

Polytrauma with severe traumatic brain injury. Case report

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Abstract

The management of polytrauma patients requires a multidisciplinary approach, usually realised by a trauma team, based on trauma protocols. This category of patients should be treated in trauma centers, which are hospitals with extensive human and infrastructure resources. The authors present the case of a 17 years old female, a car accident victim, successfully treated in Iasi Neurosurgery Hospital. As our hospital did not fulfill the requirements of a trauma center, the patient was managed in cooperation with various specialists from other hospitals. After the secondary survey, the patient presented severe traumatic brain injury (Glasgow Coma Scale 5), double fracture of the mandibula, left pulmonary contusion, grade I spleen injury, fracture of the left clavicle and left radius bone, having an injury severity score of 29. During the Neurointensive Care Unit hospitalization, the patient was submitted to multiple neurosurgical interventions. She was mechanically ventilated for 10 days, with complications during the evolution such as acute respiratory distress syndrome, bacterial meningitis and severe sepsis with hospital acquired microorganisms. During the stay in ICU, after an alternating but eventually positive evolution, the rehabilitation therapy was instituted. At discharge, after a length of stay (LOS) of 69 days, the patient was alert and awake with sensory aphasia and right hemiparesis and was referred to a neurologic rehabilitation clinic.

Keywords: severe traumatic brain injury, polytrauma, severe sepsis, ARDS

Introduction

Polytrauma is a syndrome determined by the action of different powerful agents (e.g. mechanical, chemical) that affect at least two regions of the human body, with at least one of the injury having a lethal potential. The newer definition of polytrauma implies the use of injury severity score (ISS) - ISS must be equal or greater than 17 (2) (table 1). The etiology and seriousness of polytrauma has very much changed in the last century due to the development of mankind (warfare techniques, car industry) .

We report the case of a car accident victim. The patient was admitted in the Intensive Care Unit (ICU) of Iasi Neurosurgery Hospital with an ISS of 24, having severe traumatic brain injury, chest and abdominal trauma and multiple bone fractures, the brain injury being the most severe of all the traumatic injuries.

Case presentation

The patient T.A., female, aged 17, victim of a car accident, without other known associated pathology or drug abuse

history was brought by the ambulance at the Emergency Department of Iasi Neurosurgery Hospital in deep coma (Glasgow Coma Scale 5 – E1V1M3), intubated, sedated, mechanically ventilated. The neurologic exam revealed spontaneous flexion of both arms and miotic, equally in diameter and reactive pupils. The patient was haemodynamically stable, with breath sounds present equally bilateral and left basal rales. The cerebral native CT-scan revealed bilateral fronto-temporal haemorrhagic contusions, left occipital hematoma and laceration, with diffuse cerebral edema without midline shift and left parieto-occipital comminutive fracture. The patient had received mannitol 1 gram per kilogram in the ambulance. As our hospital did not have the resources of a trauma center and as the cerebral injuries did not require emergent surgical intervention (except for the insertion of an intracranial catheter for intracranial pressure (ICP) monitoring, not available in our hospital at that time), the patient had to be sent to the Surgery Clinic for the diagnosis and treatment of the chest, abdomen and extremity injuries.

AIS Score	Injury
1	Minor
2	Moderate
3	Serious
4	Severe
5	Critical
6	Unsurvivable

The patient returned to our clinic after a few hours, diagnosed with left basal pulmonary contusion, grade I spleen injury, double fracture of the mandibula, fracture of the left clavicle and distal

epiphysis of the left radius bone, none of which had necessitated emergency intervention.

The patient was admitted in the Intensive Care Unit (ICU) of the Neurosurgery Hospital, as the cerebral injuries had the highest severity. She received the standard ICU care (vital signs monitoring, CVP monitoring via a central venous line, nasogastric tube, hourly diuresis monitoring). She received analgesia and sedation with fentanyl and propofol, and was mechanically ventilated in IPPV Autoflow assist mode (intermittent positive pressure ventilation). The ventilator parameters were set to maintain the paCO_2 (partial pressure of carbon dioxide in arterial blood) between 30 and 35 mm Hg (mild hyperventilation). Head of bed was raised at 45 degrees. The mean arterial pressure was maintained over 90 mm Hg (according to the guidelines - 4) without the use of vasoactive drugs. The oxygenation was good - paO_2 (partial pressure of oxygen in arterial blood) \geq 100 mm Hg at a FiO_2 (fraction of inspired oxygen) of 0.5.

The lab tests on admission (complete blood count, biochemistry, coagulation tests) were normal except for the hemoglobin (8.5 g/dl), hematocrit (25.5%) and white cell count (19800/mm³).

Serial neurological examinations were performed, as there was no ICP monitoring available. Enteral nutrition was initiated.

After 24 hours of hospitalisation, the patient developed anisocoria (left mydriasis).

Table 1
The patient's ISS (Injury Severity Score) based on AIS (Abbreviated Injury Scale) on ICU admission (2)

Region	Injury Description	AIS	Square Top Three
Head & Neck	Cerebral Contusion	4	16
Face	double fracture of the mandibula	1	
Chest	left basal pulmonary contusion	2	4
Abdomen	grade I spleen injury	2	4
Extremity	fracture of the left clavicle fracture of the distal epiphysis of the left radius bone	1 1	
External	none	0	
Injury Severity Score			24

The CT scan showed, apart from the injuries presented at admission, the augmentation of the cerebral edema, compression of the left lateral ventricle and important midline shift towards right. As the neurological status did not improve after intermittent boluses of mannitol, the surgeons performed an emergent left decompressive craniectomy.

After the intervention, the neurological status improved - pupils would become equal and reactive with a GCS of 6 (E1V1M4).

On the second and third postoperative day, the patient began to present pulmonary rales and fever ($\leq 38,8^{\circ}\text{C}$) (being mechanically ventilated) and hypoxemia despite higher values of FiO_2 . In addition, the chest X-ray showed bilateral lung infiltrates; corroborated with a low hypoxemia score ($\text{PaO}_2/\text{FiO}_2$ under 200 mmHg), and a suggestive clinical context (pulmonary contusion), all these criteria sustained the diagnosis of

acute respiratory distress syndrome (ARDS) (3).

The ventilation mode was switched to BIPAP (Bi-level Positive Airway Pressure), with a lung protective strategy ventilation (low tidal volumes were used). Still, the alveolar recruitment measures were impeded by the use of low values of PEEP (positive end expiratory pressure) because of the cerebral edema..

During the first 2 postoperative days, it was difficult to adapt the patient to the ventilator whilst trying not to aggravate the cerebral edema shown on the postoperative day 1 CT scan (figure 2). This is why we chose to paralyse the patient with atracurium. Intermittently we stopped the muscle relaxant for neurologic examinations.

The paralysis only slightly improved the hypoxemia score and was stopped after 48 hours. After that, we increased the PEEP values to 10 – 12 cm H₂O, which improved the oxygenation.

In the third postoperative day the bacteriology came positive in the tracheal secretions for *Pseudomonas aeruginosa*, with negative hemocultures, urine and feces cultures. The chest X ray was suggestive for bronchopneumonia; the lab tests showed, apart from the inflammatory syndrome, a severe anemia (hematocrit 19%), but also moderate thrombocytopenia with altered coagulation tests, a low albuminemia and unconjugated hyperbilirubinemia – signs of liver failure. The anemia was hemolytic. The patient became oliguric, with a serum creatinine value of 2.2 miligrams per deciliter. With the procalcitonin test positive, we suspected severe sepsis of pulmonary origin. (Surviving Sepsis Campaign, 2004). At that time, the patient had a SOFA (Sequential Organ Failure Assessment) score of 14 (6).

We performed initial fluid resuscitation (first 6 hours) according the Surviving Sepsis Campaign 2004 guidelines (5).

The patient received broad spectrum antibiotics until the positivation of cultures; afterwards, as guided by the antibiogram (the strain had sensibility for imipenem) She also received erytrocite mass, fresh frozen plasma and albumin. For two days we initiated inotrope (dobutamine – 5 μ grams/kilogram/minute) and vassopressor therapy (dopamine – 6–10 μ grams/kilogram/minute) because of the hemodynamic instability. We did not have the possibility of inserting a Swan Ganz catheter, which would have been useful for managing the hemodynamic parameters.

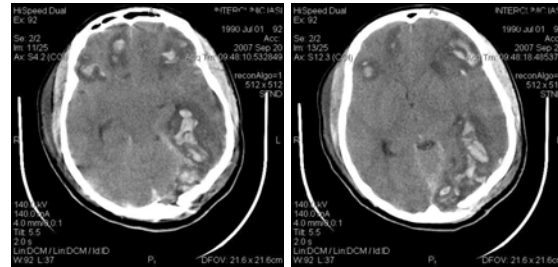


Figure 1 Cerebral CT scan on admission (see text for explanations)

The patient responded well to the therapy. Gradually, the vasopressors were stopped. The hypoxemia score gradually improved, correlated with the chest X-ray images. Still, the patient continued to present pulmonary rales and subfever, which resolved after three days. The renal and liver failure responded well to fluid resuscitation. There was no need for renal replacement therapies.

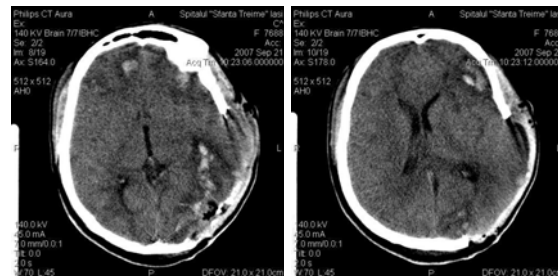


Figure 2 CT scan: postoperative day 1. One can observe the brain herniation through the craniectomy space

Through the seriated neurological evaluations, the patient began to present reactivity at pain stimuli (withdrawal on the left side, right hemiplegia) – GCS – 6. The pupils were equal and reactive. The haemorrhagic cerebral injuries began to resolve, and the CT scan performed at day 4 showed the diminution of the mass effect as the brain herniated through the craniectomy defect. (Figure 3).

During the 8th postoperative day, the patient opened her eyes. After a CPAP weaning protocol, she was detubated (Glasgow coma scale 10). Antibiotherapy was continued (imipenem plus levofloxacin).

After a period of relative stability, the cultures from the tip of the central venous catheter became positive also for *Pseudomonas aeruginosa*, with sensibility for imipenem.

In the meantime, several complications of trauma and surgery developed – hydrocephalus, external CSF (cerebrospinal fluid) fistula and after that, meningitis with coagulase-negative *Staphylococcus* susceptible at linezolid and vancomycin.

A continuous lumbar drainage was instituted (because of the meningitis, an external ventricular drainage was contraindicated) and vancomycin was added in the therapy. The fistula and meningitis gradually resolved.

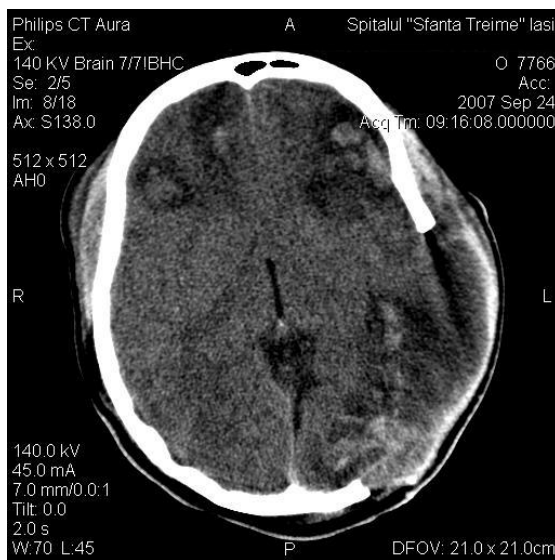


Figure 3 CT scan: postoperative day 4 (see text)

After 7 days of continuous lumbar drainage, for the definitive treatment of hydrocephalus a ventriculo-peritoneal shunt was put in place.

During the evolution, the haemorrhagic injuries showed resorption, but an ischemic area in the left frontal lobe persisted (see Figure 4).

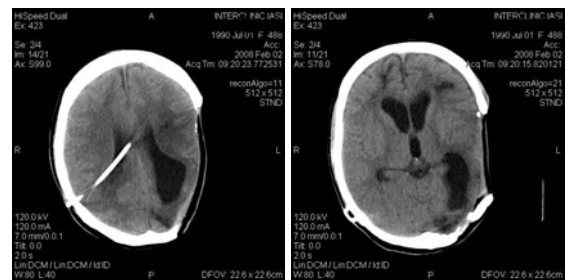


Figure 4 CT scan: 18 weeks after the accident

After 56 days in the ICU, the patient was transferred to a maxillo-facial surgery clinic, for the definitive treatment of the mandibula fracture. She was sent back after 2 days.

During the time spent in the ICU, the neurologic rehabilitation therapy was instituted. The patient was discharged with right hemiparesis, sensory aphasia and deglutition disturbances, receiving food via the naso-gastric tube. She was referred to a neurologic rehabilitation clinic.

Within one month, the deglutition disturbances ameliorated, and the aphasia partially resolved. After 8 months, the patient returned to our clinic for cranioplasty.

Discussion

The main issues in the management of this patient with severe TBI were the lack of a trauma center in Iasi and the lack of

ICP monitoring in our hospital. The trauma patient is one of the most critical patients. The management of polytrauma should be realised in a trauma center, by a trained trauma team (1). Transportation between different hospitals for diagnosis should be avoided, if possible. Still, we had to send the patient for the evaluation of the chest and abdominal injuries, as our hospital was the first medical facility the patient had been brought to.

The initial neurologic deterioration took place with the patient being sedated, mechanically ventilated, with the ventilatory parameters correlated with the serial ABGs and with a mean arterial pressure kept above 90 mm Hg, but without an ICP monitor. Despite its controversies, the management of high ICP was realised by decompressive craniectomy, which allowed the prolapse of the edematous brain through the edges of the craniectomy defect.

The challenges of this case were the management of the combination between severe head trauma and ARDS, and of the severe sepsis, with MSOF (multi systemic organ failure) – respiratory, liver, renal failure.

In evolution, this case presented multiple septic complications. The postoperative external CSF fistula was the cause of the meningitis with a nosocomial bacteria.

The patient was hospitalized for 69 days. The hospitalisation costs were around 20000 RON.

Conclusion

The decompressive craniectomy, despite the controversies, proved efficient.

The management of ARDS combined with severe TBI was difficult during the first two postoperative days. Severe sepsis was treated according to the latest guidelines. The alternating clinical evolution of the patient was due to the hospital acquired microorganisms, with multiple antibiotic resistance.

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