

ICP Monitoring in Patients with Severe Head Injury

Ağır Kafa Travmalı Hastalarda Kafa İçi Basınç İzlenmesi

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Abstract : This study includes 40 consecutive cases of penetrating and non-penetrating head injury with an admission Glasgow Coma Scale score of 8 or less treated in our department between 1993 and 1996. The patients were divided into two groups. Group 1 cases were treated without ICP monitoring and analysed retrospectively. Group 2 patients were treated with ICP monitoring and analysed prospectively. According to the results, ICP monitoring emerged as a useful technique in the assessment and management of patients with severe head injury. The effect of ICP monitoring on prognosis was statistically significant.

Key Words: Head injury, ICP monitoring, treatment

Özet : Bu çalışma 1993-1996 yılları arasında kliniğimizde tedavi edilen delici ve künt kafa travmalı, giriş Glasgow Koma Ölçeği puanı 8 veya altında olan hastaları içermektedir. Hastalar iki grupta incelenmiştir. Grup 1'deki hastalar kafa içi basınç izlenmeksizin tedavi edildi ve geriye dönük olarak incelendi. Grup 2'deki hastalar kafa içi basıncı izlenerek tedavi edildi ve ileri dönük olarak incelendi. Sonuçlara göre, çalışmamızdaki ağır kafa travmalı hastaların takip ve tedavisinde kafa içi basınç izlenmesi yararlı bir yöntemdir. Kafa içi basıncın izlenmesi prognoz üzerine istatistiksel olarak anlamlı etki yapmıştır.

Anahtar Sözcükler : Kafa travması, kafa içi basınç izlenmesi, tedavi

INTRODUCTION

Although Quincke was the first to report intracranial pressure (ICP) elevations via lumbar puncture (LP) in 1911, Queckenstend, Ayala and Ayer studied all aspects of ICP and demonstrated possible pressure alterations with position and respiration, determined the normal limits of ICP (21). Meyers and Browder had observed ICP in all patients suffering head injury via LP and observed pressure values which did not correlate with clinical condition (21). Guillaume and Janny managed to record ICP on a chart in 1951 (21).

In 1961, Lundberg published his first report about a patient group with recorded ICP measurements and defined waveforms known with

his name (8). In the same year, Landfitt et al. investigated the effects of experimental intracranial lesions on ICP, cerebral blood flow and vital signs (6,7). Currently, fiberoptic devices are used for monitoring ICP in the subdural, intraparenchymal and intraventricular compartments. The term of severe head injury is generally used for defining patients with head trauma and Glasgow Coma Scale (GCS) score of 8 or less (18).

In this study, ICP monitoring in patients with severe head injury, and effects of monitoring on prognosis were investigated.

CLINICAL MATERIAL AND METHODS

This series includes 40 consecutive cases of penetrating or non-penetrating head injury treated

in our department between 1993 and 1996. ICP monitored 17 patients and ICP non-monitored 23 patients were studied prospectively and retrospectively, respectively. Of 40 patients, 34 (85 %) were male and 6 (15 %) female. The average age was 25.25 ± 13.8 . The admission GCS score of all the patients was 8 or less. All of the patients underwent computerized tomography (CT) before admission.

Cases with traumatic lesions in the basal ganglia or brainstem, patients with a GCS score of 3, and without spontaneous respiration and direct / indirect pupil reaction were excluded.

Patients were grouped as follows :

Group 1 : This prospectively studied group included patients with or without penetrating head injury. ICP monitoring was performed in all patients. Of these 13 were male, and 4 female. The average age was 25.5 ± 7.7 . ICP monitoring was carried out by Camino waveform display direct pressure monitor (model V420), waveform recorder (model 427), and OLM ICP monitoring kit (model 110-4B) (Camino Laboratories, San Diego, California, USA).

Computerized tomography was performed in all patients before admission to the Intensive Care Unit (ICU). ICP was not monitored in patients with normal initial CT scan showing uncompressed basal cisterns. All patients were intubated after administration of neuromuscular blocking agent vecuronium bromide (Norkuron ampules, Organon Teknika, İstanbul, Turkey) with an initial dose of 0.08-0.1 mg/kg IV, and a maintenance dose of 0.03-0.05 mg/kg every 30 minutes IV. Midazolam (Dormicum ampule, Roche, İstanbul, Turkey) was given as sedative with an initial bolus dose of 0.03-0.3 mg/kg IV, and then subsequently 0.03-0.2 mg/kg/hr IV. The intraparenchymal device and Camino 420 monitor were used for ICP monitoring. The parenchymal device was situated into the right frontal region ignoring the side of the pathology in all cases except one. All patients with or without surgical treatment had the following treatment protocol:

1. The patient was placed in a 30° head-up position.

2. Mannitol (Mannitol 20 % solution, Eczacıbaşı, İstanbul, Turkey) was given in a dose of 0.25 mg/kg when ICP persisted over 20 mm Hg for more than 5 minutes; however, the dose was increased up to 1 g/kg when ICP did not show decrement. If there was

no ICP decrement or operative pathology on CT scans, mannitol was administered in the dose 0.25 mg/kg/4 hrs. In the cases with an ICP lower than 20 mm Hg, mannitol was not given routinely.

3. Dexamethasone (Dekort ampules, Deva, İstanbul, Turkey) was given in a dose of 4 mg/6 hrs in adults intravenously.

4. Furosemide (Lasix ampules, Türk-Hoechst, İstanbul, Turkey) was given routinely in a dose of 10 mg/12 hrs intravenously.

5. Hyperventilation was carried out keeping arterial PCO_2 between 25 to 35 mm Hg. Hyperventilation was not applied to the cases with ICP lower than 20 mm Hg. They were mechanically ventilated for 48 hrs in order to protect from external stresses.

6. Fluid was restricted in adults (75 ml/hr).

Group 2: This retrospectively studied group had 23 patients (21 male and 2 female) with penetrating and non-penetrating head injury, showing a GCS score of 8 or less who were treated without ICP monitoring. The average age was 27.3 ± 16.8 . Computerized tomography was carried out in all the patients before admission to the ICU. The following protocol was used as antiedema treatment in all patients whether they were treated surgically or non-surgically.

1. Head was elevated up to 30°.

2. Mannitol was given in a dose of 0.25 mg/kg/4 hrs.

3. Dexamethasone was given in a dose of 4 mg/6 hrs in adults.

4. Furosemide 10 mg was administered every 12 hrs.

5. Patients with insufficient respiration were intubated and ventilated, others with spontaneous respiration were supported with O_2 via mask.

6. Fluid was restricted in adults (75 ml/hr).

Transducer Placement Technique

The site, 1 cm anterior to coronal suture and 2 cm lateral to sagittal suture (Kocher's point) was infiltrated with 3 cc of prilocine (Citanest 2% vial, Eczacıbaşı, İstanbul, Turkey). After local anesthesia, parasagittal skin incision of 0.5 cm was done and

periosteum was elevated. A drill hole of 0.5 cm was performed by a hand drill (Codman, USA). The dura was confirmed and punctured by a stylet. The screw through which the transducers would be inserted, was fixed to the cranium in a clockwise manner. The transducer-tipped catheter was connected to Camino model V420 intracranial pressure monitor with waveform display and model 427 recorder. After set up and connection, the catheter was zeroed to atmospheric pressure via the potentiometer screw on the hub of the catheter and inserted into the parenchyma through the screw fixed on the cranium. Then the catheter was fixed within the screw. After the disappearance of manipulation originated fluctuations (at least 30 minutes) accurate intraparenchymal ICP values were measured.

FINDINGS AND OUTCOME

The data from the groups are summarized in the Tables I and II. The two groups were compared statistically regarding age and GCS score on admission. The differences were insignificant ($p=0.46$, and $p=0.94$).

In the second group, analyzed retrospectively and treated without ICP monitoring, 19 of 23 patients died (82.5 %). In the first group, studied prospectively and treated with ICP monitoring, 7 of 17 patients died (41.2 %). The Glasgow Outcome Scale (GOS) scores are observed to decline when ICP values increase ($p=0.45$). There was remarkable difference between GOS scores of the groups ($p=0.0011$).

Mann-Whitney U and Wilcoxon rank sum W tests were used for statistical analysis.

DISCUSSION

All treatment modalities of severe head injury aim to decrease ICP. Preserving the ICP in normal ranges decreases the mortality rate in head injury (1,2,9,17,18). Our study aims to emphasize the definitive role of ICP monitoring in planning the treatment of severe head injury. In the recent years, fiberoptic devices are in clinical practice for ICP monitoring (4). In our study, we used transducer-tipped fiberoptic intraparenchymal devices. In 1977, Mille et al. stressed the importance of ICP in 160 patients with severe head injury and determined a mortality rate of 69 % in patients with initial pressures over 40 mm Hg (12). In 9 cases of our series with an initial pressures of 40 mm Hg and more, mortality rate was 55.5 %. Controversy and conflict about the application of ICP monitoring exist in the literature

(4). Neither of the current techniques of ICP monitoring is perfect and they have both advantages and disadvantages in various aspects (4). Most significant advantages of ICP monitoring by transducer-tipped fiberoptic device parenchymally are easy application and operation which excludes problems often experienced in fluid-filled ICP monitoring systems. It also offers simple measurement of ICP in patients with compressed and/or distorted ventricles that preclude possibilities for intraventricular pressure recordings (4). The fiberoptic device has two disadvantages. Recalibration is impossible and the fiberoptic device may fracture (5).

The side for ICP monitoring is also controversial. Some authors state that ICP measurements between the lesion side and the contralateral side may alter since the intracranial compartment is divided into two compartments by the falx (20). However Yano et al. did not observe any differences in ICP measured bilaterally using subarachnoid methods (23). In our study, we did not use bilateral measurements in any case. Except a particular case, we applied all devices to the right frontal region regardless of lateralizing signs.

In 1986, Smith et al. did not determine any significant difference between severe head injury patients treated with or without ICP monitoring accompanied by mannitol administration. That study underlined that ICP monitoring did not have significance in the management of severe head injuries (19). But this conclusion was not accepted by Marshall et al. (10). We found in our study that mannitol decreased ICP in short time in Group 1. Another advantage of ICP monitoring is availability of numerical data about the patient's state, without which experienced and qualified ICU staff is required for the clinical assessment of the patients (3).

Active treatment has been started when ICP endured over 25 mm Hg for more than 5 minutes but it is also observed that better outcome rates are obtained when abrupt treatment is started at ICP levels between 15 and 20 mm Hg (18). Therefore we started osmotic treatment in our patients with ICP more than 25 mm Hg lasting more than 5 minutes.

ICP monitoring is specified not only for patients with severe head injury. Nornes and Magnaes monitored patients with intracranial aneurysm and defined spike elevation anteceding aneurysmal rupture which may indicate necessity of urgent

TABLE I. Individual data of patients in Group 1.

No	Age	S	IS	CT Features	Sur.	GOS	TH	Additional Pathology	Trauma	Examination on discharge
1	30	M	4	Bilateral frontal HC + edema	+	1	8	-	GSW	-
2	39	M	5	Right frontal HC + edema	+	1	4	-	GSW	-
3	23	M	6	Right frontal HC	+	5	23	-	GSW	Left upper limb paresis
4	25	M	6	Right frontoparietal ASDH	+	1	20	Pulmonary embolism	Accident	-
5	21	M	4	Cerebral edema + shift	-	1	6	Multiple rib Fr. + Bilat. hemothorax	Accident	-
6	21	M	6	Left parietal HC	+	4	51	-	GSW	Aphasia
7	12	F	5	Cerebral edema	-	5	10	-	Head injury	Normal
8	21	M	3	Right parietal ASDH + left frontal HC + edema	-	5	34	Fr. of left tibia	Accident	Normal
9	21	M	7	Traumatic SAH + cerebral contusion	-	5	18	Acute abdomen + diaphragmal rupture	Accident	Normal
10	32	F	3	Left parietal epidural hematoma	+	1	5	-	Accident	-
11	3	F	3	Diffuse axonal injury	-	1	2	-	Accident	-
12	21	F	5	Severe cerebral edema	-	4	25	-	Fall from a height	Hemiparesis
13	22	M	7	Left frontal HC	-	5	21	-	Head injury	Normal
14	24	F	6	HC	+	1	6	-	GSW	-
15	23	M	8	Cerebral edema	-	5	12	-	Fall from a height	Normal
16	25	M	7	Minor right parieto-occipital IC hematoma + edema	-	5	22	Left tibial Fr.	Accident	Normal
17	22	M	5	Left parieto-occipital HC + edema	-	4	14	-	Accident	Right hemiparesis

HC : Hemorrhagic contusion, Sur. : Surgical treatment, IS : Initial GCS score, GOS : Glasgow Outcome Scale score, TH : Time of hospitalization, GSW : Gunshot wounds, Fr. : Fracture, ASDH: Acute subdural hematoma, PT: Pneumothorax, SAH: Subarachnoid hemorrhage, S: Sex, IC: Intracerebral

TABLE II. Individual data of patients in Group 2.

No	Age	S	IS	CT Features	Sur.	GOS	TH	Additional Pathology	Trauma	Examination on discharge
1	27	M	6	Edema	-	1	7	-	Accident	-
2	21	M	7	Left parietal depression fr.+right frontotemporal ASDH + HC	+	1	4	-	Accident	-
3	38	M	4	HC + edema	-	1	135	-	Accident	-
4	21	M	7	Edema	-	1	4	Subcapsular splenic hematoma+ PT	Fall from a height	-
5	6	F	6	Edema + HC	-	1	5	Fr. of humerus + femur	Accident	-
6	51	F	8	Edema	-	1	2	-	Accident	-
7	18	M	8	Edema	-	3	44	-	Accident	Ambulated with support
8	50	M	4	Left sylvian ASDH	+	1	6	-	Accident	-
9	9	M	3	Severe edema+HC	-	1	2	-	Accident	-
10	21	M	8	Frontal-intraventricular hematoma+SAH	+	1	4	-	Fall from a height	-
11	21	M	8	Edema+HC	+	1	3	-	GSW	-
12	35	M	7	Right fronto-temporal epidural hematoma	+	5	18	-	Accident	Normal
13	23	M	3	Left lateral ventricular hematoma+SAH+Right IC hematoma+Right occipital HC	-	1	1	-	Accident	-
14	23	M	7	HC	+	4	24	-	GSW	Left hemiparesis
15	6	M	4	Left temporo-parietal ASDH	+	4	72	-	Accident	Tremor+paresis of right hand
16	20	M	7	Left parietal ASDH+edema	+	1	20	-	Accident	-
17	24	M	5	Edema+hematoma in brainstem	-	1	12	-	Accident	-
18	66	M	5	Bilateral parietal ASDH	+	1	1	Fr. of femur + PT	Accident	-
19	60	M	7	Left frontal IC hematoma + fronto-parietal ASDH	+	1	7	PT	Accident	-
20	21	M	6	HC	+	1	8	-	GSW	-
21	43	M	4	Edema	-	1	30	Leford II Fr.	Fall from a height	-
22	19	M	3	Right fronto-temporal epidural hematoma	+	1	5	-	Fall from a height	-
23	5	M	3	Edema	-	1	6	Fr. of left tibia +fibula	Accident	-

HC : Hemorrhagic contusion, Sur. : Surgical treatment, IS : Initial GCS score, GOS : Glasgow Outcome Scale score, TH : Time of hospitalization, GSW : Gunshot wounds, Fr. : Fracture, ASDH: Acute subdural hematoma, PT: Pneumothorax, SAH: Subarachnoid hemorrhage, S: Sex, IC: Intracerebral

surgery (15). Mullen et al. determined optimal arterial blood pressure levels in patients with aneurysm and its relation with ICP (13).

The complications of ICP monitoring are infection and hemorrhage (5). The superficial infections in the entrance region of the probe are rare and insignificant. Ventriculitis is a fatal complication especially when it is not early detected. The reported rate of ventriculitis ranges between 1 to 10 % in patients with ventriculostomy (5,11,14).

Although the infection rate is fairly low in subdural techniques, these measurements are not reliable (16). Many authors investigated the relation between the time of monitoring and infection (5, 11, 14, 16). However Winfield et al. could not find a reasonable relation between infection and the time of monitoring in a series of 212 patients (22). We did not observe any infection in our cases.

The second major complication of ICP monitoring is the intracerebral hemorrhage (14). The risk is higher in the patients with coagulopathy. In our cases, hemorrhage due to monitoring was not seen.

In our study, 19 of 23 patients (82.5 %) of Group 2 investigated retrospectively and treated without ICP monitoring died. Seven of 17 patients (41.2%) of Group 1 investigated prospectively and treated with ICP monitoring died. Comparing the GOS scores in the groups, a meaningful difference exist ($p=0.0011$). Many studies in the literature underlined the age of the patient and the initial GCS score as the most efficient factors determining outcome (2, 12). In our study, these factors were analyzed statistically and there was not significant difference between outcomes as to initial GCS score and age ($p=0.916$, and $p=0.46$). Although there was not any significant difference between the groups regarding the time of hospitalization, the maximum hospitalization times in Groups 1 and 2 were 51 days (6th case) and 135 days (3rd case), respectively.

These results suggest that ICP monitoring emerged as a useful method in the assessment and management of patients with severe had injury in our study and has been applied routinely in our department for patients with penetrating or nonpenetrating head injury.

The avoidance of external stresses and anesthesia of the patient for hyperventilation to regulate the respiration are necessary for the entire

treatment of head injury. We were not able to observe the patterns of respiration in comatose patients when we did not perform ICP monitoring. We had this opportunity with anesthesia and artificial respiration of the patients. As emphasized before, the patient can not be anesthetized when we monitor the patient by the means of clinical examination. The basis of the success in the Group 1 are routine sedation, mechanical ventilation and hyperventilation when it is needed to manage the patients.

There are two causes of secondary brain damage following severe head injury. These are hypoxia and ischemia. Ischemia is primarily caused by shock and elevated ICP. In severely head-injured patients with coma 65 % hypoxia, and 16 % shock are observed. These two factors are significant in the prognosis of the patient (5). Another reason in the increment of GOS scores of our patients in Group 1 is the protection of possible secondary brain damage by ICP monitoring and artificial ventilation.

The most significant advantage of ICP monitoring is evaluation of intracranial hypertension which accompanies clinical deterioration.

In this study, we emphasized the significance of ICP monitoring with fiberoptic devices in the follow up, and management of patients with severe head injury. We consider ICP monitoring as a crucial modality in the follow up of the patients with severe head injury.

In conclusion, according to these data, it is not an exaggeration to state that follow up and treatment of the severely head-injured patients without ICP monitoring will not be as satisfactory as desired.

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