

## DISPROPORTION BETWEEN ICP-CPP DATA AND NEUROLOGIC FINDINGS IN A PATIENT WITH SEVERE HEAD TRAUMA: CASE REPORT

Mladen Novkoski<sup>1</sup>, Aleksandar Gopčević<sup>1</sup>, Mijo Kelečić<sup>1</sup>, Željko Ivanec<sup>1</sup>, Darko Bedek<sup>2</sup> and Mladen Perić<sup>1</sup>

<sup>1</sup>Department of Anesthesiology and Intensive Care, and <sup>2</sup>Department of Radiology, Sestre milosrdnice University Hospital, Zagreb, Croatia

**SUMMARY** – A 48-year-old man with relative hepatic impairment, thrombocytopenia and chronic alcoholism was admitted to intensive care unit six hours after head trauma. Computed tomography (CT) scan at admission indicated cerebral edema and subdural hematoma. Glasgow Coma Score (GCS) was 5, and his pupils were 4 mm in diameter without response. Decompression craniotomy with evacuation of hematoma was performed, and a ventricular catheter was introduced for intracranial pressure (ICP) monitoring. After the operation, there was no change in the neurologic status. ICP/ CPP and jugular vein oxygen saturation (SvO<sub>2</sub>) were continuously monitored and data were collected in hour-to-hour manner. Therapeutic goal was to optimize ICP and cerebral perfusion pressure (CPP) in accordance with the Guidelines for the Management of Severe Head Injury. Mannitol and barbiturates were not used in therapeutic procedures. Normocapnic values of PCO<sub>2</sub> were maintained. During the first six hours postoperatively, the patient was stabilized hematologically and hemodynamically. At 24 hours postoperatively, there was no improvement in the neurologic status in spite of optimal values of ICP/ CPP and SvO<sub>2</sub>: GCS 5, pupils 4 mm with no response. The reason for this was detected on 24-hour CT scan, which revealed thalamic and pontine hemorrhage (mesencephalon lesion) with ischemia in the occipital region.

**Key words:** *Brain injuries, complications; Brain injuries, diagnosis; Predictive value of tests; Hematoma, complications; Intensive care, methods; Case report*

### Introduction

Head trauma is the most common problem encountered in daily routine of neurosurgical intensive care unit (ICU). The choice of appropriate diagnostic procedures and treatment protocols, and almost always an unpredictable outcome make the management of this group of patients highly demanding, yet a great challenge for physicians.

### Case Report

A 48-year-old man was admitted to ICU after he had been examined at another hospital where acute hemispheric subdural hemorrhage with marked consciousness disturbance had been verified. Six hours had elapsed from the injury. Heterohistory data revealed the patient to suffer from chronic alcoholism and relative hepatic impairment. On admission, his Glasgow Coma Score (GCS) was 5 (motor response 3, eye movements 2, verbal response 1), the pupils were 4 mm in diameter with no response, and APACHE II was 20. There were no signs of lateralization, and Babinski's sign was negative. Other injuries included thorax contusion with no rib fracture, and lacerations and contusion of the left zygomatic region. Com-

Correspondence to: *Mladen Novkoski, M.D.*, Department of Anesthesiology and Intensive Care, Sestre milosrdnice University Hospital, Vinogradska c. 29, HR-10000 Zagreb, Croatia

Received May 17, 2000, accepted in revised form August 17, 2000

puted tomography (CT) scan revealed an acute subdural hematoma of 3 cm in diameter with displacement of the ventricular system. There also were signs of brain edema.

Adequate perfusion pressure, intracranial pressure (ICP) control, improvement of hemodynamic parameters, avoiding hypercapnia, proper oxygenation, and prevention of infection were set as the main goals of diagnostic and therapeutic procedures<sup>1</sup>. On admission to ICU, the patient was intubated, peripheral veins were cannulated with large bore cannulas, invasive blood pressure monitoring was established, and after short optimization of vital signs the patient was transferred to operating room. Osteoclastic craniotomy with evacuation of subdural hematoma was performed and a catheter was introduced into the left lateral ventricle for ICP monitoring. After the operation, the patient was transferred to ICU. A central venous catheter was inserted in the subclavian vein, the right jugular vein was cannulated, and a fiberoptic catheter was introduced for continuous monitoring of the jugular vein oxygen saturation (SvjO<sub>2</sub>).

Laboratory findings showed elevated hepatic enzymes (AST 80, ALT 56), marked thrombocytopenia (65/ml), and elevated glucose level (8.4 mmol/L). Coagulation tests, electrolyte measurements, and ABS were normal. CT scan performed at 24 hours postoperatively showed the state after osteoclastic craniotomy and evacuation of subdural hematoma; fresh blood in the pons, right tha-

lamic region and ventricular system; and ischemic lesions in the occipital region and basal ganglia. There was no improvement of the neurologic status although normal values of ICP and cerebral perfusion pressure (CPP) were achieved. GCS was 5 (motor response 3, eye movements 1, verbal response 1), and the pupils were 4 mm in diameter with no response. There were no signs of lateralization, and Babinski's sign was negative. Reflexes were preserved except for swallowing. Control CT scan performed at 48 hours showed unchanged findings. As there was no amelioration, invasive ICP and SvjO<sub>2</sub> monitoring was discontinued at 120 hours from admission. The patient died on day 8 after admission.

In current protocols for severe head injury, the goal of treatment is to achieve ICP < 20 mm Hg and CPP between 60 and 80 mm Hg. Recently, a value of 60 mm Hg has become a therapeutic hallmark, as this value has been associated with adequate tissue oxygenation<sup>2</sup>. An important issue also is the earliest possible normalization of CPP to prevent reperfusion injury. On the other hand, excessive CPP values may be detrimental and lead to cerebral hypoxemia, as reported for both animal models and humans<sup>3,4</sup>. ICP is another variable monitored and it should not exceed 20 mm Hg. If early intracranial hypertension is recorded, the outcome is poor even if hypertension is reduced during subsequent therapeutic process<sup>5</sup>. In daily therapeutic and diagnostic procedures, we use

Table 1. Values of Glasgow Coma Score (GCS), intracranial pressure (ICP), mean arterial pressure (MAP), cerebral perfusion pressure (CPP) and jugular vein oxygen saturation (SvjO<sub>2</sub>)\*

Day	GCS	ICP (mm Hg)	MAP (mm Hg)	CPP (mm Hg)	SvjO <sub>2</sub> (%)	Pupils (mm)	
						L	D
On admission					4	4	
	5	71				-	-
After operation						4	4
	5	24	84	70	55	-	-
1						4	4
	5	21	102	79	71	-	-
2						4	4
	5	17	85	63	78	-	-
3						4	4
	4	23	87	69	79	-	-
4						4	4
	4	24	102	78	78	-	-
5						4	5
	3	23	85	62	76	-	-

\*values represent an average 24-hour measurement in hour-to-hour manner

Guidelines for the Management of Severe Head Injury, issued by the Brain Trauma Foundation. The protocol approach to severe head injury is advantageous to individualized treatment because ICP and CPP oscillations are lower<sup>6</sup>. Although prospective randomized trials to validate current scheme of treatments are lacking, it has been widely accepted that appropriate intravascular volume, correction of ICP and CPP, careful introduction of mannitol in therapy before ICP monitoring has been established are of utmost importance. Hyperventilation should be avoided, while barbiturates are only used when other therapies fail. There is strong evidence of useless administration of corticosteroids in therapy for severe head trauma.

In some circumstances, there is the lack of neurologic improvement even if the monitored parameters are good. The reason for this lies in infratentorial lesions and lesions of deep structures of the brain. The patient described here had injuries to the mesencephalon and pons. There also was evidence of ischemia in the occipital region, probably as a secondary injury. As this type of injuries can hardly be appropriately managed, they lead to fatal outcome, as differentiated from supratentorial lesions, which has been reported for both animal models and in humans<sup>4</sup>. Early detection of these lesions is also hardly possible because of the dynamics and pathophysiology of brain trauma, including secondary brain injury. Thus, most activities

should be focused on appropriate primary medical treatment within the 'golden hour', in order to prevent hypoxia and hypotension, supplemented with other support procedures.

## References

1. The Brain Trauma Foundation. Guidelines for the management of severe head injury, 1995.
2. KIENING KL, HARTL R, UNTERBERG AW, SCHNEIDER GH, BARDT T, LANKSCH W. Brain tissue PO<sub>2</sub> monitoring in comatose patients – implications for therapy. *Neurol Res* 1997;19: 233-40.
3. UNTERBERG AW, KIENING KL, HARTL R, SARRAFZADEH AS, BARDT T, LANKSCH WR. Multimodal monitoring in patients with head injury – evaluation of the effects of treatment on cerebral oxygenation. *J Trauma Injury Infect Crit Care* 1997;42 (Suppl S):S32-S37.
4. KROPSTEDT SN, KERN M, THOMALE UW, SCHNEIDER GH, LANKSCH WR, UNTERBERG AW. Effect of cerebral perfusion on contusion volume following impact injury. *J Neurosurg* 1999;90:520-6.
5. SIGNORINI DF, ANDREWS PJD, JONES PA, WARDLAW JM, MILLER JD. Adding insult to injury: the prognostic value of early secondary insults for survival after traumatic brain injury. *J Neurol Neurosurg Psychiatry* 1999;66:26-31.
6. MCKINELY BA, PARMLEY CL, TONNESON AS. Standardized management of intracranial pressure: a preliminary clinical trial. *J Trauma Injury Infect Crit Care* 1999;46:271-9.

## Sažetak

### NESRAZMJER IZMEĐU PODATAKA ZA INTRAKRANIJSKI I CEREBRALNI PERFUZIJSKI TLAK TE NEUROLOŠKIH NALAZA U BOLESNIKA S TEŠKOM OZLJEDOM GLAVE: PRIKAZ SLUČAJA

*M. Novkoski, A. Gopčević, M. Kelečić, Ž. Ivanec, D. Bedek i M. Perić*

Muškarac star 48 godina s relativnim oštećenjem jetre, trombocitopenijom i kroničnim alkoholizmom u anamnezi primljen je u jedinicu intenzivnog liječenja šest sati nakon ozljede glave. Nalaz kompjutorizirane tomografije (CT) kod prijma pokazao je subduralni hematoma i edem mozga. Kod primitka, Glasgow Coma Score (GCS) bio je 5, zjenice 4 mm izokorične i nereaktivne. Učinjena je dekompresijska kraniotomija s evakuacijom hematoma, te je postavljen kateter za mjerenje intrakranijskog tlaka (ICP). Kontinuirano su mjereni ICP, cerebralni perfuzijski tlak (CPP), zasićenje jugularne vene kisikom (Sv<sub>2</sub>O<sub>2</sub>) i CO<sub>2</sub>. Nakon 24 sata nije došlo do neurološkog poboljšanja unatoč tome što su postignute željene terapijske vrijednosti svih praćenih parametara. Kontrolni CT mozga pokazao je krvarenje u ponsu i talamusu, te ishemiju okcipitalno, zbog čega je izostalo poboljšanje neurološkog statusa. Kasna dijagnostika i nedostupnost za terapiju dubokih i infratentorialnih ozljeda mozga problem su u svakodnevnom liječenju ovih bolesnika.

*Ključne riječi: ozljede mozga, komplikacije; ozljede mozga, dijagnostika; prognostička vrijednost pretraga; hematoma, komplikacije; intenzivna skrb, metode; prikaz slučaja*

