test makes it possible to avoid these vascular reactions. During the bolus injection test, definition of peak pressure, induced unwanted ICP changes and absence of ICP fall after obtained peak pressure are other sources of error[19].

COMPUTERISED INFUSION TEST

By means of computerized analysis of the ICP signal a more exact filtration of normal fluctuations or artefacts is possible.

Czosnyka et al. wrote a program making it possible to measure Rout together with other CSF parameters in a very short time and with an uncomplicated set up.During lumbar infusion with a constant infusion

rate and monitoring of the lumbar pressure, the analogue pressure signal from the pressure amplifier is converted and collected in the program using a standard personal computer.

The ICP signal is processed by spectral analysis, allowing filtration of artifacts. From the non-linear regression curve of ICP during infusion the program computes the static measurement of Rout, Fr and PVI. If steady state is not obtained, or if the infusion rate is changed during the test, it is possible to compute Rout from the non-linear ICP –time regression curve.

Rout values measured by the computerized infusion test compared to values obtained by conventionally measured Rout, in patients representing a broad spectrum from normalcy, normal pressure hydrocephalus, syringomyelia to idiopathic intracranial hypertension, show a good correlation with no variation between Rout values from the two tests.

The computerized infusion test is simple, quick, less invasive than a lumboventricular perfusion test and takes only 30-40 mins, including the lumbar puncture and the calculation. It is also faster than the steady state servo controlled method[17].

LUMBOVENTRICULAR PERFUSION TEST:

The lumboventricular perfusion test employs a ventricular drain inserted through a frontal burr hole and, after 24 hr ICP monitoring, but it increases the risk of complications, especially infection[18].

After the 24 hr baseline ICP has been obtained, Rout is measured with the patient in a lateral position. The cannula in the right lateral ventricle is connected to the pressure monitoring system, and after the lumbar puncture the cannula is connected to an infusion pump.An infusion of ringer's lactate starts at a rate of 0.5-2ml/min and if necessary this may be increased to 4-5ml/min. The outlet of the outflow tube is elevated in steps to increase the ICP and at each level CSF overflow is measured[17].

2.**Radioisotope dilution methods**

Radioisotope introduced intrathecally either by a suboccipital/lumbar route or into the ventricular system may depict production, transport and resorption of CSF. The tracer is followed by a gamma camera both in space and time over 1,6 and 24 hrs.

This method has been widely used to illustrate the anatomical and dynamic conditions of the CSF compartment, and also most recently with MRI, and to select patients with normal pressure hydrocephalus for CSF shunting[17,18].

3.**Bolus injection tests**

The bolus technique is fast and simple, but is based on complicated mathematical computations. A rapid intrathecal injection of a small volume –the bolus, results in an instant increase in pressure in ICP followed by a decline in pressure.

The rise in ICP depends on the compliance of the craniospinal space and the decrease in ICP on both compliance and Rout. From the changes in pressure and the injected volume, Rout can be calculated.

The bolus method seems more valuable in patients with increased ICP (high elastance/low compliance), that is CVST or IIH than in patients with normal pressure hydrocephalus (low elastance/high compliance)[18].

NORMAL Rout VALUES

Ekstedt et al, in a study of Rout using lumbar infusion method found a mean Rout of 9.1 mm Hg /ml/min. Gjerris et al, confirmed that one measurement is sufficient and gives reliable information in patients with disturbances of CSF circulation[17].

REPRODUCIBILITY AND RELIABILITY OF ROUT MEASUREMENT

The results of Rout measurement over more than one examination in 146 patients with normal pressure hydrocephalus and idiopathic

intracranial hypertension in both ventricular and lumbar infusion test are reproducible and reliable.

Borgesan described a variation of not more than 5% during repeated Cout measurement, and the same was found with only a short interval between two Rout measurements.

The Copenhagen group found a high correlation between Rout values obtained from the ventricular and lumbar route. They also demonstrated that atleast 6hrs to elapse after insertion of the ventricular catheter before measurement of Rout is performed and that one measurement is sufficient and gives reliable information in patients with disturbances of CSF circulation[18,19].

MATERIALS AND METHODS

The study was done at Institute of Neurology, Government General Hospital and Madras Medical College, Chennai during the period of September 2010 to January 2013.

Patients admitted in the hospital with the clinical suspicion of cerebral venous sinus thrombosis were included in the study after confirming the diagnosis by MRI with MRV.

Detailed clinical history and examination were recorded. Among this group those patients with general contraindications for lumbar puncture procedure such as bleeding disorders, thrombocytopenia(platelet count <100,000), unequal supratentorial pressure, skin infection at puncture site etc.. were excluded.

All those included in the study were subjected to lumbar puncture and CSF flow dynamics study by modified bolus injection method, the MIN method using bedside saline manometer.

METHOD OF CSF DYNAMICS STUDY:

Bolus injection method modified at the Madras Institute Of Neurology using a simplified bedside saline manometer and standardized by VG Ramesh et al has been used for studying the CSF flow dynamics[20].

Patient is positioned in left lateral position for performing lumbar puncture. The equipment consists of 20G lumbar puncture needle, a

saline stand,measuring tape,3-way adapter, intravenous set and normal saline. The measuring tape is mounted on the saline stand. The intravenous set is filled with normal saline upto 11cms, which in turn is mounted over the saline stand. The zero level should correspond to the spine of the patient in left lateral position.

Now lumbar puncture is performed in the L3-L4 interspace and the needle is connected to the 3-way adapter. The opening pressure is (Po) is noted down after the saline column stabilizes. A volume of normal saline (rV),which is usually 5-10 ml is injected into the intrathecal space at the rate of 1ml/sec, through the three way adapter. After the saline column stabilizes the peak pressure (Pp) is noted down. After the elapse of particular time, which is t in minutes, Pt is recorded. Rout (CSF outflow resistance) is calculated using the Marmarou's Formula, which is as follows,

$$PVI = rV/\log (Pp/Po)$$

$$Rout = tPo/PVI[\log Pt(Pp-Po)/Pp(Pt-Po)] \text{ cms of water/ml/min.}$$

The final value is divided by 1.36 to convert it into mmHg/ml/min[20].

RESULTS AND ANALYSIS

AGEWISE DISTRIBUTION OF CASES

Among the total 30 cases, the minimum age is 14yrs and the maximum age is 47 yrs. Mean age is 31yrs. Hence, our study group is distributed over young adult population as shown in Table 1.

Statistics	Age
N	30
Mean	31.00
Minimum	14
Maximum	47

Table 1.

GENDER DISTRIBUTION

Among the total of 30 cases, 16 were male and 14 were female as shown in Table 2&fig 1.

Gender	Frequency	Percent
Male	16	53.3
Female	14	46.7
Total	30	100.0

Table 2.

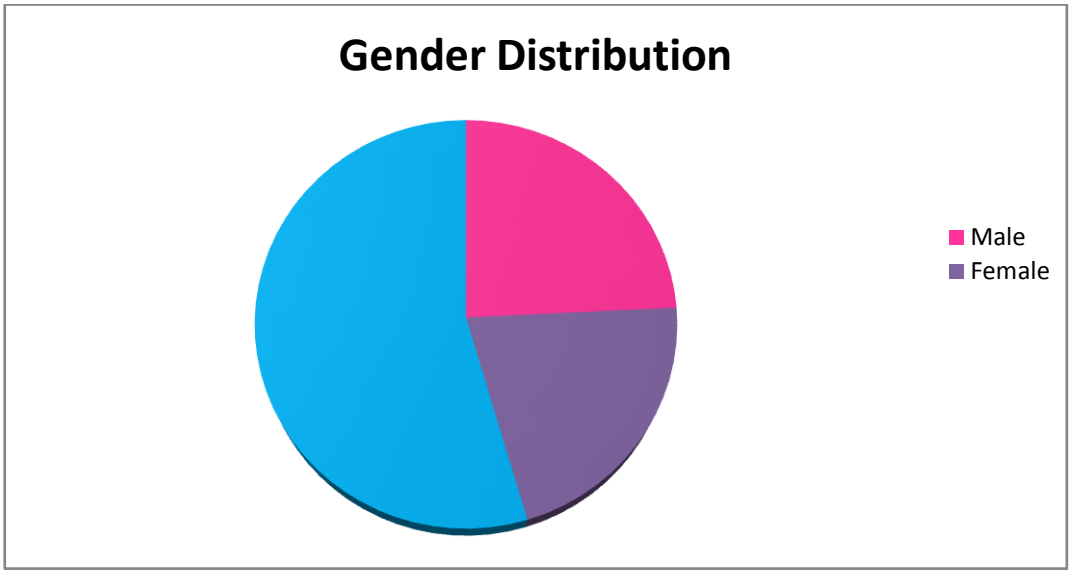

Fig 1.Gender Distribution

FREQUENCY DISTRIBUTION OF SYMPTOMS

Only 13.3% of study patients did not complain of headache, whereas the majority i.e. 86.7% did have headache as the presenting complaint as shown in Table 3.

Headache	Frequency	Percent
Nil	4	13.3
Present	26	86.7
Total	30	100.0

Table 3.

As shown in table 4., 18 cases had papilledema attributing to a majority of 60%. 12 cases did not have papilledema at admission.

Papilledema	Frequency	Percent
Nil	12	40.0
Present	18	60.0
Total	30	100.0

Table 4.

FREQUENCY DISTRIBUTION OF SINUS THROMBOSIS

17 cases had thrombosis involving superior sagittal sinus, which accounts for a total of 56.7%. Transverse sinus is also equally involved with 56.7%, Sigmoid sinus in 30% of cases and Straight Sinus in 10% of cases were involved. These are shown in Table 5,6,7,8 respectively & fig 2..

SSS	Frequency	Percent
No	13	43.3
Yes	17	56.7
Total	30	100.0

Table 5.

Transverse Sinus	Frequency	Percent
No	13	43.3
Yes	17	56.7
Total	30	100.0

Table 6.

Sigmoid Sinus	Frequency	Percent
No	21	70.0
Yes	9	30.0
Total	30	100.0

Table 7.

Straight Sinus	Frequency	Percent
No	27	90.0
Yes	3	10.0
Total	30	100.0

Table 8.

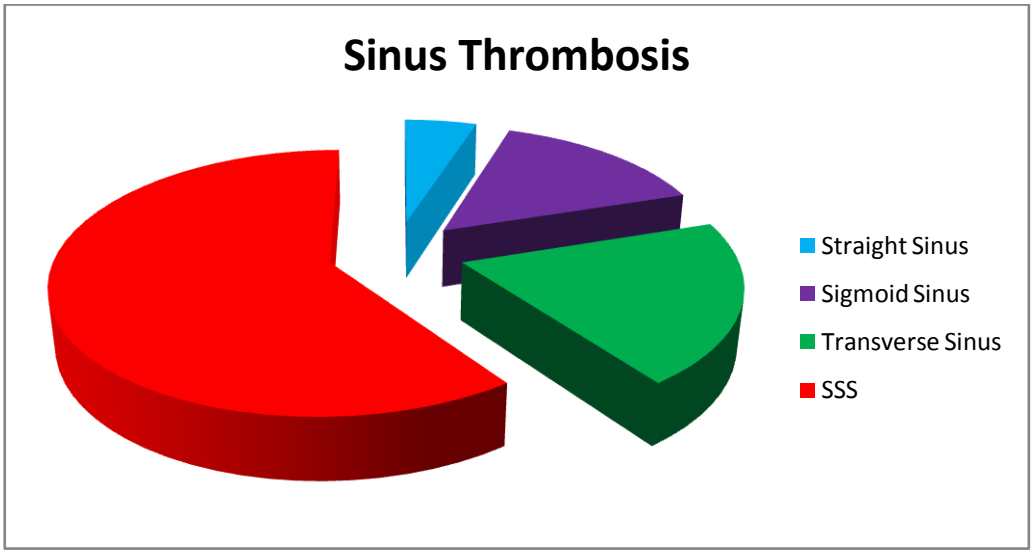

Fig 2. Frequency distribution of sites of sinus thrombosis

CSF PROTEIN

11 cases (36.7%) , had CSF protein level outside the normal range of 40 mg/dl,(Table 9).

CSF protein	Frequency	Percent
< 40 mg/dl	19	63.3
> 40 mg/dl	11	36.7
Total	30	100.0

Table 9.

CSF CELL COUNT

Only one among the 30 cases had an abnormal CSF cell count >5 cells/ dl. The CSF cell count was normal in 96.7% of cases as shown in Table 10.

CSF cell count	Frequency	Percent
≤ 5	29	96.7
> 5	1	3.3
Total	30	100.0

Table 10.

CLINICAL OUTCOME ANALYSIS

14 patients had shown improvement in clinical symptoms, whereas the majority i.e. 16 patients (53.3%) complained of persistent headache.(Table 11)&fig 3.

Outcome	Frequency	Percent
Improved	14	46.7
Persistent Headache	16	53.3
Total	30	100.0

Table11.

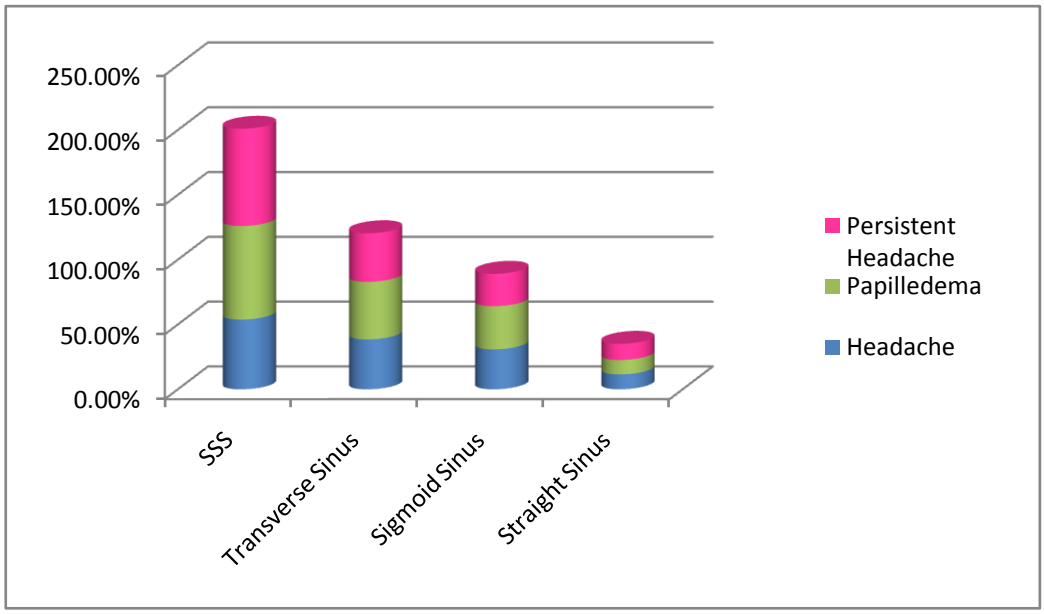

Fig 3. Analysis of sinus thrombosis with head ache, papilledema &clinical outcome .

FREQUENCY DISTRBUTION OF Po AND Rout

As shown in Table 12 and Table 13, the mean opening pressure was 21.90 cm H2O and the mean Rout was 41.83 mm Hg /ml /min. The maximum and minimum Rout measured were 73 mm Hg/ml/min and 6 mm Hg/ml/min respectively.

Statistics	Po (cm H2o)
N	30
Mean	21.90
Minimum	10
Maximum	72

Table 12.

Statistics	Rout (mm Hg)
N	30
Mean	41.83
Minimum	6
Maximum	73

Table 13.

ANALYSIS OF Rout AND Po IN RELATION TO SINUS THROMBOSIS

The independent samples t-test has shown statistical significance for elevated Rout in relation to the site of sinus thrombosis in patients with Superior sagittal sinus (P value = 0.001) and straight sinus (P value = 0.001).This is shown in Table 14,Table 17&fig 4.

However, the level of opening pressure (Po) rise has not shown any statistically significant relation with the site of sinus thrombosis.

Independent samples t-test to compare the mean values

	SSS	N	Mean	Std. Deviation	P-Value
Rout (mm Hg)	No	13	26.27	13.933	0.001
	Yes	17	53.74	18.582	
Po (cm H2o)	No	13	21.46	16.277	0.857
	Yes	17	22.24	6.026	

Table 14.

	Transverse Sinus	N	Mean	Std. Deviation	P-Value
Rout (mm Hg)	No	13	46.12	18.087	0.349
	Yes	17	38.56	23.803	
Po (cm H2o)	No	13	19.46	5.753	0.314
	Yes	17	23.76	14.211	

Table 15.

	Sigmoid Sinus	N	Mean	Std. Deviation	P-Value
Rout (mm Hg)	No	21	39.83	21.013	0.446
	Yes	9	46.50	23.188	
Po (cm H2o)	No	21	23.52	12.632	0.240
	Yes	9	18.11	6.972	

Table 16.

	Straight Sinus	N	Mean	Std. Deviation	P-Value
Rout (mm Hg)	No	27	39.31	21.199	0.001
	Yes	3	64.55	1.928	
Po (cm H2o)	No	27	21.30	11.711	0.393
	Yes	3	27.33	7.095	

Table 17.

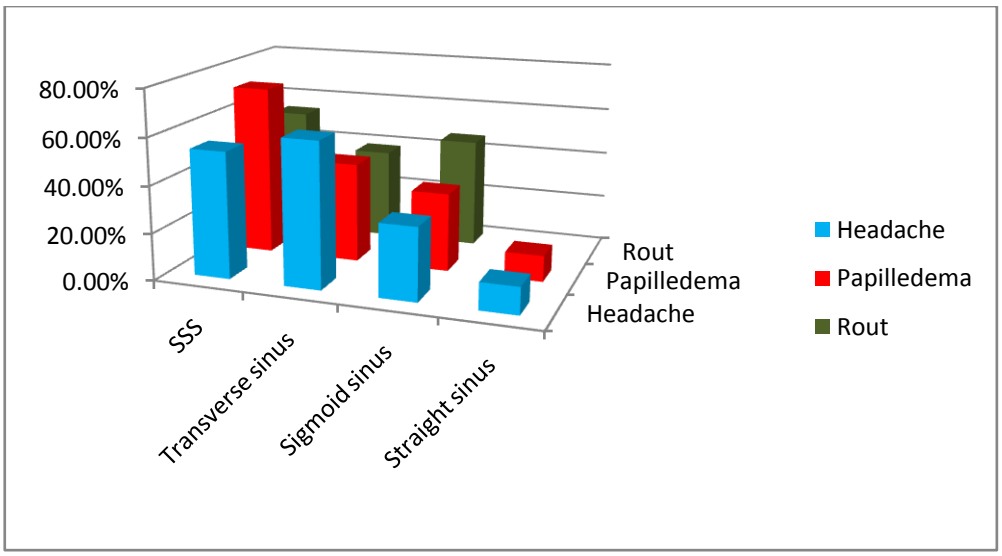

Fig 4.Analysis of sinus thrombosis with Rout, Papilledema & Headache.

ANALYSIS OF Rout AND PO IN RELATION TO CLINICAL SYMPTOMS

The mean Rout in patients with and without headache were 42.65 and 36.55 respectively. The outflow resistance is in a higher range in those presenting with headache, but this has not shown statistical significance (P = 0.606). Table 18.

The mean opening pressure in patients with headache was 22.50, which is in a higher range when compared to those without headache (mean =18.00). However, this is not found to be significant statistically (P=0.472).Table 18

	Headache	N	Mean	Std. Deviation	P Value
Rout (mm Hg)	Nil	4	36.55	21.999	0.606
	Present	26	42.65	21.755	
Po (cm H2o)	Nil	4	18.00	4.967	0.472
	Present	26	22.50	12.034	

Table 18.

The mean Rout (49.35) is also at a higher range in patients with papilledema when compared to those without papilledema (mean Rout = 30.56). This has shown significance statistically also (P = 0.016).Table 19.

The mean Po (25.00) is at a higher range in patients with papilledema than in patients without papilledema (mean Po =17.25). This is significant statistically with a P value of 0.067. Table 19.

	Papilledema	**N**	**Mean**	**Std. Deviation**	**P-Value**
Rout (mm Hg)	Nil	12	30.56	20.247	0.016
	Present	18	49.35	19.345	
Po (cm H2o)	Nil	12	17.25	6.904	0.067
	Present	18	25.00	12.852	

Table 19.

ANALYSIS OF Rout AND Po IN RELATION TO CLINICAL OUTCOME

In patients who complained of persistent headache , the mean Rout is high (49.81) and this is statistically significant (P =0.027).

The mean Po in patients with persistent headache was 20.56 and in those who improved was 23.43.Table 20.

	Outcome	**N**	**Mean**	**Std. Deviation**	**P-Value**
Rout (mm Hg)	Improved	14	32.72	21.339	0.027
	Persistent Headache	16	49.81	18.822	
Po (cm H2o)	Improved	14	23.43	16.190	0.531
	Persistent Headache	16	20.56	4.442	

Table 20.

ANALYSIS OF CLINICAL SYMPTOMS WITH SINUS THROMBOSIS

Among the 17 patients who had SSS thrombosis, 14 complained of headache (P = 0.613). 16 among 17 patients who had transverse sinus thrombosis complained of headache (P = 0.290), 8 out of 9 patients with sigmoid sinus thrombosis had headache (P = 0.999), all the 3 patients with straight sinus involvement had headache (P = 0.999). Tables 21,22,23,24.

Cross Tables

Chi-Square test to compare the proportions

SSS	Headache				Total	
	Nil		Present			
	N	%	N	%	N	%
No	1	25.0	12	46.2	13	43.3
Yes	3	75.0	14	53.8	17	56.7
Total	4	100.0	26	100.0	30	100.0

Chi-Square Tests	P-Value
Fisher's Exact Test	0.613

Table 21.

Transverse Sinus	Headache				Total	
	Nil		Present			
	N	%	N	%	N	%
No	3	75.0	10	38.5	13	43.3
Yes	1	25.0	16	61.5	17	56.7
Total	4	100.0	26	100.0	30	100.0

Chi-Square Tests	P-Value
Fisher's Exact Test	0.290

Table 22.

Sigmoid Sinus	Headache				Total	
	Nil		Present			
	N	%	N	%	N	%
No	3	75.0	18	69.2	21	70.0
Yes	1	25.0	8	30.8	9	30.0
Total	4	100.0	26	100.0	30	100.0

Chi-Square Tests	P-Value
Fisher's Exact Test	0.999

Table 23.

STRAIGHT SINUS	Headache				Total	
	Nil		Present			
	N	%	N	%	N	%
No	4	100.0	23	88.5	27	90.0
Yes	0	.0	3	11.5	3	10.0
Total	4	100.0	26	100.0	30	100.0

Chi-Square Tests	P-Value
Fisher's Exact Test	0.999

Table 24.

72.2% of those with SSS thrombosis had papilledema with significant P value of 0.035.Table 25.

SSS	Papilledema				Total	
	Nil		Present			
	N	%	N	%	N	%
No	8	66.7	5	27.8	13	43.3
Yes	4	33.3	13	72.2	17	56.7
Total	12	100.0	18	100.0	30	100.0

Chi-Square Tests	P-Value
Pearson Chi-Square	0.035

Table 25

44.4% with transverse sinus involvement had papilledema with P=0.098.Table 26.

Transverse Sinus	Papilledema				Total	
	Nil		Present			
	N	%	N	%	N	%
No	3	25.0	10	55.6	13	43.3
Yes	9	75.0	8	44.4	17	56.7
Total	12	100.0	18	100.0	30	100.0

Chi-Square Tests	P-Value
Pearson Chi-Square	0.098

Table 26.

33.3% of those with sigmoid sinus thrombosis (P =0.704), 11.1% with straight sinus involvement had papilledema (P =0.999). Table 27, 28.

SIGMOID SINUS	Papilledema				Total	
	Nil		Present			
	N	%	N	%	N	%
No	9	75.0	12	66.7	21	70.0
Yes	3	25.0	6	33.3	9	30.0
Total	12	100.0	18	100.0	30	100.0

Chi-Square Tests	P-Value
Fisher's Exact Test	0.704

Table 27.

STRAIGHT SINUS	Papilledema				Total	
	Nil		Present			
	N	%	N	%	N	%
No	11	91.7	16	88.9	27	90.0
Yes	1	8.3	2	11.1	3	10.0
Total	12	100.0	18	100.0	30	100.0

Chi-Square Tests	P-Value
Fisher's Exact Test	0.999

Table 28.

ANALYSIS OF RELATION OF SINUS THROMBOSIS WITH CLINICAL OUTCOME

75% of patients with SSS thrombosis complained of persistent headache which has shown statistical significance also (P =0.030).Table 29.

SSS	Outcome				Total	
	Improved		Persistent Headache			
	N	%	N	%	N	%
No	9	64.3	4	25.0	13	43.3
Yes	5	35.7	12	75.0	17	56.7
Total	14	100.0	16	100.0	30	100.0

Chi-Square Tests	P-Value
Pearson Chi-Square	0.030

Table 29.

37.5% of patients with transverse sinus thrombosis had persistent headache with a significant P value of 0.024.Table 30.

TRANSVERSE SINUS	Outcome				Total	
	Improved		Persistent Headache			
	N	%	N	%	N	%
No	3	21.4	10	62.5	13	43.3
Yes	11	78.6	6	37.5	17	56.7
Total	14	100.0	16	100.0	30	100.0

Chi-Square Tests	P-Value
Pearson Chi-Square	0.024

Table 30.

25% of patients with sigmoid sinus thrombosis had persistent headache (P =0.694).Table 31.

12.5% of patients with straight sinus thrombosis had persistent symptoms (P =0.999). Table 32.

SIGMOID SINUS	Outcome				Total	
	Improved		Persistent Headache			
	N	%	N	%	N	%
No	9	64.3	12	75.0	21	70.0
Yes	5	35.7	4	25.0	9	30.0
Total	14	100.0	16	100.0	30	100.0

Chi-Square Tests	P-Value
Fisher's Exact Test	0.694

Table 31.

STRAIGHT SINUS	Outcome				Total	
	Improved		Persistent Headache			
	N	%	N	%	N	%
No	13	92.9	14	87.5	27	90.0
Yes	1	7.1	2	12.5	3	10.0
Total	14	100.0	16	100.0	30	100.0

Chi-Square Tests	P-Value
Fisher's Exact Test	0.999

Table 32.

DISCUSSION

Cerebral venous thrombosis commonly affects young adult population mainly females during pregnancy and puerperium. This condition is being diagnosed more frequently now a days, owing to high clinical suspicion and advent of modern neuroimaging modalities like MRI, MRV and CT Venography.

Early diagnosis and highly active interventional treatment approaches have led to considerable reduction in morbidity and mortality over the past few decades[22,23].

In developing countries like India, a significant change in the causative factor has been observed for past few years. Unlike septic causes, aseptic conditions such as antiphospholipid antibody syndrome, inflammatory diseases, prothrombotic conditions like prothrombin gene mutation, Factor V Leiden, Antithrombin III deficiency,hyperhomocystinemia etc, have become common etiological factors[24,25].

The presence of other secondary risk factors like oral contraceptive use, alcohol consumption and dehydration in persons already affected with prothrombotic mutations lead to increased incidence of CVST. With the availability of improved laboratory facilities, now we are able to find out etiology in more number of patients[26].

The venous sinuses are the main drainage channels for the cerebrospinal fluid,especially the superior sagittal sinus due to plenty of arachnoid villi and granulations. In CVST, there is significant disturbance of CSF flow and absorption because of increasing pressure in the thrombosed sinus. This is a condition with high elastance and low compliance[27].

According to the Munro-Kellie doctrine, this in turn will lead to raised intracranial pressure.This basic pathophysiology is the reason behind the patients clinically presenting with severe headache and papilledema[28,30].

Early diagnosis and treatment helps in lowering the ICP by recanalising the venous sinus. Over time, there will be establishment of collaterals which may also lead to better drainage of CSF[31].

In practical clinical situations, we were able to observe that more often patients who have suffered venous sinus thrombosis visit the outpatient clinic complaining of persistent headache. The persistence of headache is a huge burden for the patient affecting their productive life.

Pathophysiologically, this may be attributed to the permanently altered CSF flow dynamics, the study of which may help us for better understanding of this common clinical problem. Hence we attempted to study the CSF flow dynamics in patients affected with CVST.

We measured the CSF opening pressure by performing lumbar puncture. We used the MIN modified Bolus injection method to measure the Rout (outflow resistance), which is the reciprocal of compliance. Our method is simplified modification of the well accepted Marmarou's Bolus injection test, which can be performed bedside using a saline manometer. This method has been studied and standardized by VG Ramesh et al[20].

Previously in our institute the same method was used in the study by Vidhya et al in patients with NPH (19 patients), Post traumatic hydrocephalus (23 patients) and Post meningitic hydrocephalus (8 patients). They have measured the Rout and shown good prognosis after shunt surgery done based the threshold Rout values.

Mahesh et al from our institute have studied the CSF flow dynamics in patients with IIH (47 cases) and have shown that measurement of Rout along with CSF opening pressure will increase the diagnostic possibility of IIH from 60% to 90%.

Our study is a cross sectional study including 30 patients diagnosed as CVST confirmed with MRI/MRV, for whom we have measured the CSF flow dynamics.

The mean age of the study group is 31 yrs. Among the total 30 cases, 16 were male and 14 were female. 86.7% did complain of headache and 60% had papilledema. 56.7% of the study group had SSS

thrombosis, transverse sinus thrombosis in 56.7%, 30% had sigmoid sinus thrombosis and 10% had straight sinus thrombosis. Of the total 53.3% had persistent headache even after treatment. Among those who complained of persistent headache, 75% had SSS thrombosis (P=0.035).

The mean Rout is 41.83 mm Hg/ml/min and higher range of Rout was present in patients with SSS thrombosis (P=0.001).

The mean Rout in those with persistent headache was in a higher range (49.81mm Hg/ml/min) with P=0.027. 72.2% with SSS thrombosis had papilledema (P=0.035).

Hence, in our study SSS and transverse sinus were commonly affected. The raised ICP symptoms such as headache and papilledema were more common in those with SSS thrombosis. The mean Rout and Po in patients with superior sagittal sinus thrombosis were at a higher range, when compared to other sites of sinus thrombosis. This explains the reduced compliance of CSF drainage in patients with SSS thrombosis leading to altered CSF flow dynamics and in turn raised ICP.

Also,the same group of patients with involvement of SSS thrombosis with high Po and Rout did complain of persistent headache even after medical treatment.

Only few studies have been done so far regarding CSF flow dynamics in CVST, among them the notable one is study by Ekstedt et al[21] in 10 patients with SSS thrombosis. They have studied the CSF

dynamics by constant pressure infusion method and followed up over a period of 15 yrs. A total of 70 dynamic studies were done.

They have concluded that there was a clear increase in intracranial pressure due to rise in pressure of major dural sinus. There was a persistent increase in ICP which declined only gradually. But according to them this had no significant clinical impact as change in CSF absorption played only a minor role in elevating the ICP and none of the patients developed hydrocephalus.

However, in our study we were able to demonstrate that reduced compliance or elevated outflow resistance significantly contributes to the raised ICP especially in those with involvement of SSS thrombosis. Also, we were able to show that this had significant clinical impact over the patient in the form of persistent headache.

Park et al[29], in their study of 'New Concept of cerebrospinal fluid dynamics in cerebral venous sinus thrombosis' have stated that , sagittal sinus differs from other sinuses in that greater impediment of CSF absorption occurs due to presence of arachnoid villi in these sinuses which will contribute to greater rise in ICP, leading to a higher likelihood of ventricular dilatation.

However , they have observed that external CSF drainage did not improve sinus thrombosis in these patients and one should be careful

while performing external CSF drainage in patients with sagittal sinus involvement.

In our study we were able to demonstrate that increased outflow resistance especially in major dural sinus thrombosis is the major contributing factor towards raised intracranial pressure and that higher Rout at diagnosis mostly leads to persistent headache in this group of patients producing significant clinical impact.

However, these patients need to be followed up for a longer duration with repeat CSF dynamic studies and neuroimaging, so that the requirement of additional long term treatment to reduce raised ICP can be recommended.

CONCLUSION

1. The cerebrospinal fluid opening pressure Po and the outflow resistance Rout were in a higher range in patients with cerebral venous sinus thrombosis.
2. The opening pressure Po and outflow resistance Rout were higher in patients with superior sagittal sinus thrombosis when compared to other sites of sinus involvement.
3. The patients with superior sagittal sinus thrombosis had the higher incidence of headache ,papilledema and also did complain of persistent headache even after medical treatment.
4. The increase in Rout suggests that the raised ICP due to reduced compliance will take longer time to normalise inspite of recanalisation secondary to anticoagulant therapy.
5. In view of persistent headache, these patients will require long term medical treatment for raised ICP.
6. For unexplained reasons, these group of patients do not develop hydrocephalus inspite of raised ICP.

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ABBREVIATIONS

CSF	:	Cerebrospinal Fluid
ICP	:	Intracranial Pressure
CVST	:	Cerebral Venous Sinus Thrombosis
MRI	:	Magnetic Resonance Imaging
MRV	:	Magnetic Resonance Venography
Po	:	Opening Pressure
Pp	:	Peak Pressure
PVI	:	Pressure Volume Index
Rout	:	Outflow Resistance
Cout	:	Conductance to Outflow
IIH	:	Idiopathic Intracranial Hypertension
NPH	:	Normal Pressure Hydrocephalus
CT	:	Computed Tomography
MIN	:	Madras Institute Of Neurology

PROFORMA

Name :

Serial No :

Age :

IP No :

Sex :

Address :

DOA :

DOD:

Phone No :

Diagnosis :

DO Dynamics study :

History :

Clinical Finding :

Investigations :

1. Radiology –
    1. CT Brain
    2. MRI Brain with MRA and MRV
    3. CSF Analysis

CSF Flow Dynamics :

P_0 : P_P : P_t : t : rv :

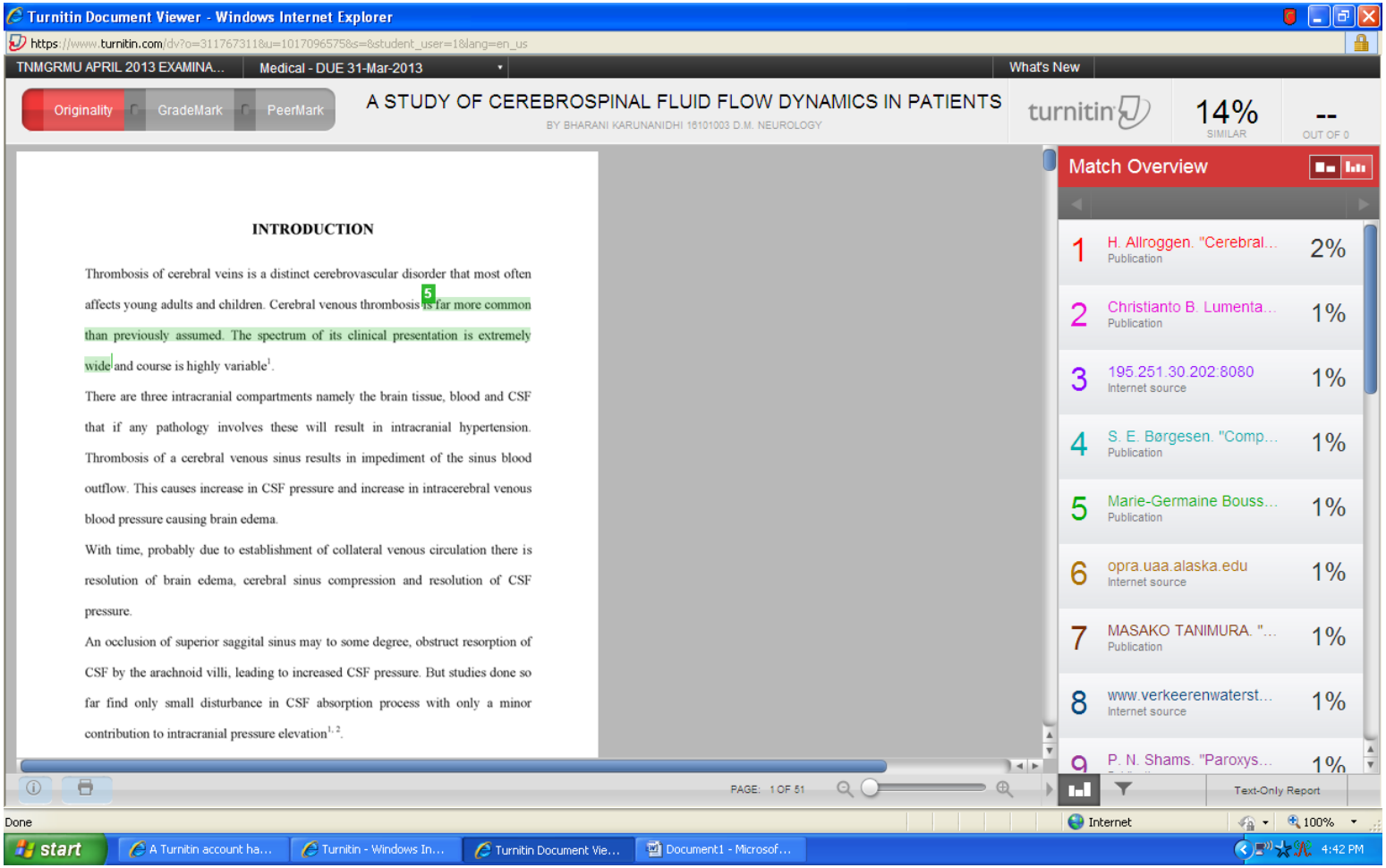
P_{VI} :

R_{out} :

Clinical Outcome :

Serial no.	Age	Sex	Headache	Papilledema	Duration	Clinical features	Po(cm H2o)	PVI	Rout (mm Hg)	MRI Brain With MRV	CSF Protein	CSF Sugar	CSF Cells	Outcome
1	22	F	present	present	1month	vision lose (neurobrucellosis)	72	23.13	11.7	Rt transverse sinus thrombosis	210mg/dl	98mgs/dl	10 lym	improved
2	32	M	present	present	1week	no deficit	23	24.14	54.21	SSS,LT transverse,sigmoid sinus	34mg/dl	53mg/dl	no cells	improved
3	40	F	present	present	10 days	no deficit	12	22.62	49.29	Lt sigmoid sinus thrombosis	72mg/dl	48mg/dl	no cells	persistent headache
4	14	F	present	nil	2weeks	rt ear discharge	10	13.81	34.85	rt sigmoid sinus thrombosis	75mg/dl	44mg/dl	no cells	improved
5	33	M	present	present	1month	b/L lateral rectus palsy,diplopia	21	27.53	73.45	sss,RT transverse,sigmoid sinus	40mg/dl	70mg/dl	no cells	persistent headache
6	27	M	present	present	1month	diplopia	18	34.32	62.45	SSS /rt frontotemporal h'rhage	25mg/dl	74mg/dl	no cells	persistent headache
7	30	M	present	nil	3 days	no deficit	10	24.51	5.86	rt transverse/sigmoid sinus	35mg/dl	55mg/dl	2 lym	improved
8	45	F	present	present	20days	diplopia	31	32.89	43.86	rt transverse/sigmoid sinus	75mg/dl	46mg/dl	5 lym	improved
9	30	M	present	present	5days	Lt hemiparesis/UMN facial palsy	26	22.83	65.89	SSS/straight sinus	40 mg/dl	73mg/dl	no cells	persistent headache
10	32	F	present	present	10days	rt hemiparesis/GTCS	33	24.56	70.89	SSS/rt transverse sinus	24mg/dl	105mg/dl	3lym	improved
11	46	M	present	present	4days	Lt hemiparesis/UMN facial palsy	20	32.32	65.34	SSS/rt transverse/sigmoid sinus	20mg/dl	62mg/dl	no cells	persistent headache
12	32	M	nil	present	10days	rt hemiparesis/GTCS	16	21.76	58.97	SSS/bl frontal h'hagic infarct	40mg/dl	72mg/dl	2lym	improved
13	35	M	present	nil	1month	rt hemiparesis/rt focal seizures	35	31.35	62.34	SSS/rt transverse/straight sinus	45mg/dl	79mg/dl	no cells	improved
14	28	M	present	nil	1month	Lt focal seizure	15	24.67	71.89	SSS/rt transverse/sigmoid sinus	43mg/dl	76mg/dl	no cells	persistent headache
15	47	M	present	present	3months	no deficit	15	23.14	21.67	Rt transverse sinus thrombosis	34mg/dl	89mg/dl	4lym	improved

16	24	F	present	nil	15days	no deficit	15	25.65	18.9	Rt transverse sinus thrombosis	32mg/dl	98mg/dl	no cells	improved
17	34	M	present	present	10days	diplopia	21	32.76	65.42	SSS/straight sinus	45mg/dl	108mg/dl	no cells	persistent headache
18	45	F	nil	present	5days	GTCS	23	25.76	51.78	SSS thrmbosis	40mg/dl	100mg/dl	2lym	persistent headache
19	21	F	present	nil	1month	rt focal seizure	25	31.45	32.65	Lt transverse sinus thrombosis	32mg/dl	105mg/dl	no cells	persistent headache
20	25	M	present	present	15days	no deficit	26	21.32	59.8	SSS thrmbosis	45mg/dl	97mg/dl	no cells	persistent headache
21	32	F	present	nil	1month	rt hemiparesis	18	23.12	23.78	Lt transverse sinus thrombosis	32mg/dl	100mg/dl	2lym	improved
22	43	M	nil	present	12days	GTCS	21	23.56	19.78	LT transverse/sigmoid sinus	42mg/dl	99mg/dl	3lym	improved
23	23	F	present	nil	3months	no deficit	18	21.45	46.76	Rt transverse sinus thrombosis	46mg/dl	87mg/dl	no cells	persistent headache
24	31	F	present	nil	1month	no deficit	17	20.98	21.65	SSS thrmbosis	43mg/dl	76mg/dl	no cells	persistent headache
25	22	F	present	present	3days	no deficit	25	23.56	43.21	SSS thrmbosis	40mg/dl	65mg/dl	no cells	persistent headache
26	34	M	present	nil	15days	rt focal seizure	17	21.21	15.65	Rt transverse sinus thrombosis	42mg/dl	89mg/dl	no cells	improved
27	21	M	present	present	20days	no deficit	21	24.32	17.89	SSS thrmbosis	40mg/dl	90mg/dl	2lym	persistent headache
28	43	M	present	nil	1month	no deficit	15	21.2	16.75	Lt transverse/sigmoid sinus	32mg/dl	102mg/dl	no cells	persistent headache
29	20	F	nil	nil	2days	GTCS	12	25.56	15.67	SSS thrmbosis	25mg/dl	100mg/dl	no cells	improved
30	19	F	present	present	1month	no deficit	26	21.23	52.67	SSS thrmbosis	34mg/dl	98mg/dl	3lym	persistent headache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INTRODUCTION
Thrombosis of cerebral veins is a distinct cerebrovascular disorder that most often affects young adults and children. Cerebral venous thrombosis is far more common than previously assumed. The spectrum of its clinical presentation is extremely wide and course is highly variable[1].
There are three intracranial compartments namely the brain tissue, blood and CSF that if any pathology involves these will result in intracranial hypertension. Thrombosis of a cerebral venous sinus results in impediment of the sinus blood outflow. This causes increase in CSF pressure and increase in intracerebral venous blood pressure causing brain edema.
With time, probably due to establishment of collateral venous circulation there is resolution of brain edema, cerebral sinus compression and resolution of CSF pressure.
An occlusion of superior saggital sinus may to some degree, obstruct resorption of CSF by the arachnoid villi, leading to increased CSF pressure. But studies done so far find only small disturbance in CSF absorption process with only a minor contribution to intracranial pressure elevation[1, 2].
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