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A STUDY OF LONG TERM PROGNOSIS IN CEREBRAL VENOUS THROMBOSIS

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ABSTRACT

Background and Purpose: Very little is known about the long-term outcome of patients with cerebral venous thrombosis (CVT), particularly regarding the risk of residual epilepsy and further thrombotic events. **Methods**: We retrospectively studied 35 patients with age range between 18 - 37 years who were diagnosed CVT by clinical findings and confirmed by CT scan were followed up for mean of 21.8 months. Information on death, neurological status, seizures, recurrent CVT, other thrombotic events, and subsequent pregnancies was obtained from direct observation and mail.

Results: 26 patients (74.28%) had no neurological sequelae while 9 patients (25.71%) had neurological deficit with 1 of these 9 patients initially presented with isolated intracranial hypertension had blindness due to optic atrophy ,while remaining 8 patients who had focal signs at presentation were left with various cognitive or focal deficits. **Conclusion**: CVT has an essentially good long term prognosis. The frequency of long term epilepsy is low, suggesting that long-term

anticonvulsant treatment is not necessary in the majority of cases. A second

CVT or another thrombotic episode occurred in 12% of patients, stressing the need in a minority of cases for long-term anticoagulation

Keywords; Cerebral venous thrombosis, neurological deficit, prognosis, pregnancy.

INTRODUCTION

Cerebral venous sinus thrombosis (CVT) is a disease with potentially serious consequences, which usually affects young to middle aged people. Bouser ¹ and Einhäupl ² studied the outcome of patients with cerebral venous sinus thrombosis and showed that it may vary from complete recovery to permanent neurological deficits, as a natural course of the disease. Cerebral venous thrombosis for many years has been diagnosed mainly at autopsy. This has led

to the description of a very rare and lethal disease characterized clinically by headache, papilledema, seizures, focal deficits, coma, death and pathologically by haemorrhagic infarction contraindicating the use of anti-coagulants according to Kalbag & Woolf ³, Krayenbuhl ⁴. The introduction and widespread use of cerebral angiography, CT of the brain and more recently MRI ⁵ has made early diagnosis of CVT possible and had completely modified knowledge of this condition ^{6, 7}. Einhäupl² and Brujin⁸ shown that treatment with anticoagulant drugs leads to a moderate benefit for patients with CVT compared with placebo treatment, but the

mortality after treatment with anticoagulant drugs is still 5% to 10%.

Little is known about the long-term outcome of patients with CVT, since there is no study specifically dedicated to the subject. We report the long-term prognosis of 35 patients with CVT.

MATERIAL AND METHODS

Patients who had been diagnosed CVT by detailed history including obstetric history, mode of delivery, onset of symptoms, clinical findings, neurological examination and by CT scan after their discharge from the hospital was followed up. They were specifically asked for presence of any of the following; headache, seizures, sensorimotor and visual disturbances.

Data was also collected regarding recurrence of CVT, deep vein thrombosis,

pulmonary embolism and any other health problem and hospitalization and subsequent pregnancies. Other relevant information regarding socio-economic status, use of oral contraceptives and other medications was also collected.

RESULTS

The main characteristics along with suspected etiology are summarized in table 1. 20 (57.1%) patients received anticoagulants, initially heparin for few days (5 to 7 days) followed by oral anticoagulants for 3 to 4 months unless there was any contraindication for anticoagulation. These patients had haemorrhagic infarcts before treatment, however no worsening was observed after anticoagulation therapy. Anticonvulsant was used only in 20patients who had seizures in the acute stage; anticonvulsants were tapered off gradually after 3 to 4 months unless attacks persisted.

The majority of the patients 26 of 35 had no neurological sequelae during follow up. 9 patients continued to suffer from various degrees

of neurological impairment as summarized in table 2. Seizures were observed only in few patients (6 out of 20) who had seizures and focal signs in the acute stage. These seizures appeared in the first year after CVT.

Regarding the thrombotic risk, 4 patients had recurrence of CVT within 12 months of the first episode. None of the 4 patients with recurrence was on long term anticoagulation.

DISCUSSION

The interval between delivery and onset of symptoms varied with about 90% of them developed symptoms within 7 to 10 days of delivery. This has been attributed to increased platelet adhesiveness during pregnancy and puerperium with peak increase by 10th postpartum day when the incidence of CVT is highest. Focal deficits such as hemiparesis and hemisensory disturbance, seizures, impairment of level of consciousness occur in one third to three quarters of cases.

Predominant CT scan finding in all the patients was haemorhagic infarction. 60% of these patients had superior sagittal sinus thrombosis and other 40% had deep venous system and cortical veins involvement .Haemorrhagic infarction occurred in nearly all patients principally affecting the cortex and adjacent white matter . Viilringer⁹ observed that this is thought to be primarily due to elevated venous and capillary pressure caused by persistence of thrombosis.

Haemoglobin was found to be less than 6.6 gm % in most of the patients, significantly low haemoglobin and packed cell volume are seen in most patients.

26 of 35 patients with CVT recovered with no permanent neurological impairment and 9 patients were left with neurological damage. This confirms that these two main modes of presentation carry a different prognosis regarding the risk of sequelae, as seen by

Bouser¹, Ameri⁶, and Barinagarrementeria¹⁰. No worsening was observed in patients who received anticoagulant agents in the acute stage, as this was not controlled study it was difficult to conclude that the low rate of sequelae observed in the present study is related to the wide use of anticoagulants.

The frequency of epilepsy beyond the acute attack was low in contrast to those in Nagpal RD¹¹ study. However, the low risk of recurrent seizures and the very low risk of late recurrences, it is appropriate to maintain anticonvulsant therapy for a year and to taper off gradually thereafter. If seizure recurs, anticonvulsant should be given on a long term basis.

Heparin is considered the treatment of first choice in patients with CVT by most experts, because it is safe and probably beneficial as also seen in Frey JL¹² study. Nevertheless, it is clear that a substantial number of patients with CVT may recover completely without any treatment ^{1,} Thrombotic events occurred in 9 patients during follow up. When no cause is found, long term anticoagulation is not indicated after the initial CVT. However, if there is a recurrent CVT or another venous thromboembolic event, then long term anticoagulation is preferred. The systemic heparin treatment is not warranted unless there is an underlying thrombophillia or a known cause of venous thrombosis.

CONCLUSION

In conclusion, the long term prognosis of CVT in the study was essentially good. The risk of long standing epilepsy and of CVT recurrence were low in most cases, there is no need for long term anticoagulant or anticonvulsant

treatment. In the absence of known thrombophillia, heparin treatment during further pregnancies does not seem to be required. It should, however be emphasized that this is a retrospective study and that prospective studies

need to better assess the long term prognosis of patients with CVT.

REFERENCES

- 1. Bousser M, Russell RR. Cerebral venous thrombosis. London: WB Saunders, 1997.
- Einhäupl KM, Villringer A, Haberl RL, et al. Clinical spectrum of sinus venous thrombosis. In: Einhäupl KM, Kempski O, Baethmann A, eds. Cerebral sinus thrombosis Experimental and clinical aspects. New York: Plenum Press, 1990; 149-156.
- 3. Kalbag RM, Woolf AL. Cerebral venous thrombosis. London, UK; Oxford University Press; 1967.
- 4. Krayenbuhl HA. Cerebral venous and sinus thrombosis. Clin Neurosurg; 1967; 14; 1 24.
- 5. Thron A, Wessel K, Linden D Scroth G. Superior sagittal sinus thrombosis; neuroradiological evaluation and clinical findings. J Neurol; 1986; 162; 779 785.
- 6. Ameri A, Bousser MG. Cerebral venous thrombosis .Neurol Clin; 1992; 10; 87 111.
- Preter M . Langzietprognose bei Hirnvenen und Sinusthrombosen. Eine Katamnese von 77 patienten. Munich , Grrmany ; Ludwig – Maximillians –university ; 1995 . Doctoral thesis.
- 8. Bruijn de SFTM, Stam J, for the Cerebral Venous Sinus Thrombosis Study Group. Randomised placebo-controlled trial of anticoagulant treatment with low-molecular-weight heparin for cerebral sinus thrombosis. Stroke 1999; 30: 484-488.
- Viilringer A, Mehraen S. Pathophysiological aspects of cerebral sinus venous thrombosis.
 J Neuroradiol; 1994; 21; 72 – 80.
- Barinagarrementeria F, Cantu C, Arredondo H. Aseptic cerebral venous thrombosis;

proposed prognostic scale. J Stroke Cerebrovasc Dis; 1992; 2; 34 –39.

- 11. Nagpal RD. Dural sinus and cerebral venous thrombosis. Neurosurg Rev; 1983; 6; 155 160.
- 12. Frey JL, Muro GJ, McDougall CG, et al. Cerebral venous thrombosis: combined

intrathrombus rtPA and intravenous heparin. Stroke 1999; 30:489-494.

Table-1: Main characteristics of patients of CVT at diagnosis.

Age	18 – 37 years
Onset of symptoms after delivery:	
0 – 48 hours	8 (22.8 %)
48 hrs – 7 days	25 (71.4 %)
After 7 days	2 (5.71 %)
<u>Clinical presentation</u> :	
Focal signs	26 (74.2 %)
Intracranial hypertension	22 (62.8 %)
Seizures	20 (57.10 %)
Altered consciouness	16 (45.7 %)
Suspected causes:	
Anemia	20 (57.1 %)
Infection	9 (25.7%)
Oral contraceptives	0
Unknown	6 (17.1 %)
Heparin treatment	20 (57.1 %)

Table –2: Long term sequelae in patients of CVT.

Sequelae No. of Patients	<u>Initial presentation</u>
No neurological deficit 26 (74.28%) Residual neurological deficit 9 (25.72%)	Headache, vomiting.
a) Optic atrophy & blindness 1	prolonged ICH(months)
b) Seizures alone 6	ICH , hemiparesis ,coma, seizures.
c) Neuropsychiatric disturbances 1 (dementia, behavioural changes, memory loss)	akinetic mutism , hemiparesis
d) Hemispheric deficit 1	Aphasia, hemiparesis.

CVT –Cerebral venous thrombosis, ICH –Intracranial hypertension.