Received: 2.2.2006 **Accepted: 2.8.2006**

Original Article

Severe head injuries and intracranial pressure monitoring outcome in Southern Iran

Majid Reza Farrokhi, Mousa Taghipour**

Abstract

BACKGROUND: Head injury is still a major cause of death and disability. Despite advances in intensive monitoring and clinical practice, little data is available to show the predictive value of intracranial pressure monitoring in assessment of the outcome of head injuries. This study was undertaken to evaluate this predictive value and is the first Iranian study in which ICP monitoring has been included.

METHODS: In a prospective study from September 1999 to September 2003, all head- injured patients (53 patients) with GCS of 4-8 who were admitted to Nemazee Hospital of Shiraz University of Medical Sciences were included in this study. Subarachnoid screw method or ventricular catheter via ventriculostomy was used to determine intracranial pressure. Patients were monitored for 3 days and were followed for two years at 6-month intervals.

RESULTS: Car accidents were the most common cause of head injury (43.3%) and 43.3% of patients had GCS of 8. Sixty percent of patients had abnormal intracranial pressure. The patients were most commonly in their first decade of life (18.8%) and 81% of patients were male. Controlling increased intracranial pressure was successful in 60% of patients and resulted in a decrease of mortality rate from 60% to 15%.

CONCLUSIONS: Early treatment of increased intracranial pressure in head injury patients would be beneficial in reducing mortality and morbidity rates.

KEY WORDS: Southern Iran, head injury, outcome, intracranial pressure.

JRMS 2006; 11(4): 248-251

Head injury remains one of the leading causes of death and disability in children [1,2], while significant progress in prognosis of brain injury has been achieved over the last 20 years [2]. Intracranial pressure (ICP) monitoring still plays a key role in the management of patients at risk of severe head injuries [3]. ICP is considered an effective downstream pressure of the cerebral circulation [4] and its rise in head-injured patients subsequent to uncontrollable brain swelling is the only and the most frequent cause of death [5]. Hence, optimizing ICP is important in the management of severe traumatic brain injuries [6] and the data obtained from ICP monitoring must be interpreted carefully and the patients should receive constant and competent medical attention [7]. The first recorded observation of clinical significance of patients with elevated ICP seems to have taken place in Egypt [8]. Increased ICP led to changes in blood pressure, heart rate, and respiration and served as an index of the degree of intracranial hypertension [9].

Half of the patients who had suffered head injuries and had a Glasgow Coma Scaling (GCS) score of 7 needed monitoring due to elevation of ICP [8]. Several devices are applied for ICP monitoring including noninvasive extracranial instruments, as well as intraventricular instruments [10].

This study was performed to evaluate the

*Department of Neurosurgery, School of Medicine, Shiraz University of Medical Sciences, Shiraz, Iran
Correspondence to: Dr Majid Reza Farrokhi, Assistant Professor, Department of Neurosurgery, School of Medicine, Shiraz University of Medical Sciences, Shiraz, Iran. e-mail: farokhim@sums.ac.ir

predictive value of ICP monitoring in outcome assessment of severe head injuries using the subarachnoid screw method and ventricular catheter via ventriculostomy.

Methods

In a prospective study from September 1999 to September 2003, all head-injured patients (53 cases) with GCS of 4-8 who were admitted to Nemazee Hospital of Shiraz University of Medical Sciences as a multitrauma center were entered in this study. Subarachnoid screw method or ventricular catheter via ventriculostomy was used to monitor ICP. Monitoring was performed using Richmond screws in 40 patients and ventricular catheter in 13. After ICP monitoring in the operating room, the patients were transferred to ICU and monitoring was continued for 3 days while an ICP of 15 mmHg was considered normal. In case of 15-25 mmHg, the head was elevated up to 30°, the patients were hyperventilated; if agitated they were sedated and body temperature was decreased if elevated. In case of ICP between 25 and 30 mmHg, hyperventilation was performed to reach $PaCo_2$ of 25-30 mmHg using osmotic agents such as mannitol with a loading dose of 1 g/kg and maintenance dose of 0.25 mg/kg for 24 hours. CSF drainage was performed when possible. Finally, in cases of ICP exceeding 30 mmHg, barbiturates were administered with a loading dose of 3-5 mg/kg intravenously and maintenance dose of 1.5 mg/kg/min for 24 hours. Once discharged, the patients were monitored at 6-month intervals for two years. In the emergency room, all patients were classified neurologically based on GCS score. Also, their brain stem reflexes such as light, corneal and Doll's eye status were checked. Radiological studies including skull X-rays and CT scanning were performed after stabilization of the patients' condition. The patients were examined for any neurological defects such as weakness, speech disorders, and extent of post-traumatic amnesia. They were also classified by Glasgow Outcome Score (GOS) after one year.

Results

Ten patients suffered from multiple trauma and the most common age range was the first decade of life (18.8%). Forty-three patients were male and the rest were female. Car accidents were the most common cause of head injury (54.6%) while motorcycle accidents (20.7%), falling (15%), bicycle accidents (1.8%) and assaults (1.8%) were the other causes in the mentioned order. Eight percent of patients had abnormal stem reflexes, while 9.4%, 11.3% and 15% had GCS scores of 4, 5 and 6, respectively. GCS scores of 7 and 8 were observed in 20.7% and 43.3% of patients, respectively. Radiological studies showed that 30.1% of patients had skull fractures (linear and basilar). The most common finding in CT scan was small tearing hemorrhages in various sites of the brain (cortex, white matter, centrum semiovale, and brain stem) which was secondary to diffuse axonal injury (26.4%). Intracranial hematoma including subdural (23.2%), epidural (7.5%), intracerebral (3.7%) and intracerebellar contusion (9.4%) was also noticed. In 9.4% of patients, CT scan was normal without any evidence of hemorrhage, tight ventricles or closed basal cisterns. In 5.6% of cases, the ventricles were squeezed and the basal cisterns were partially closed with no evidence of mass lesion, hematoma or visible brain tissue (tight brain). Fifteen percent suffered from tight brain with contusion or hemorrhage. Intraventricular hematoma, intracerebellar hematoma, tissue tear hemorrhage, and mixed conditions were detected in 11.3%, 0.9%, 26.4% and 3.7% of patients, respectively. Forty-five percent of patients had normal ICP while ICP of 15-25, 26-30 and greater than 30 mmHg were demonstrated in 32%, 18.8% and 3.7% of cases, respectively. Mortality rate in patients with ICP less than 25 mmHg, 25-30 mmHg, and more than 30 mmHg was 25%, 50% and 25%, respectively, while the overall mortality rate measured 5%. Causes of mortality were reported to be CNS problems (50%) including brain edema, dry light brain, and central herniation, and medical problems (50%) including sepsis and gastrointestinal bleeding. Two pa-

tients with ICP>30 mmHg were refractory to usual management of elevated ICP and received pentobarbital.

CT scan was normal in 9.3% of the patients, but 20% had normal CT scan with high ICP. ICP monitoring in 11 patients with tight brain showed normal ICP (27%). Normal CT scan was a risk factor of high ICP in 20% and tight brain was the risk factor of ICP in 73% of patients.

Good recovery was seen in 71% of patients. GOS after one year follow-up for classes I, II, III, IV and V was 27%, 43.8%, 6.3%, 6.3% and 16.6% respectively, while 9.4% of patients were missed during follow-ups. The correlation between GOS and ICP after one-year follow-up is shown in table 1.

Table 1. The correlation between GOS and ICP after one year follow up in severe head-injured patients

GOS	ICP	15-25 mm Hg	25-30 mm Hg	>30 mm Hg	Total
I	7	3	3	-	13
II	12	7	2	-	21
III	-	2	1	-	3
IV	1	1	1	-	3
V	2	2	2	2	8
Total	22	15	9	2	48

Discussion

Traumatic brain injury is considered as the most common cause of intracranial hypertension [11], while the functional status of the CNS correlates poorly with the level of ICP [12]. The simplest and cheapest way to monitor ICP is application of Richmond subdural screw which was used in 40 patients in this study and showed that 55-60% of cases had closed severe head injuries with elevated ICP. Our results were in agreement with several studies [3,9,13-18]. Patients with normal CT scan may be at risk of high ICP (15-20%) [16]. Hence, severe head-injured patients whose initial CT scan is normal and not suggestive of mass lesion, midline shift, or abnormal cisterns, remain at risk of developing significant secondary cerebral insults due to elevation of ICP [19]. We treated the patients with ICP more than 15 mmHg similar to Marshal, 1979 and Levin, 1977 [20,21].

Our results showed a mortality rate of 15%, indicating a decrease in mortality rate with early management and ICP monitoring [16].

As with monitoring devices, there are advantages and disadvantages as well as cost issues in developing countries such as Iran. The simplest and most economical device with the least infection potential is probably the subdural screw or ventricular catheter which guides decisions and prevents treatment delays. Tamaki et al (2004) showed that increased ICP is associated with increase in cardiac index and systemic vascular resistance, explaining circulatory disturbances in patients with severe head injuries [22]. Thus, ICP monitoring may differentiate patients with poor clinical outcome from those with good prognosis; this underlines the importance of primary injuries and the secondary role of intensive care management in determining the final outcome.

Acknowledgment

The authors would like to thank Dr. Davood Mehrabani for editorial assistance.

References

1. Morenski JD, Tobias JD, Jimenez DF. **Recombinant activated factor VII for cerebral injury-induced coagulopathy in pediatric patients. Report of three cases and review of the literature**. *J Neurosurg* 2003; 98(3):611-616.
2. Vigue B. **[Update on medical management of severe head trauma]**. *Neurochirurgie* 2003; 49(6):583-594.
3. Stocchetti N, Longhi L, Magnoni S, Roncati ZE, Canavesi K. **Head injury, subarachnoid hemorrhage and intracranial pressure monitoring in Italy**. *Acta Neurochir (Wien)* 2003; 145(9):761-765.
4. Buhre W, Heinzel FR, Grund S, Sonntag H, Weyland A. **Extrapolation to zero-flow pressure in cerebral arteries to estimate intracranial pressure**. *Br J Anaesth* 2003; 90(3):291-295.
5. Ragaisis V. **[Brain contusion: morphology, pathogenesis and treatment]**. *Medicina (Kaunas)* 2002; 38(3):243-249.
6. Huynh T, Messer M, Sing RF, Miles W, Jacobs DG, Thomason MH. **Positive end-expiratory pressure alters intracranial and cerebral perfusion pressure in severe traumatic brain injury**. *J Trauma* 2002; 53(3):488-492.
7. Granry JC, Dube L, Terminassian A, Frebet E, Le Rolle T. **[Multimodal monitoring of head injuries in children]**. *Ann Fr Anesth Reanim* 2002; 21(2):148-156.
8. Youmans Neurological Surgery. 4th ed. Philadelphia: WB Saunders, 1996.
9. Stendel R, Heidenreich J, Schilling A, Akhavan-Sigari R, Kurth R, Picht T et al. **Clinical evaluation of a new intracranial pressure monitoring device**. *Acta Neurochir (Wien)* 2003; 145(3):185-193.
10. Stevens WJ. **Multimodal monitoring: head injury management using SjvO2 and LICOX**. *J Neurosci Nurs* 2004; 36(6):332-339.
11. Chambers IR, Kirkham FJ. **What is the optimal cerebral perfusion pressure in children suffering from traumatic coma?** *Neurosurg Focus* 2003; 15(6):E3.
12. Heinemeyer G, Roots I, Dennhardt R. **Monitoring of pentobarbital plasma levels in critical care patients suffering from increased intracranial pressure**. *Ther Drug Monit* 1986; 8(2):145-150.
13. Kiefer M, Steudel WI. **[Modern intracranial pressure measurement techniques. Basic principles and general practice]**. *Unfallchirurg* 2002; 105(7):578-586.
14. Kiening KL, Schoening WN, Stover JF, Unterberg AW. **Continuous monitoring of intracranial compliance after severe head injury: relation to data quality, intracranial pressure and brain tissue PO2**. *Br J Neurosurg* 2003; 17(4):311-318.
15. Maldaun MV, Zambelli HJ, Dantas VP, Fabiani RM, Martins AM, Brandao MB et al. **[Analysis of 52 patients with head trauma assisted at pediatric Intensive Care Unit: considerations about intracranial pressure monitoring]**. *Arq Neuropsiquiatr* 2002; 60(4):967-970.
16. O'Sullivan MG, Statham PF, Jones PA, Miller JD, Dearden NM, Piper IR et al. **Role of intracranial pressure monitoring in severely head-injured patients without signs of intracranial hypertension on initial computerized tomography**. *J Neurosurg* 1994; 80(1):46-50.
17. Pillai S, Praharaj SS, Rao GS, Kolluri VR. **Cerebral perfusion pressure management of severe diffuse head injury: effect on brain compliance and intracranial pressure**. *Neurol India* 2004; 52(1):67-71.
18. Saul TG, Ducker TB. **Effect of intracranial pressure monitoring and aggressive treatment on mortality in severe head injury**. *J Neurosurg* 1982; 56(4):498-503.
19. Palmer MA, Perry JF, Jr., Fischer RP, Murray KJ. **Intracranial pressure monitoring in the acute neurologic assessment of multi-injured patients**. *J Trauma* 1979; 19(7):497-501.
20. Levin AB. **The use of a fiberoptic intracranial pressure transducer in the treatment of head injuries**. *J Trauma* 1977; 17(10):767-774.
21. Marshall LF, Smith RW, Shapiro HM. **The outcome with aggressive treatment in severe head injuries. Part II: acute and chronic barbiturate administration in the management of head injury**. *J Neurosurg* 1979; 50(1):26-30.
22. Tamaki T, Isayama K, Yamamoto Y, Teramoto A. **Cardiopulmonary haemodynamic changes after severe head injury**. *Br J Neurosurg* 2004; 18(2):158-163.