Section: Radiodiagnosis

Original Research Article

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

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Abstract

Background: The aim is to evaluate the findings of cerebral venous sinus thrombosis using T1W, T2W, FLAIR, diffusion weighted images, and MR venogram and to evaluate the prognosis by using restriction of diffusion in diffusion weighted images. **Materials and Methods:** It is prospective cohort study of 48 patients with cerebral venous thrombosis diagnosed by MRI and MRV. **Result:** Cerebral venous thrombosis incidence is not uncommon in males. Presence of altered sensorium and focal neurological deficits at admission predicts poor outcome. Puerperium is a risk factor for cerebral venous thrombosis. CT is a good first line investigation for Cerebral venous thrombosis. Presence of intra parenchymal hemorrhage and parenchymal hypodense lesions in CT are associated with poor outcome. MRI features of poor outcome are presence of intra parenchymal haemorrhage, Straight sinus thrombosis, Presence of deep parenchymal T2 hyperintensities, Restriction of diffusion. **Conclusion:** In our study of cerebral venous thrombosis t 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 hyperintensities, intra parenchymal hemorrhage and straight sinus thrombosis are poor prognostic factors.

INTRODUCTION

Acute stroke is one of the major causes 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 Masquerader of diseases. Without CT or MRI many cases would be missed since the clinician is unsuspected of this disease. Nth 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.[1,2] 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.

MATERIALS AND METHODS

Our study is a prospective cohort study of 48 patients carried out at Department of Radiodiagnosis, Chalmeda Anand Rao Institute of Medical Sciences, Karimnagar between December 2020 June 2022. All the 48 patients were subjected to MRI & MRV. MRI was performed using 1.5 Tesla super conducting General Electric HDxt Signa using head coil.

The patient is placed in supine position in the MR gantry with head coil positioned. Multiplanar scout sections obtained for planning the sequences. Whole brain MR. from vertex to the foramen magnum

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including the base of skull are taken using axial coronal and sagittal sections. All the patients presented with CNS signs & symptoms some suspicious of CVT and others with vague features.

Inclusion criteria

All Males and females of all age groups confirmed by MRI & MRV as cerebral venous Sinus thrombosis.

Exclusion Criteria

Patients with MR incompatible devices or implants, Patients on life Support systems, claustrophobia.

Clinical features at admission were analysed concentrating on Headache, Seizure -Focal or generalized, Altered sensorium, coma, Focal neurological deficit Hemiparesis paraparesis, monoparesis, CN palsy, blindness, dysphasia.

MRI Findings

1. Hemorrhage- The age of the parenchymal hemorrhage whether acute. sub-acute, 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, interior Transverse sinuses are noted. Cortical 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 because of failure of membrane Na+ K + ATPase due to decreased ATP since, in ischemia. hypoxia results in decreased ATP production. Due to pump failure Na+ accumulate intracellularly and along within water molecules are trapped intracellularly.

T2 WI cannot differentiate these two types a 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 acro6s a slice as in blood low 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 20 fourier transformation. 20 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 1echnique MR venograms obtained. Motion Compensation gradients, and short echo times educes Signal loss secondary to motion induced phase effect.

Diffusion weighted images

DWI area set of sequences in which an added gradient is applied to the protons apart from the normal slice selection, fr6quency 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 um diameter. In the DWI sequence strong gradient dephasing pulse is first applied. Then after an interval of 40 1o 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 no signal loss The time interval of 40msec is taken since the proton under normal body conditions would travel a distance of about 10 to 15 um 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.

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 ls 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 hypo intensity 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 intra parenchymal in location with surrounding hypodensity The hemorrhage can be due to

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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 vasogenlc edema due to venous Congestion or an infarct. CT cannot differentiate the above two. The hypodense lesions are classically bilateral and subcortical in location.

Follow up 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.

RESULTS

There is no significant association between age, gender of the patients and the prognosis. [Table 1]

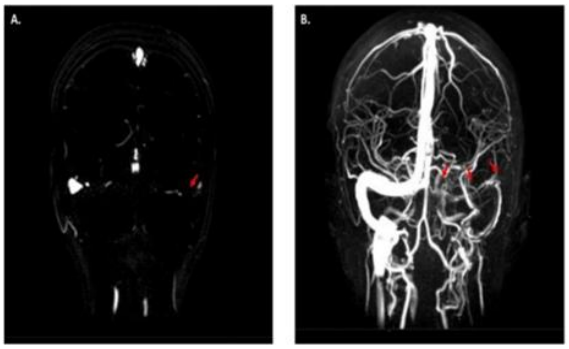

Figure 1: MR venography (MRV image of a patient with acute sinus thrombosis of the left transverse and sigmoid sinus: A) Coronal PC MRV image. B) PC MRV maximum intensity projection (MIP) with absence of flow in the left transverse and sigmoid sinus(arrows).

Out of 35 female patients 27 were puerperal patients. There is no significant association with prognosis. 40(83.3%) patients out of 48 presented with complaints of headache.

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

There is no stastically significant association between headache or seizure with prognosis(p>0.05).

9(18.7%) out of 48 patients presented with altered sensorium. Out of this 9 patients 5 had poor outcome and stastically significant (p<0.01). 6 out of 9 patients who presented with deficit had poor outcome and stastically significant (p<0.01). [Table 2]

Presence of hemorrhage in CT is associated with poor. Outcome. This association is highly significant(p<0.01). There is no stastically significant association of thrombosis in CT with outcome (p>0.05).

66% of patients with hypodensity had poor outcome when compared to 7.7% of patients without hypodensity which has high statistical significance. [Table 3]

20 patients (41.6%) presented with haemorrhage in MRI 8 patients out of this had poor outcome with high significance.

81.3% patients presented with SSS thrombosis, 68.7% presented with TS Thrombosis. No significant association of SSS, TS Thrombosis with poor outcome. 20.8% patients had straight sinus thrombosis. It is significantly associated with poor outcome (<0.001).

There is no significant association of T2 hyper intensity in superficial parenchyma with poor outcome. 9 out of 48 patients had deep parenchymal hyperintensity of which 8 had poor outcome which shows a highly significant association(p<0.01).

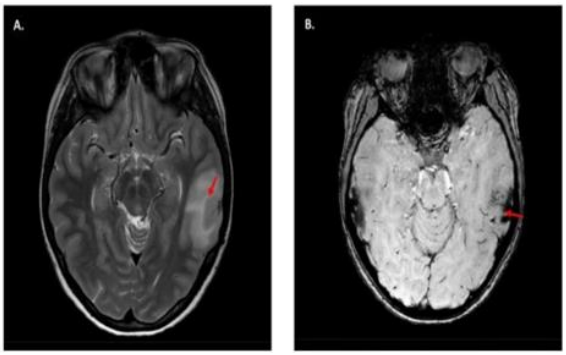

Figure 2: MR images of a patient with left temporal cortical vein thrombosis with venous haemorrhagic infarction. A) T2 weighted image showing a region of increased signal intensity in the cortex and subcortical white matter in the left temporal lobe. B) Susceptibility weighted image showing multiple showing susceptibility artefacts indicating haemorrhagic transformation in the pathological region with pronounced blooming artefacts within the thrombosed cortical veins (arrow).

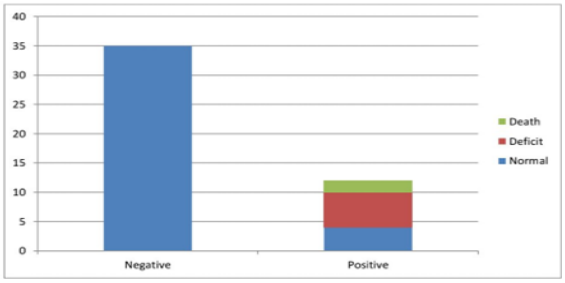

Figure 3: Magnetic resonance imaging- Diffusion Restriction

Table 1: Demographic details in present study.

Follow up	Normal		Deficit		Death		Total	
	Count	%	Count	%	Count	%	Count	%
<25	12	80	3	20	0	0	15	100
25-35	12	80	1	6.7	2	13.3	15	100
35-45	8	80	2	20			10	100
>45	7	87.5	0	0	1	12.5	8	100

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Total	39	81.25	6	12.5	3	6.25	48	100
Gender								
Females	28	72.8	5	83.3	2	66.7	35	72.9
Males	11	28.2	1	16.7	1	33.3	13	27.1

Table 2: Clinical presentation in present study

Follow up	Negative		Positive		Total	
	Count	%	Count	%	Count	%
Puerperium						
Normal	7	87.5	21	77.8	28	80
Deficit			5	18.5	5	14.3
Death	1	12.5	1	3.7	2	5.7
Total	8	100	27	100	35	100
Headache						
Normal	8	100	31	77.5	39	81.3
Deficit			6	15	6	12.5
Death			3	7.5	3	6.3
Total	8	100	40	100	48	100
Seizure						
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	17	100	48	100
Altered sensorium						
Normal	35	89.7	4	44.4	39	81.3
Deficit	4	10.3	2	22.2	6	12.5
Death		0	3	33.3	3	6.3
Total	39	100	9	100	48	100
Neurological deficit						
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	14	100	48	100

Table 3: Computerized tomography in present study.

Follow up	Negative		Positive		Total	
	Count	%	Count	%	Count	%
Parenchymal hemorrhage						
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	13	100	48	100
Superior sagittal sinus thrombosis						
Normal	12	100	27	75	39	81.3
Deficit			6	16.7	6	12.5
Death			3	8.3	3	6.3
Total	12	100	3.6	100	48	100
Transverse thrombosis						
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	2.4	100	48	100
Straight sinus thrombosis						
Normal	37	84.1	2	50	39	81.3
Deficit	5	11.4	1	25	6	12.5
Death	2	4.5	1	25	3	6.3
Total	44	100	4	100	48	100
CT-Hypodensity						
Normal	36	92.3	3	33.3	39	81.3
Deficit	3	7.7	3	33.3	6	12.5
Death	0	0	3	33.3	3	6.3
Total	39	100	9	100	48	100

Table 4: Magnetic resonance imaging in present study.

Follow up	Negative		Positive		Total	
	Count	%	Count	%	Count	%
MRI haemorrhage						
Normal	27	96.4	12	60	39	81.3
Deficit	1	3.6	5	25	6	12.5
Death			3	15	3	6.3
Total	28	100	20	100	48	100

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MRI -superior sagittal sinus thrombosis						
Normal	9	100	30	76.9	39	81.3
Deficit			6	15.4	6	12.5
Death			3	7.7	3	6.3
Total	9	100	39	100	48	100
MRI -Transverse thrombosis						
Normal	12	80	27	81.8	39	81.3
Deficit	3	20	3	9.1	6	12.5
Death			3	9.1	6	6.3
Total	15	100	33	100	48	100
MRI -Straight sinus thrombosis						
Normal	34	89.5	5	50	39	81.3
Deficit	4	10.5	2	20	6	12.5
Death			3	30	3	6.3
Total	38	100	1-	100	48	100

Table 5: Magnetic resonance imaging T2 hyperintensity superficial and deep

Follow up	Negative		Positive		Total	
	Count	%	Count	%	Count	%
MRI- T2 hyperintensity superficial						
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	34	100	48	100
MRI- T2 hyperintensity Deep						
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	9	100	48	100

DISCUSSION

In our study in 48 patents 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 İS not uncommon among males and should be a differential diagnosis in any patient with suspicious CNS signs and symptoms. Sex does not play a role in outcome of CVT in our study. Jayantee Kalita, Varun K Singh et al,[3] conducted a study on total of 160 CVST patients. Their median age was 29.5 years, and 76 (47%) were females. Among the 35 female patients 21 were in puerperal period 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 non-pregnant female.

In the study of Appenzeller et al,[4] pregnancy, puerperium, OCP are good prognostic indicators. In our study the percentages 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.[4,5]

Appenzeller S et al,[4] study showed that twenty four patients who presented with CVT had the following symptoms Headache -75% Vomiting-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%

Al -Multi F, Amuluru K Sahni R et al,[6] conducted a retrospective multicentre cohort study of 13,500 consecutive patients with COVID-19 who were hospitalized between March1 and May 30, 2020 with COVID-19, twelve had Imaging-proved cerebral venous thrombosis with an incidence of 8.8 per 10,000 during 3 months, which is considerably higher than the reported incidence of cerebral venous thrombosis in the general population of 5 per million annually. There was a male preponderance (8 men, 4 women) and an average age of 49 years (95% CI, 36-62 years; range, 17-95 years). The number of patients with parenchymal haemorrhage detected by CT is 13 out of which 7 had poor outcome. his found that this sign is a poor prognostic indicator with P 0.01. According to Ferro, Canhao et al,[5] haemorrhage on admission CT is associated with poor outcome.

The incidences of thrombosis in CT are Superior Sagittal Sinus-75%, Transverse 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 P0.001. CT was normal in 9 patients i.e, 18.7%.

41.6% of patients had Intra parenchymal hemorrhage detected by MRI. 8 patients out of this had poor outcome. That is 8 out of the 9 poor outcome group

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had hemorrhage. This is a statistical significant association. The presence of thrombosis detected by MRl are Superior Sagittal Sinus-81.3%, Transverse Sinus-68.7%, Straight Sinus-20.8%. This corresponded with similar statistics in literature.

The presence of thrombosis in superior sagittal sinus and Transverse sinus is not associated with poor outcome whereas straight sinus thrombosis is associated significantly with poor outcome. Ferro, Canhao et al,[5] 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 Straight sinus thrombosis is seen in 4 patients in CT whereas in MRI in 10 patients showing the greater pickup rate in MRI. the poor outcome of patients with deep cerebral venous thrombosis.

Cumurelue R Crassard I, et al,[7] study shows 17 patents with isolated headache an presenting symptom showed Transverse sinus involvement being most common. Detection of deep T2 hyperlntense lesions has a poor outcome. In our study9 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 T2 hyperintense lesions 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 infraction.

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 ie., 100% sensitive in predicting outcome. But 4 patents who had - restricted diffusion recovered completely ie it is less specific in predicting the outcome. According to Mulin et al,[8] 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.

The Wassay M. Bakshi et al,[9] reviewed 3 cases with CVT. Two patients showed hyperintensity in DWI implying cytotoxic edema and one showed hypointensity showing vasogenic edema. Reversible DWI restricted lesions are shown in the studies of Ducreax, Oppenhium et al Sarma D, Farbe et al.[10,11] Seven patients with CVT and T2 hyperintensities were evaluated with DWI. Five patients had hyperintensity 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.

According to Sachdeva Virender et al,[12] Forty-three patients with subacute (30) chronic (13) CSVT were identified (32 male, 11 females). Median age was 37 years. The presenting complaints were blurred vison 34 (79%), headaches in 25 (58%), vomiting 12 (28%), and diplopia 11 (26%). Eleven patients had associated sixth cranial nerve palsy.

Favrole, Gulchard, Crosslandl et al,[13] patients with CVT were evaluated for diffusion within the clots. It was observed that restricted diffusion within the clot is a predictor for poor recanalization in follow-up scans.

Canhao P; Ferro JM et al,[5] A multinational prospective study including 624 patients with cortical venous thrombosis and evaluation of the causes for death in the acute phase. Mortality rate was 3.4% within 1 month. Causes of death were transtentorial hearniation and multiple lesions. Independent predictors of death were coma, altered sensorium, deep CVT, right sided haemorrhage, and posterior fossa lesions.

Karthikeyan D.S Vijay T Kumar et al,[14] observation showed the presenting symptoms as 76% 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%.

Breteau, Mourier, Vehier et al 3 years follow up of 55 patients showed 45 patients showed no focal neurological deficits and 10 patients either dead or have focal neurological deficits.

CONCLUSION

In our study of cerebral venous thrombosis t 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 hyperintensities, intra parenchymal 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.

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