Traumatic Brain Injury Part V: Complications and Outcomes

Table of Contents

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First Edition Update 2022

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Intended Learning Outcomes

TBI Part V: Complications and Outcomes

1. Summarise and describe late complications of severe TBI
2. Describe the diagnosis and treatment post-traumatic hydrocephalus
3. Describe the management of the sunken skin flap syndrome
4. Discuss the appropriate management of post-traumatic meningitis
5. Outline the principles of identification and management of carotid-cavernous fistulae
6. Desribe outcome prediction in patients with severe TBI
7. Explain the importance of audit and benchmarking in ICUs managing patients with TBI

eModule Information

Relevant Competencies from CoBaTrICE

TBI Part V: Complications and Outcomes

- **2.9** Monitors and responds to trends in physiological variables
- **2.10** Integrates clinical findings with laboratory investigations to form a differential diagnosis
- **3.6** Recognises and manages the patient with neurological impairment
- **6.3** Manages the care of the patient following craniotomy under supervision
- **6.5** Manages the pre- and post-operative care of the trauma patient under supervision
- **7.1** Identifies and attempts to minimise the physical and psychosocial consequences of critical illness for patients and families
- **11.7** Describes commonly used scoring systems for assessment of severity of illness, case mix and workload
- **11.8** Demonstrates an understanding of the managerial & administrative responsibilities of the ICM specialist

1. Complications and Outcomes

1. 1. Late complications of traumatic brain injury

1. 1. 1. Post-traumatic hydrocephalus

Incidence and pathophysiology: Approximately two thirds of all patients suffering from severe brain injury will develop post-traumatic enlargement of the ventricles. True post-traumatic communicating hydrocephalus, may occur in up to 1–5% of patients.

In those patients who go on to develop post-TBI hydrocephalus, it occurs in up to 25% within two weeks of injury and in 90% within six weeks.

Meningitis causes impaired absorption of CSF in the arachnoid villi as it flows over the convexities. Primary and/or secondary decompression are now also considered a cause of hydrocephalus, probably due to a reduction of vault surface area for CSF reabsorption.

Diagnosis: Post-traumatic hydrocephalus is normally slowly progressive. Its clinical presentation is variable, making diagnosis a challenge especially in severely impaired TBI patients. The main clinical markers are prolonged recovery from coma and delay in rehabilitation. CT scans will reveal progressive ventricular enlargement and compression of the cerebral sulci (see below). Typically there is a plateau in patient recovery from coma or sometimes a decline after initial progress. Additional investigations may include lumbar puncture, CT cisternography and radionuclide absorption tests.

Often however, the differential diagnosis between atrophy and hydrocephalus remains unclear, and it may be uncertain in patients who are more disabled or in a vegetative state whether the treatment of hydrocephalus will improve patient function. In such cases, a CSF withdrawal test is indicated: liquor is continuously drained by means of an external spinal catheter and improvements of neurologic performance can be recorded to guide the decision to place a ventricular shunt.

Treatment: consists of CSF shunts, either ventriculo-peritoneal or ventriculo-atrial. Even after decompressive craniectomy, there is a high incidence of hydrocephalus which may persist after cranioplasty or bone-flap replacement. In some of these cases, depending on the evidence from serial clinical and CT follow-up, definitive shunting may be required.

In text References

(Licata et al. 2001; De Bonis et al. 2010; Rufus et al. 2021)

1. 1. 2. Sunken skin flap syndrome

The ‘sunken skin flap syndrome’ (SSFS) is defined as a secondary neurological deterioration in the presence of a sinking skin flap in patients with large craniectomies. The syndrome occurs several weeks to months after decompressive craniectomy and is frequently related to being in the upright position and to daytime-dependent, fluctuating headaches. Concurrent with the sinking of the skin flap and the underlying brain tissue, a deterioration of neurological function with new symptoms and signs is observed. These include focal signs, disturbances of consciousness, epileptic seizures and neuroendocrine abnormalities.

These patients are at risk of paradoxical herniation if they undergo diagnostic lumbar puncture or therapeutic lumbar CSF drainage. Although several changes in CSF hydrodynamics, cerebral blood flow and brain metabolism have been described as partial aspects of the pathophysiology, full understanding of the underlying condition is still lacking. The incidence of this phenomenon is rare and the associated syndrome is sometimes not appreciated. Cranioplasty leads to clinical improvement in several cases and may avoid secondary deterioration.

Cranioplasty is therefore a therapeutic as well as a cosmetic procedure. The intensivist should be aware of SSFS and of other delayed complications of external decompression when considering whether secondary decompressive craniotomy is indicated as a therapy for unresponsive ICP.

In text References

(Chieregato. 2006; Stiver. 2009)

1. 1. 3. Post-traumatic meningitis

Incidence: The reported incidence of this complication is 5–15%. In good centres, the incidence of meningitis, which is associated with ICP measurement catheters or craniotomy, is less than 1%.

Pathophysiology: Risk factors include open, contaminated wounds, CSF leaks (rhinorrhea or otorrhea) through basal skull fractures, and ventricular catheters for ICP measurement. The causative organisms tend to be those normally colonising the nasopharynx such as *Streptococcus pneumoniae, Hemophilus influenzae, Neisseria meningitidis*, and Staphylococci species.

Diagnosis: In all TBI patients with fever of unknown origin, post-traumatic meningitis must be in the differential diagnosis especially if intracranial air or rhinorrhea/otorrhea has suggested an open skull fracture or if a ventricular drain is in place. Other common clinical and laboratory findings include neck stiffness, leukocytosis and increased C-reactive protein. The diagnosis is confirmed by isolation of the

causative organism from CSF cultures. The diagnosis is supported by decreased glucose (<50 mg/dL; 2.8 mmol/L) and elevated white blood cell count (>10,000/mm3) with a high proportion of leukocytosis (>75%).and a high CSF lactate. Additional common CSF findings include elevated protein (>100–500 mg/dL).

Note

Do not necessarily consider a CSF specimen, which is culture positive, as diagnostic of infection if no signs of inflammation are detected in the CSF specimen or in the patient. Consider the possibility that CSF has been contaminated during sampling or that the catheter is colonised.

Microbiological aetiology: Is often evaluated by reviewing CSF cultures. Ventricular catheter associated ventriculitis is commonly due to *Pseudomonas aeruginosa, Staphylococcus aureus, Staphylococcus epidermidis* and sometimes, in epidemic forms, *Acinetobacter baumannii*.

Prophylaxis: Antibiotic prophylaxic in patients with basilar skull fractures has not been proved to be effective in preventing meningitis.

Persistent post-traumatic cerebrospinal fluid leakage should be treated surgically. In the case of external drainage, routine replacement of the catheter to prevent infection does not seem effective in preventing infection.

Treatment: Meningitis has to be treated immediately as the mortality of untreated bacterial meningitis is high.

As soon as CSF has been sent for gram-stain and culture, high-dose antibiotics are commenced on a best guess basis if warranted clinically

Note

Post-traumatic meningitis is an emergency.

Note

If the diagnosis is suspected, discuss it with the patient's consultant neurosurgeon urgently. Discuss whether it is safe to perform a lumbar puncture in any comatose patient in view of the risk of brain herniation when increased ICP may be present. In addition to blood culture, try to obtain a specimen of CSF before starting antibiotics.

Task

Test your knowledge

If the gram-stain is positive and suggestive of Streptococcus pneumoniae, what antibiotic would be appropriate?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

Intravenous penicillin (20–30 M/day) is usually indicated.

If it was suggestive of staphylococcal infection, what might be the appropriate anti-staphylococcal antibiotics?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

Flucloxacillin, oxacillin, vancomycin (if MRSA suspected), and perhaps supplementation with rifampicin.

The choice would be made on the basis of local surveillance reports and individual history of antibiotic exposure of the patient.

In the case of Gram-negative organisms what might be an appropriate antibiotic?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

A third generation cephalosporin (e.g. high-dose ceftriaxone, cefotaxime which penetrate well into CSF) or a carbapenem antibiotic.

Consider the effect of such antibiotic in the selection of multiresistant organisms and ensure adequate therapy once bacterial aetiology is determined. Treatment will be modified as indicated when culture and sensitivity results become available. In the case of shunt related infections, infected ventricular drains should be removed and replaced since acute hydrocephalus can complicate meningitis. In selected cases, give consideration to intrathecal administration of antibiotics which is feasible and efficacious in *Staphylococcus aureus, Staphylococcus epidermidis* (vancomycin and gentamycin) and mutiresistant Acinetobacter infection (consider colistin).

In text References

(Ratilal, Costa and Sampaio. 2006)

1. 1. 4. Carotid-cavernous (C-C) fistulae

Pathophysiology: C-C fistulae result from injuries to the internal carotid artery as it passes through the sinus cavernosus. They are more common in blunt trauma than in open injuries, often associated with a basal skull fracture. The tear between the carotid artery and the cavernous sinus causes a high-flow fistula into the sinus resulting in venous hypertension of the sinus and the superior ophthalmic vein, the petrosal sinus and the pterygoid venous plexus. Venous hypertension may cause impairment of cranial nerves II, III, IV, and VI. More serious complications include ipsilateral loss of vision, subarachnoid and intraparenchymal haemorrhage, or ischaemia due to steal of blood from the territory of the major arteries.

Diagnosis: The classical clinical picture includes pulsating exophthalmus and chemosis. In some cases, the sound of the fistula can be heard by a stethoscope pressed onto the temporal region.

Transcranial Doppler can also confirm the diagnosis and cerebral angiography usually shows the fistula.

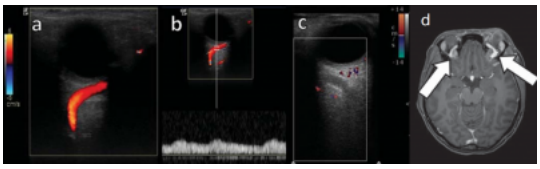

Figure 21:Image diagnosis confirmation: a) Blood flow in superior ophthalmic vein at color doppler study b) arteralized at spectral Doppler superior ophtalmic vein with low resistance flow, c) Superior ophthalmic vein, d) Magnetic resonance correlation in contrast-enhaced T1-weighted sequence. Modified from Santos et al.

Treatment: The usual treatment for a C-C fistula is closure of the leak by endovascular treatment. If endovascular procedures fail, open surgery is indicated.

Note

If you suspect a C-C fistula, call your local neurologist/neurosurgeon to perform transcranial Doppler.

In text References

(Fusco and Harrigan. 2011; Dos Santos et al. 2014)

References

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1. 2. Outcome from severe head injury

An integral part of ICU activity should be a registry where the long-term outcome of patients is recorded. This may be organised locally, regionally or nationally and helps to evaluate the clinical value and cost-effectiveness of intensive care medicine. In the specific field of TBI, outcome is more complex as disability rather than survival is a more sensitive outcome.

Intensive care physicians are familiar with the acute care of TBI but ICU treatment of the patient with severe head injury is only the beginning of a lengthy process of recovery which continues with early rehabilitation and ends with the patient returning, as nearly as possible, to his/her former life.

Gaining experience of recovery and rehabilitation after TBI is valuable linking of the ICU phase of care with outcome. This experience may help in decision-making in the ICU and may help to plan rehabilitation needs.

The broad outcomes that can be expected after severe head injury in adults are as follows; One third of patients die, one third recover, one third remain disabled. These data are drawn from ten large series of head-injured patients in industrialised countries.

Task

Test your knowledge

What factors are most likely to affect outcome?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

- the nature and extent of intracranial and extracranial damage
- the depth and duration of post-traumatic coma
- the patient's age
- general medical health and previous state of function
- the quality of available clinical care.

1. 3. Prediction of outcome

Much effort has gone into devising ways to predict outcome from TBI as:

- the condition is serious
- it consumes expensive resources
- recovery is prolonged
- relatives understandably wish to be given accurate information about prognosis.

Prediction of outcome is basic to decisions concerning diagnosis, prognosis and therapy. Unfortunately even the most sophisticated prediction models have an individual accuracy of only 80%, and can therefore only supplement and not supplant clinical judgment. Families often find it hard to live with such uncertainty for so long and require much support. You should consider your role in this regard in the early stages of managing a patient with TBI.

Note

The factors of most use in assessing outcome after severe head injury during the early days and weeks are indicated below.

Recovery is most rapid in the early weeks. Unfortunately, this can lead to over-enthusiastic prediction of long-term outcome (especially of cognitive function) by those with limited clinical experience of the later stages of rehabilitation. Over 90% of patients have reached their final category in terms of the relatively crude Glasgow Outcome Scale (see below) at six months, and most international trials on head injury still focus on this measure of outcome. With optimum rehabilitation, further gains can be made over a much longer period, up to two years and sometimes longer in the case of younger adults.

Note

Most recovery occurs within six months.

In text References

(Murray et al. 2007)

References

- Murray GD, Butcher I, McHugh GS, Lu J, Mushkudiani NA, Maas AI, Marmarou A, Steyerberg EW., Multivariable prognostic analysis in traumatic brain injury: results from the IMPACT study., 2007, PMID:17375997

1. 4. Measurement of outcome

Outcome after TBI is measured according the Glasgow Outcome Scale (GOS), originally published in 1975 by Jennett. This scale is in 5 points – dead, vegetative state, severely disabled, moderately disabled, and good recovery. For analysis, it is common to dichotomize outcome as favourable (good recovery and moderate disability) or unfavourable (severe disability, persistent vegetative state). More recently (1998) each of the top three categories, good recovery, moderate recovery, severe disability have been split into a further two levels (upper and lower) to improve discrimination.

The score should be obtained during a personal visit and interview with the patient. However, for practical reasons, a postal and/or a telephonic interview are frequently used. Both scales apply a standardised questionnaire and have been validated.

(Tsyben et al. 2018) compared in 2018 the Glasgoow outcome score (GOSE) with the SF-36 questionaire, generalised linear mixed model (GLMM) to predict physical and mental outcome in TBI observing that still exist tremendous variability between scores, GOSE and outcomes suggesting additional factors that may play a role in determining outcome.

Other reviews observed existing models for predicting prognosis in the acute phase after TBI are not situable for use at the individual patient level and should not be used to set limits for acute care interventions

Interpretation of outcome results for each department should be standardised to patients' severity. Observed outcomes should be compared with the expected outcomes. Standardisation can be done, using a model based on logistic regression (IMPACT) and comparison of GOS scale values through the sliding dichotomy concept.

In text References

(Jennett and Bond. 1975; Teasdale et al. 1998; Murray et al. 2005; Steyerberg et al. 2008; Tsyben et al. 2018; Carter et al. 2016)

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1. 5. Audit

Challenge

You should regularly review the outcome of your patients and compare the treatment results of your ICU with the international standards outlined above. Do not use 'outcome at discharge' but try to follow up your patients for six months at least. This may require meeting them and their families to discuss what gains have been made and what problems have emerged during the rehabilitation process. Rehabilitation facilities vary considerably throughout Europe, and you should try to visit those relevant to your institution and understand how they form another part of the clinical chain of care.

Many patients with severe head injury die. It is good practice for those who were involved in the care to hold a mortality conference to review the patient's injuries and the management in an open and non-judgmental way. This process of audit or quality improvement is becoming increasingly fundamental to clinical practice as it encourages efforts to improve the quality of patient service.

Doctors and ICUs need to know the outcome of their patients weighted on the basis of illness/injury severity. Using a few variables, usually not more than ten, you can build a database and apply the formula used by established prognostic models (IMPACT). If possible, try to obtain mature data from organisations who build the prognostic model to compare its performance with your data, thus creating a

calibration model. In such way you can build an observed/predicted ratio concerning mortality or severe disability. Monitoring your data will allow early recognition of higher than expected rates of death or severe disability

Task

Rewiew the IMPACT website and calculate the IMPACT score for 2 of your patients.

2. Conclusion

As there is no effective treatment of primary brain damage, the key issues for the intensive care practitioner, in the early management of patients with traumatic brain injury, are to prevent and/or minimise the risk (and effects) of secondary brain injury. Ascertaining the severity of the primary brain injury at the earliest possible opportunity is important as this will assist you in:

1. detecting secondary brain injury
2. determining the effects of treatment and
3. predicting outcome.

Intensive care management of patients with severe TBI comprises high quality general care and various strategies aimed at preventing or reducing secondary brain damage while the underlying pathology is resolving. The primary focus is to maintain, within an acceptable range, the increase of ICP and to maintain adequate cerebral perfusion with oxygenated blood. The management should be individualised on the basis of physiological knowledge.

It is important to maintain prognostic balance in every step of the care to avoid both undertreatment and overtreatment. In ICU, control of the factors involved in maintaining the cerebral perfusion pressure – such as arterial and venous pressures, intracranial pressure and pCO_2 is mandatory. Avoidance of metabolic derangements such as hyperglycaemia and hyponatraemia is also important and meticulous attention to all aspects of Critical Care management is a key factor in improving outcome after head injury.

2. 1. Key points in TBI

1. TBI is defined as brain injury caused by external force or trauma
2. The main cause of TBI in high income countries is ROAD TRAFFIC ACCIDENTS related trauma and in low and middle income countries is VIOLENCE related trauma
3. The main role of the ICU in TBI is to facilitate the dynamic management process in TBI patients since the prehospital stages to the transition to ward and/or rehabilitation service in order to treat and prevent all possible TBI consequences and long term complications
4. The main aim in the early management of patients with TBI is to prevent and minimise the risk of secondary brain injury and treat emergent threats from primary injury
5. Clinical history is a very important tool in the assessment of TBI patients and must include the neurological state, mechanism of injury, past medical history and medications and possible medical reasons that triggered the accident
6. Immediate assessment must include ABC steps in order to secure airway, adequate ventilation and organ perfusion
7. SpO_2, $etCO_2$, SBP and MAP are important tools in the management of TBI patients
8. Fluid resuscitation can be guided by multiple parameters including dynamic haemodynamic parameters of fluid responsiveness which can be measured by ultrasound, PiCCO and LIDCO systems or photoplethysmography
9. Isotonic crystalloids are first line solutions in fluid resuscitation
10. Colloids, hypotonic solutions and hypertonic solutions must be avoided in fluid resuscitation
11. The ICP value >25mmHg is an independent trigger to lower ICP
12. There’s no difference between mannitol or hypertonic solution in osmotherapy
13. Serum osmolarity, blood pressure and electrolytes must be monitored during osmotherapy
14. Use of tranexamic acid (TXA) is not supported by evidence in isolated TBI. Current evidence supports use of TXA in poly-trauma patients, including those with TBI
15. Neurogenic pulmonary oedema and cardiac stunning after TBI are the result of catecholamine storm in response to acute increase of intracranial pressure after trauma
16. Basic monitoring for patients with TBI includes: ECG, vital signs, pulse oximetry, capnography
17. Admission laboratory for patients with TBI must include: haemoglobin, leukocytes, platelets, electrolytes, coagulation parameters, blood type, urea and creatinine, liver enzymes
18. Coagulation must be closely monitored specially in patients with history of anticoagulants or antiplatetelet agents
19. In patients with Vitamin K antagonist treatment, this treatment must be suspended and the effects be reverted
20. CT Trauma scan includes Head, cervical spine CT, angiography of thorax, abdomen and pelvis
21. The main objective of ICU staff regarding patient’s relatives communication is to provide reassurance, regular updates on progress and obtain supplemental important such as medications taken by the patient and must be led by a senior member of the staff
22. Neurological examination and ICP monitoring are extremely important to guide management and calculate prognosis, GCS is the main scale to evaluate general level of consciousness in patients with TBI
23. Referral criteria to specialised trauma centre are: need for neurosurgical intervention, extracranial severe TBI, Sylvian traumatic subarachnoid haemorrhage and patients with simultaneous intracranial and extracranial lesions