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Decompressive Hemicraniectomy Versus Medical Management in Cerebral Venous Thrombosis with Signs of Raised Intracranial Pressure

Original Article

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ABSTRACT

Background: *Mass effect is the major cause of death in cortical venous sinus thrombosis and there is no clear cut the role of decompressive hemicraniectomy.* ***Aims:*** *To study the outcome of patients with large venous infarct and acutely increased intracranial pressure subjected to either conservative treatment or decompressive surgery.*
Materials & Methods: *20 consecutive patients admitted with moderate to large venous infarct were examined for features of raised intracranial pressure (ICP) . Cranial CT and or MRI and CT venogram or MR venogram and or cerebral DSA were examined for site of Sino venous occlusion.*
Results: *There were 20 patients with 10 each in conservatively and surgically treated group. Cranial CT/MRI head revealed large venous infarct with midline shift and mass effect in all patients. Overall 15 of 16 survivors had good outcome at 3 months without any significant residual deficit irrespective of mode of treatment used. There is no statistical difference between medical and surgical groups in mRS 1 month, 3 months and death with p value 0.651, 0.185 and 0.474 respectively.*
Conclusion: *Patient with large venous infarct with signs of raised intracranial pressure carries good overall outcome with timely care irrespective of conservative or surgical management.*

Key words: *cerebral venous thrombosis, raised intracranial pressure, decompressive hemicraniectomy.*

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INTRODUCTION

Early reports of cortical venous thrombosis based on angiography have reported mortality of 30-40%.[1] Recent reports have varied rates of 4-33% mortality. [3-9] Major cause of death in cortical venous sinus thrombosis is either transtentorial herniation due to unilateral hemorrhagic infarction or diffuse cerebral edema due to multiple lesions.[9,10] In view of these findings, there is a growing interest in decompressive hemicraniectomy to reduce intracranial tension and herniation. Decompressive surgery for severe neurological condition such as coma, bilateral fixed pupils or bilateral lesions, in 1/3 cases can have a good recovery.[16] However, there is difficulty in identifying ideal time and subset of patients benefiting from decompressive hemicraniectomy. It is indeed a dilemma whether surgery should be undertaken at the time of deterioration in sensorium or performed early in relatively preserved consciousness level with features of raised ICP clinically and radiologically. There are no clear-cut guidelines though decompressive hemicraniectomy is recommended in cortical sinovenous thrombosis.[1,11] Guideline from Stroke American Hearth Association stated that in patient with neurological deterioration due to severe mass effect or intra cranial hemorrhage, decompressive hemicraniectomy may be

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considered but with low evidence (class IIb level of evidence C).[16] A large multicentre prospective study on CSVT has shown low level of consciousness (GCS<9) on admission, seizure on admission, mental state disorder, right sided hemorrhage, posterior fossa lesion on initial scan, deep CVT as independent risk factors related to mortality at 30 days.[13] We have retrospectively analyzed data of patients with large venous hemorrhagic infarct with features of raised ICP admitted in stroke unit of tertiary medical center, Pacific Medical College and Hospital, Udaipur over last 2 years subjected to either surgery or maximal conservative management on a nonrandomized basis.

METHODS

Patients with CT/MRI and MRvenogram and or cerebral DSA proven cortical sinovenous thrombosis having ≥ 3mm midline shift were included. All patients had signs of raised intracranial pressure with modified Rankin scale of 5 (dependent) prior to start of treatment at the admission. Consciousness was assessed by Glasgow coma scale (GCS) and severity of Sino venous thrombosis by Sino venous thrombosis scale (SVT).[6] Outcome at 1 month and 3 months was assessed by modified Rankin scale (mRS) and mortality at any time during 3 months. Pupillary asymmetry, hyperventilation, decerebration, decortication and motor signs on the non-hemiplegic side were taken as surrogate markers of raised ICP. The patients with renal, cardiac, liver failure and malignancy were excluded. The midline shift (MLS) was measured by measuring distance of maximal shift of septum pellucidum. Subfalcian and transtentorial herniation were noted. The patients were subjected to surgery and conservative treatment on a nonrandom basis. Both groups of patients were eligible for both forms of treatments and neurosurgical referral was sought for all the patients. Conservative treatment consists of conventional heparin for 10 days followed by oral anticoagulation to keep INR between 2-2.5, antiedema measure like mannitol, acetazolamide and glycerol as per requirement, anticonvulsant and specific treatment depending upon etiology. All patient underwent routine biochemical workup like blood sugar, urea, serum creatinine, electrolyte, complete hemogram, prothombin time, activated partial thromboplastin time, INR, serum homocysteine, ANA, RA factor, APLA, and VDRL.

RESULTS

There were 10 patients each in conservative and surgery group with no statistical difference in age, gender, mean SVT except for midline shift. (Table no 1). CT brain revealed midline shift of 7.9mm±2.4mmm (range 4-13.3mm) in conservatively treated group and 10.5mm ±2mm (range 7.7mm-13.7mm) in surgically treated group. CT/MR venogram and or cerebral DSA revealed sinovenous occlusion in superior sagittal sinus in 15; lateral sinus right 2, left 4; straight sinus 1; sigmoid sinus right 2, left 4; internal jugular vein in one, vein of Labbe in 1 and cortical vein in 1 patient. Subfalcian herniation were present in all patients while evidence of trans tentorial herniation was present in the entire patient in surgical group while 4 in conservatively treated group.

Effect of therapy: Following conservative treatment in the form of antiedema measures, anticonvulsant and heparin followed by warfarin with an overlap of 5 days and target INR was kept between 2-2.5 in addition to specific treatment as per etiology of the sinovenous thrombosis. All the patients in conservative group were severely disabled, bedridden and required constant nursing and attention (mRS 5). At 1 month 4 were independent (mRS 0-1), 2 were moderately disabled but able to walk without support (mRS 3) while 4 were moderately severe disabled and required support to walk (mRS 4). At 3 months all the patient barring one was independent (mRS 0-1). In 1 patient follow up at 3 months was not available but he could walk with support at 1-month follow-up. There was no mortality in this group. In the surgical group also, all patients were severely disabled, bedridden and required constant nursing and attention (mRS 5), of whom 2 died, 1 was moderately severe disabled and required support to walk (mRS 4) at 1 month, 3 were moderately disabled but able to walk without support (mRS 3) while 4 were independent (mRS 0-1) at 1 month. At 3 months follow up was available in 7 out of the 8 survivors, of whom 6 were independent at 3 months (mRS 0-2) while one required some assistance to walk (mRS 4). There is no statistical difference between medical and surgical groups in mRS 1 month, 3 months and death with p value 0.651, 0.185 and 0.474 respectively (Table 2). Of the two patients died after decompressive hemicraniectomy first had involvement of opposite hemisphere in the form of large hemorrhagic infarct on day 4 and succumbed on next day (Fig 1). Mortality in second patient occurred on day 7 and it was due to development of bilateral occipital infarct due to trans tentorial herniation.

Table 1: Database Medical group and Surgical group

	Medical group (n 10)	Surgical group (n 10)	p value
Age (years)	32.5± 9.5	36.6± 16.8	0.761
Gender (M:F)	3:7	4:6	1.000
Mean GCS	10.9± 3.4	8.3± 3	0.172
Mean SVT	9.8± 3.8	13± 3.9	0.080
Midline shift	7.9± 2.4	10.5± 2.0	0.019

Table 2: Outcome on medical group and surgical group

Outcome	Medical group	Surgical group	p value
mRS at 1month			
0-2	4	4	0.651
3-4	6	4	
5-6	0	2	
mRS at 3 months			
0-2	9	6	0.185
3-4	0	1	
5-6	0	2	
Mortality in 3months	0	2	0.474

Table 3: Dead cause of the two cases

Mortality	Age	Sex	GCS	SVT	MLS (mm)	day	Cause
1.	24	F	5	16	12.3	5	Raised ICP with involvement of opposite side irreversible herniation with established infarct
2.	42	F	6	12	8.5	6	

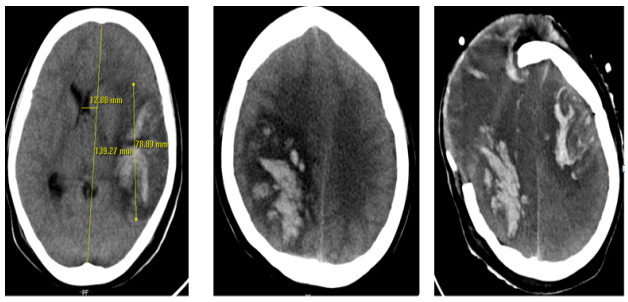

Fig 1: midline shift (MLS) was measured by measuring distance of maximal shift of septum pellucidum.

DISCUSSION

There is lack of randomized control trials comparing maximal conservative treatment and decompressive hemicraniectomy due to many reasons. First is rarity of the disease itself, secondly lack of clear-cut guidelines regarding the timing and mode of treatment in the given situation, third is the response to medical management is usually so good that it seems unethical to subject patients to surgery in view of lack of robust justification in favor of surgery even if there is large hemorrhagic infarct with midline shift and features of raised intracranial pressure. Reason in favour of surgery could be that most of the patients are young in CSVT so surgery might provide better outcome in accordance with a recent study on decompressive hemicraniectomy. This study in large hemispheric arterial infarction revealed that outcome depends on age and not on timing of surgery, side of infarct, pupillary nonreactvity, and midline shift.[14] Decompressive hemicraniectomy was recommended many years ago for sinovenous thrombosis[1] and rarely reported recently.[2,11] As evident in recent trials, the major cause of death in sinovenous thrombosis is commonly transtentorial herniation due to large hemorrhagic infarction or diffuse cerebral edema due to bilateral lesion apart from other causes like pulmonary embolism and sepsis. Presence of hemorrhagic component increases mortality rates up to 69%.[6] In the present study outcome in conservative group was excellent although outcome is also good in surgical patients who survived. Reasons of this difference in outcome can be that decompressive hemicraniectomy mandates withholding anticoagulation, and thus runs the risk of extension of the thrombosis which may result in increased morbidity and mortality, because this is the critical period during which edema exerts its deleterious effects. At the same time this was the period of evolution of thrombosis also and patient was virtually without anticoagulation as anticoagulation is reversed before surgery and not restarted for at least 24-48 hours depending upon surgery. So, patient may remains in sub therapeutic APTT range for 1-2 days postoperatively as anticoagulation is again resumed and target APTT may not be achieved instantly. Therefore patients may be without adequate anticoagulation for a period of 1-4days, and it is well established that adequate anticoagulation is the single most important reason which improved mortality from 30-40%[6] to present level of 3-4% in acute phase.[13] This was the probable reason of death in first patient as he had involvement of opposite hemisphere also postoperatively. His anticoagulation was stopped 6 hours before surgery and restarted 24 hours after surgery and target INR was achieved in next 4 hours. Secondly as vasogenic edema plays a major role in sinovenous thrombosis as compared to cytotoxic edema so aggressive antiedema measures with 3 hourly mannitol 0.25-0.5gm/kg might be more useful compared to arterial infarct and may provide sufficient protection for patient to sustain the critical period of raised intracranial pressure. Thirdly there is no consensus regarding the adequate bone flap removal. Lastly surgery might carry some risk of post-operative complication and sepsis. Second patient died on 7th post-operative day as trans tentorial herniation did not reverse following decompressive surgery. Both patient in mortality group had low GCS (5 and 7), with features of raised intracranial pressure with mid line shift of (12.3mm and 8.5mm) but they had presence of brain stem reflexes preoperatively. Both died in first week (day 5 and 7) of surgery and none had features of sepsis or any other postoperative complication so possible explanation were extension of thrombosis as in first case with involvement of opposite hemisphere postoperatively and due to raised ICP in setting of established bilateral infarct due to trans tentorial herniation (Table no 3) . Contribution of pulmonary embolism in causing increased mortality and morbidity in absence of adequate anticoagulation is also a likely mechanism. All the patients in surgical group had evidence of trans tentorial herniation but 8 out of 10 recovered so presence of this feature alone did not always portray grim outcome. Considering the sample size, and retrospective nature of the study we need to have larger randomized control study to address this issue and arrive at final conclusion. Till that time surgery should probably be used as last resort.

CONCLUSION

Patient with large venous infarct with signs of raised intracranial pressure carries good overall outcome with timely care irrespective of conservative or surgical management.

REFERENCES

1. Krayenbuhl H. Cerebral venous and sinus thrombosis. Clin Neurosurg. 1967;14:1–24.
2. Jourdan C, et al, Evaluation of the clinical benefit of decompression hemicraniectomy in intracranial hypertension not controlled by medical treatment Neurochirurgie 1993, 39:304-310.
3. Bousser MG, Chiras J, Bories J, Castaigne P. Cerebral venous thrombosis:a review of 38 cases. *Stroke*. 1985;16:199 –213.
4. Cantu C, Barinagarrementeria F. Cerebral venous thrombosis associated with pregnancy and puerperium: review of 67 cases. *Stroke*. 1993;24: 1880–1884.
5. deVeber G, Andrew M, Adams C, Bjornson B, Booth F, Buckley DJ, Camfield CS, David M, Humphreys P, Langevin P, MacDonald EA, Gillett J, Meaney B, Shevell M, Sinclair DB, Yager J; Canadian Pediatric Ischemic Stroke Study Group. Cerebral sinovenous thrombosis in childhood. N Engl J Med. 2001;345:417– 423.
6. Einhaupl KM, Masuhr F. Cerebral venous and sinus thrombosis: an update. Eur J Neurol. 1994;1:109 –126.
7. de Bruijn SFTM, de Haan RJ, Stam J; for the Cerebral Venous Sinus Thrombosis Study Group. Clinical features and prognostic factors of cerebral venous sinus thrombosis in a prospective series of 59 patients. J Neurol Neurosurg Psychiatry. 2001;70:105–108.
8. Ferro J, Correia M, Pontes C, Baptista M, Pita F (VENOPORT). Cerebral vein and dural sinus thrombosis in Portugal: 1980–1998. Cerebrovasc Dis. 2001;11:177–182.
9. Ferro JM, Lopes MG, Rosas MJ, Ferro MA, Fontes J; for the Cerebral Venous Thrombosis Portuguese Collaborative Study Group (VENOPORT). Long-term prognosis of cerebral vein and dural sinus thrombosis: results of the VENOPORT Study. Cerebrovasc Dis. 2002; 13:272–278.
10. Canhão P, José M. Ferro, Arne G. Lindgren, Marie-Germaine Bousser, Stam, Fernando. Causes and Predictors of Death in Cerebral Venous Thrombosis. Stroke 2005; 36; 1720-1725
11. Stefini R, Latronico N, Cornali C, Rasulo F, Bollati A. Emergent decompressive craniectomy in patients with fixed dilated pupils due to cerebral venous and dural sinus thrombosis: report of three cases. Neurosurgery. 1999;45:626 –30.
12. Einhaupl KM, Villringer A, Meister W, Mehraein S, Garner C, Pellkofer M, Haberl RL, Pfister HW, Schmiedek P. Heparin treatment in sinus venous thrombosis. Lancet. 1991; 338:597– 600.
13. Ferro JM, Canhao P, Stam J, Bousser MG, Barinagarrementeria F; ISCVT Investigators. Stroke. 2004 Mar; 35(3):664-70.

14. Rabinstein A. A., Mueller-Kronast N., Maramattom B. V., Zazulia A. R., Bamlet W. R., Diringer M. N. and Wijdicks E.F.M..Factors predicting prognosis after decompressive hemicraniectomy for hemispheric infarction Neurology 2006; 67;891-893
15. Ferro JM, et al, Decompressive surgery in cerebrovenous thrombosis, Stroke AHA, 2011,42, 2825-2831
16. Saposnik G, et al, Diagnosis and management of cerebral venous thrombosis, Stroke AHA, 2011