

## Serial Transcranial Doppler Study in Meningitis

R. Gupta, A. K. Mahapatra, and R. Bhatia

Department of Neurosurgery, Neurosciences Centre, All India Institute of Medical Sciences (AIIMS), New Delhi, India

### Summary

Serial transcranial doppler studies were carried out in 12 patients, who developed meningitis during their hospital stay. Blood flow velocities in large basal vessels of the anterior circle of Willis were correlated with CSF pleocytosis and CSF sugar values. Mean blood flow velocities were found to be directly proportional to the CSF white blood cell (WBC) count and were inversely proportional to the CSF sugar values.

Blood flow velocities were higher when CSF WBC count was raised. With only one exception these velocities decreased progressively with a fall in the CSF WBC count. At the time of meningitis there occurred a reduction in CSF sugar values and the blood flow velocities were significantly higher. With increase in CSF sugar values there occurred a gradual fall in the blood flow velocities.

*Keywords:* Blood flow velocities; transcranial Doppler; meningitis; CSF pleocytosis.

### Introduction

Transcranial Doppler sonography (TCD) enables blood flow velocities to be measured in basal cerebral arteries through the intact cranium [1, 9]. Vascular stenosis is of common occurrence in tubercular meningitis [7, 12, 15]. There are also reports of similar changes in the large and small vessels in patients with pyogenic meningitis [3, 6, 9, 13, 16].

Blood flow velocities are inversely proportional to the diameter of the vessel [1, 9]. To the best of our knowledge so far only in two meningitis cases have TCD studies been done [9]. The showed increased blood flow velocities in the ICA and MCA and later a decrease of its velocities when the CSF pleocytosis decreased.

It has been the purpose of this prospective study to get more insight into the dynamics of meningitis induced changes of cerebral blood flow, exploring the usefulness of TCD. It has been carried out in 12 unselected patients who developed meningitis during their

hospital stay in the neurosurgery department. An attempt was made to correlate cerebral blood flow velocities with the CSF pleocytosis and CSF sugar values.

### Material and Methods

This study included 12 patients who had meningitis, 11 had postoperative meningitis within 7 days after operation and 1 patient developed meningitis 7 months after initial operation for CSF rhinorrhoea.

The age ranged from 5 years to 52 years, and there were 9 males and 3 females. In 9 patients surgical intervention was carried out in the posterior fossa, (2 acoustic neurofibroma, 6 vermis gliomas, 1 cerebellar AVM, 1 intra third ventricular craniopharyngioma, 1 parieto-occipital glioma, and a single patient with CSF rhinorrhoea).

Five patients developed meningitis on the 2nd postoperative day, 4 patients on 3rd postoperative day, 1 patients on 6th and one on the 7th day after the operation. Patient no. 2 was operated on for a supra-sellar germinoma; 7 months later he developed CSF rhinorrhoea and meningitis.

Patients in whom the surgery was carried out close to the Circle of Willis were not included in this study to obviate the possibility of development of vasospasm by direct manipulation or the presence of blood in the basal cistern. Also excluded were the patients in whom a fronto-temporal approach was used because postoperative changes in this region could interfere with the insonation of ultra-sonic waves. The diagnosis of meningitis was based on clinical features and CSF examination.

The TCD study was carried out using the EME TC2–64B transcranial Doppler device with a probe of 2 mHz frequency. The Temporal window was chosen because the bone here is thin and the Doppler signals can readily penetrate the window, situated just above the zygomatic arch, between the ear and the orbit. The ultrasound probe is placed over temporal window. Middle cerebral artery flow is towards the transducer and is normally obtained at a depth of 40–50 mm and the ICA is at a depth of 60–65 mm. At the bifurcation of the ICA the flow is often bidirectional. No attempt was made to insonate the basilar or vertebral artery as that requires insonation through the foramen magnum and is difficult in most of

Table 1. Normal Blood Flow Velocity

Artery	Velocity (cms/second)
Middle cerebral artery (MCA)	62 ± 12
Anterior cerebral artery (ACA)	52 ± 12
Posterior cerebral artery (PCA)	42 ± 10
Internal carotid artery (ICA)	54 ± 13
Vertebral artery (VA)	36 ± 9
Basilar artery (BA)	42 ± 10

the patients. Normal blood flow velocity was studied in 10 control cases with no intracranial abnormality and their normal value given in Table 1.

Transcranial Doppler (TCD) evaluation of basal vessels of the anterior circle of Williams was performed at the time of diagnosis of meningitis. Blood flow studies were repeated at 2–3 days intervals, to evaluate the response treatment. CSF examination was repeated during the course of treatment. Three representative readings of blood flow velocities were taken into consideration, (a) at the time of diagnosis of meningitis, (b) during the treatment, (c) at the time of termination of antibiotic treatment.

Vascular-spasm was defined as mean velocities exceeding 120 cms/sec and significant increase as more than 100 cms/sec. Normal velocities ranged from 30–80 cms/sec [1, 7]. Vessels insonated were recognised by (a) depth of insonation, (b) angle of insonation, (c) flow pattern.

## Results

Blood flow velocities were almost similar on the right and left side. The mean velocities were taken for the purpose of this study. In 11 patients the mean blood flow velocities ranged from 60 to 170 cms/sec. Only in one patient (no. 10) the mean blood flow velocities ranged from 32 to 68 cms/sec (Table 2). In 11 patients progressive fall in blood flow velocities was observed with the decrease in CSF WBC count. The exception was patient no. 9, in whom inspite of decrease in the CSF WBC count, mean blood flow velocities remained higher. This patient died subsequently. An inverse relationship was also noticed between CSF sugar values and flow velocities.

## Discussion

The occurrence of intracranial vascular stenosis in meningitis cases is well documented in the literature. But in only two cases the corresponding TCD findings have been reported.

Several reports describe vascular stenosis in large basal cerebral vessels in patients with tuberculous meningitis [2, 8]. Misra *et al.* [17] from India described angiographic findings in tuberculous meningitis: narrowing and occlusion of the proximal por-

tions of anterior cerebral, middle cerebral and in supraclinoid portion of the internal carotid artery. This vascular narrowing was thought to be because of tuberculous vasculitis [8]. Similar findings were also reported by Wadia and Singhal [18], Mathew *et al.* [16]. These authors performed angiography as well as post-mortem studies in some of their cases, and further proved the role of tuberculous arteritis in causation of angiographic changes in arteries.

Polymorphonuclear infiltrations in the sub-intimal region of small arteries and veins were reported by Dodge and Swartz [5]. Certain unusual angiographic changes were observed in patients with purulent meningitis, which included arterial stenosis, occlusion and collateral blood supply. The striking feature of all types of subacute and chronic meningeal infection is the subintimal cellular infiltration of arteries. This leads to marked intimal hyperplasia rich in cells. This may lead to severe stenosis or even complete occlusion of the vessels [4, 8, 10, 14]. Lyons and Leeds [14] proposed that the exudate may surround the arteries and also invade the blood vessel wall.

There is only one single report, dealing with TCD findings in two patients with meningitis [9]. In these two cases repeated blood flow velocity measurements were carried out. In the first case CSF showed 300 WBC. Simultaneous TCD observation showed marked rise in flow velocity. The flow velocities remained high in MCA up to 70 days and in ICA up to 140 days. The second patient had a relatively less severe meningitis. TCD showed flow velocity had increased three-fold, however, flow velocity started decreasing on 10th day and came back to normal in the 4th week.

In the present study there was an increase in blood flow velocities in all but one case. The increase in mean blood flow velocities could be explained by (a) vascular stenosis because of thickening of vessel wall, (b) vasospasm as a result of deposition of exudates over the large basal vessels, (c) a combinations of both factors.

A direct relationship was observed between mean flow velocity and CSF pleocytosis. In eleven patients there occurred a decrease in flow velocities as the CSF WBC count came down. Only patient no. 9 continued to show increase in flow velocities despite a decrease in WBC counts. A possible explanation for this could be that this patient continued to have raised intracranial pressure because of postoperative brain oedema and presence of residual tumour.

Table 2. Serial Transcranial Doppler Study

Sr. no.	Name, age, and sex	Diagnosis	Time interval between operation & meningitis	CSF Examination			Lt. ICA			Lt. MCA			Rt. ICA			Rt. MCA		
				RBC	WBC	Sugar	+	++	+++	+	++	+++	+	++	+++	+	++	+++
1.	M. A., 18/M	Vermis glioma	6 d	Full	600	32/106	200	94	0.99	158	86	0.92	200	114	1.18	176	92	1.0
				0	70	70/100	128	76	0.99	150	90	0.90	126	72	0.90	160	72	0.99
				600	50	72	144	86	0.90	158	86	0.92	134	84	0.87	138	82	0.85
2.	P. S., 46/M	Postop. case of supra- sellar CSF rhinorrhea	7 m	250	1270	18	156	114	1.01	214	128	0.98	172	86	1.05	176	92	1.0
				5	80	26	126	72	0.99	200	94	0.92	150	90	0.90	158	86	0.92
				15	55	22	128	76	0.99	134	84	0.87	126	72	0.90	138	82	0.85
3.	S. K., 8/M	AVM superior surface Rt. cerebellum	2 d	403	1940	64	214	162	0.49	200	146	0.56	130	98	0.55	136	102	0.57
				1650	750	32	192	144	0.47	144	102	0.52	122	100	0.38	126	102	0.38
				Full	40	70	180	132	0.49	156	116	0.59	142	96	0.72	146	106	0.65
4.	M. K., 12/M	Vermis glioma	2 d	Full	3500	73	220	170	0.39	208	156	0.50	126	92	0.53	142	100	0.48
				Full	10	60	192	144	0.47	144	102	0.52	122	100	0.38	126	102	0.38
				3200	0	55	180	132	0.49	156	116	0.59	142	96	0.27	146	106	0.65
5.	K. A., 9/M	Vermis glioma	3 d	Full	9980	28	220	170	0.39	208	156	0.50	126	92	0.53	142	100	0.48
				Full	1800	15	214	162	0.49	200	146	0.56	130	98	0.55	136	102	0.57
				-	-	-	192	144	0.47	144	102	0.52	122	100	0.38	126	102	0.38
6.	S. D., 30/F	Vermis glioma	3 d	Full	7000	12/276	176	130	0.57	174	124	0.78	142	100	0.60	154	112	0.54
				Full	70	32/102	176	138	0.53	200	156	0.47	192	146	0.41	206	166	0.40
				30	30	36	112	86	0.45	104	78	0.46	106	78	0.50	120	86	0.45
7.	R. K., 52/M	Lt. acoustic neuro- fibroma	7 d	75	233	19	130	78	0.99	120	72	1.01	118	66	0.98	118	78	0.85
				20	30	32/85	102	60	0.89	106	58	0.94	108	64	0.90	96	56	0.89
				0	0	40/100	102	76	0.65	100	42	0.98	126	72	0.90	92	72	0.53
8.	M. K., 5/M	Vermis glioma	2 d	2400	450	35	162	118	0.56	184	142	0.58	166	122	0.55	170	128	0.47
				160	70	36	162	128	0.52	170	132	0.46	166	126	0.48	164	120	0.50
				-	-	-	102	76	0.65	98	72	0.46	98	72	0.53	92	72	0.53
9.	N. R., 20/F	Rt. parieto- occipital malignant mixed glioma	3 d	320	1900	14	172	142	0.42	128	90	0.81	100	50	0.73	140	120	0.55
				40	4800	5	202	150	0.52	214	128	0.98	138	92	0.90	154	98	0.91
				10	70	29	214	146	0.56	208	156	0.50	144	86	0.90	158	86	0.92
10.	S., 35/M	3rd ventri- cular cranio- pharyngioma	2 d	Full	900	74/213	110	48	1.33	100	48	1.67	92	48	1.56	88	44	1.19
				1420	300	26/65	100	42	0.98	102	44	1.73	80	38	1.43	80	38	1.43
				40	0	47/89	108	64	0.82	100	42	0.98	96	68	0.62	60	32	0.74
11.	N. K., 12/F	Vermis glioma	2 d	Full	3100	12	162	118	0.56	184	142	0.58	166	122	0.55	206	166	0.40
				400	300	103	162	128	0.52	170	132	0.46	166	126	0.48	200	158	0.42
				6500	45	42	176	138	0.53	174	124	0.78	170	140	0.43	170	128	0.47
12.	F., 25/M	Lt. acoustic neurofibroma	3 d	3500	600	36/130	220	170	0.39	208	156	0.38	192	146	0.41	206	106	0.40
				530	150	91/118	214	162	0.49	200	146	0.56	142	100	0.60	154	122	0.54
				150	10	88	192	144	0.47	156	116	0.59	106	78	0.50	120	86	0.45

+ peak, ++ mean, +++ P. I.

A good correlation was also observed between mean blood flow velocities and CSF sugar values. With an increase in CSF sugar value there occurred a decrease in the blood flow velocities.

Serial measurements of mean blood flow velocities by TCD devices can indirectly suggest vessel diameter, which may vary with the CSF pleocytosis. This noninvasive method can be utilized in patients with meningitis to evaluate the response of anti-meningitic treatment. If interpreted correctly this may obviate the need for repeated CSF examinations.

## References

1. Aaslid R, *et al* (1984) Evaluation of cerebrovascular spasm with transcranial Doppler ultrasound. *J Neurosurg* 60: 37–41
2. Davis DO, Tavera JM: Radiological aspect of inflammatory conditions affecting central nervous system. In: Mosberg WH Jr (ed) *Clinical neurosurgery*, vol 14. Williams and Wilkins, Baltimore, pp 192–210
3. Davis DO, Dilenge D (1970) Arterial dilation in purulent meningitis – a case report. *J Neurosurg* 32: 112–115
4. Dewitt LD, Lawrence RW (1988) Transcranial Doppler. *Stroke* 19: 915–921
5. Dodge P, Swartz M (1965) Bacterial meningitis. A review of selected aspects. *NEJM* 272: 1003–1033
6. Erris EJ, Rudikoff JC, Shapiro JH (1968) Cerebral angiography of bacterial infection. *Radiology* 90: 727–734
7. Gilsbach J *et al* (1987) Cerebral vascular spasm in aneurysm, surgery and its clinical significance. *Progr Clin Neuro Sci* 1: 125–133
8. Greitz T (1964) Angiography in tuberculous meningitis. *Acta Radiol* 2: 369–378
9. Harders A (1986) Neurosurgical application of transcranial doppler sonography. Springer, Wien New York, pp 111–114
10. James AD Jr, Hodges PJ, Jordan CE, Mathews EH (1972) Angiography and cisternography in acute meningitis due to Hemophilus influenzae. *Radiology* 103: 601–606
11. Kirkham FJ, *et al* (1988) Transcranial measurement of blood velocities in the basal cerebral arteries using pulsed Doppler ultrasound: velocity as an index of flow. *Ultrasound Med Biol* 12: 15–21
12. Leads NE, Goldberg HI (1971) Angiographic manifestations in cerebral inflammatory disease. *Radiology* 98: 595–604
13. Lehrer H (1966) Angiographic triad in tuberculous meningitis. *Radiology* 87: 829–835
14. Lyons EL, Leeds NE (1967) Angiographic demonstration of arterial vascular diseases in purulent meningitis. *Radiology* 88: 935–938
15. Lindegaard PK, Bakke Soren J, Aaslid R (1986) Doppler diagnosis of intracranial artery occlusive disorder. *Neurol Neurosurg Psychiat* 49: 510–518
16. Methew HT, Abraham J, Chandy J (1970) Cerebral angiographic features in tuberculous meningitis. *Neurology* 20: 1015–1023
17. Misra PK, Agrawal MP, Sharma NL, Chhabra DK, Dave VS (1973) Cerebral angiography in tuberculous meningitis. *Ind Paediatr* 10: 613–618
18. Wadia NH, Singhal BS (1967) Cerebral angiography in tuberculous meningitis. *Neurology (India)* 15: 127–132

Correspondence: Dr. A. K. Mahapatra, Department of Neurosurgery, All India Institute of Medical Sciences (AIIMS), Ansari Nagar, New Delhi, 110029, India.