Chapter 11

Management of Intracranial Pressure in Traumatic Brain Injury

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Additional information is available at the end of the chapter

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Abstract

Traumatic brain injury (TBI) is the result of an external force acting upon the head, causing damage to the brain. The severity of injury, mechanism by which the injury occurs, and the frequency of the high-force impact all play a role in the determination of a TBI. TBI describes a wide range of traumatic pathologies which is comprised of damage done to a multitude of cranial central nervous system components. TBI patients typically present with a series of symptoms are correlated with the presence of an intracranial injury, such as physical/cognitive difficulties. A major concern associated with intracranial injuries is the management of intracranial pressure (ICP), a resulting factor of a TBI which facilitates into intracranial hematoma and/or cerebral edema. These conditions have adverse effects on one's brain, and the immediate management and relief of intracranial pressure are crucial in avoiding hydrocephalus and brain herniation, conditions which lead to sensory loss and even death. In this chapter, we will begin by thoroughly understanding what a TBI is, its clinical presentation, and the first-tier examination to determine severity. Then, we will progress into the anatomy of the brain, followed by a thorough investigation into intracranial pressure management strategies and prognosis.

Keywords: intracranial, ICP, trauma, head injury, brain herniation, cerebral edema, hydrocephalus, shunt, skull, blood, cerebral fluid, pressure, relief

1. Introduction

The onset of increased intracranial pressure is often attributed to many pathologies such as large artery acute ischemic stroke, intracranial neoplasms, or disorders such as meningitis. The most common reason for which the onset of intracranial pressure is observed is due to

traumatic brain injuries, such as colliding one's head into a hard object as a result of an accident. By definition, an intracranial pressure that exceeds 20 mm Hg is considered high and indicative for the need of immediate treatment [1]. Through the advancement of medicine and technology, the variety of treatment options available to relieve patients of increased intracranial pressure has grown tremendously. In practice today, there exists a multitude of treatment options ranging from nonsurgical interventions to surgical interventions [2, 3]. In this chapter, we will discuss the primary pathology of patients presenting with increased intracranial pressure (ICP) as a result of traumatic brain injuries (TBIs), and we will take a generalized perspective on this pathology by discussing a multitude of topics we find crucial to your understanding of the management of ICP in TBI patients.

2. Intracranial pressure management of traumatic brain injury

2.1. The occurrence of traumatic brain injury

Traumatic brain injury (TBI) is composed of an external mechanical force, whether it be a change in acceleration or impact by projectile that causes a temporary or at times a permanent brain function impairment as well as physical damage to the human brain anatomy. It is important that we establish a clear understanding of the term TBI and its partnering term non-TBI. A traumatic brain injury is brought on by the impact generated by an external force, while a nontraumatic brain injury is brought on by internal forces such as a stroke or infection. A traumatic brain injury, which we have now learned arises from external forces, can come in two pathological forms: penetrating and nonpenetrating. This classification presents as simply as it is defined. A penetrating TBI results in several lesions starting from one's head down to the cerebral level, and these often occur in severe accidents or injuries. A clear and prime example of a penetrating TBI is one that occurs all too often to members of our military, a foreign projectile being discharged from an external high-force machine, which then strikes a human head [4]. A nonpenetrating TBI is the form that we will cover more in depth within this chapter and results from an external force acting upon the head, but it does not penetrate any layer of human anatomy. Within the clinic, the formal classification of TBIs may be reduced to open-head injury for patients presenting with traumatic brain injuries of the penetrating type, and for patients presenting with a traumatic brain injury of the nonpenetrating type, the term closed head-injury may be assigned [5]. The simplification made to these terms adds an element of simplicity for when medical professionals present cases to patient's families and loved ones.

2.2. Anatomical description of the brain, relevant to penetrating traumatic brain injuries

To aid in our understanding of traumatic brain injuries and later on the rise of intracranial pressure, it is imperative we touch upon the anatomy of the human brain such that successive sections of this chapter can be understood with a greater degree of clarity. The human brain, the core of the central nervous system, controls a vast majority of bodily processes and functions. The center of knowledge and core processing is perhaps the most important regulator

of human life, yet only weighs between three to five pounds. The first line of defense for the brain is called the cranium, often referred to as the skull, and this shields the brain with a tough bone structure [6]. The brain covering itself contains three layers: the dura, arachnoid and pia. Interestingly enough, there also exists a space between the pia and arachnoid referred to as the subarachnoid complex. This area houses a vast network of veins, arteries, and nerves, which channels both blood and electrochemical potential to the heart and back to the brain. This subarachnoid complex is prone to trauma as well as constriction or full blockage. Any trauma that may cause constriction or blockage will also pose a greater threat to the tissue of the brain. Thus far, we have discussed the cranium and the brain that it encloses; however, the brain does not fill the entire volume of the cranium, and the volume remaining is filled by cerebrospinal fluid (CSF), serving as a nutrient-rich cushion around the brain, and blood-vessels. It is important to note that the volume of the cranium is fixed, and the brain fills a fixed volume of the cranium and the cerebrospinal fluid (CSF); therefore, any trauma that may alter the volume of the cranial region can be devastating, a concept referred to as increased intracranial pressure (ICP), which we will discuss in immense detail throughout this chapter [7].

A common misconception we wish to clear up is the designation of the upper and lower brain, and it is often misunderstood that the brain is a term used only for the upper brain, the ovular shaped region; however, the lower brain that houses vital components such as the brainstem is also indeed part of the human brain. At the lowest point of the brain (brainstem), there exists a small circular opening for which the skull and the spinal cord merge to form the complete central nervous system. As we mentioned above, the brainstem in fact is one of the most important parts of the lower brain as it houses a plethora of intricate nerve fibers, which pass information from the brain to the spinal cord and to the body as a whole. Another crucial component of the lower brain is the cerebellum, a small mass of neuronal tissue that is responsible for the regulation and coordination of motor skills and balance. Without this region of the brain, the miracle of the human touch, whether it be the detailed touch of an artist or the intricate lifesaving work done at the microscopic level by a neurosurgeon, will not be possible. The ovular region of the human brain, known as the upper brain, is a large mass of neuronal tissue divided into white and gray matter. Gray matter houses neuronal cell bodies, axons, and dendrites, while white matter is entirely made up of axons, which connect other gray matter components together [8]. The upper brain is composed of the cerebral cortex, which is the largest component of the human brain, and this region is divided into two hemispheres: left and right. Uniquely enough, the right side of the brain will control left side of the body and vice versa. Despite the left and right hemisphere designations of the cerebral cortex, there are also various other designations called regional designations, and these include the frontal lobes, temporal lobe, parietal lobe, and occipital lobe. The frontal lobes are comprised of the left and right lobes, which are located directly behind the forehead, and these control one's intellectual abilities, decision making, behavior, and emotions. The temporal lobe is behind the ear and extends to the center of the head; from a bird's eye view of the brain, it is directly behind the frontal lobe and extends outward from both ears. This lobe controls speech, understanding, memory, and information retention [9]. Our ability to read, write, and understand spatial relationships is due to the efforts made by the parietal lobe, which is located at the rear of the head, specifically the upper section of the convex ovular protrusion site. Also at the rear of the head, but significantly lower, is the

occipital lobe, which controls sight. While we have covered the brain as a whole, it is important to note that throughout the cerebral cortex there are also several sites that are denoted by specific names, which we will discuss when the pathology becomes relevant to those particular areas, and these sites are rich in nerves and also house nerve centers. These are called diencephalons; a more notable diencephalon is the hypothalamus, which regulates homeostasis of the body. These include control over body temperature, hunger, thirst, and arousal. Why discuss the anatomy of the brain to this extent? We hope that since we have discussed anatomy to this extent, the discussion of traumatic brain injury to regions of the brain can be better understood. Damage to any area of the brain can result in both impairment to the functions they regulate and permanent damage to the physical anatomy leading to cognitive deficiencies as well [10, 11].

2.3. Classifying traumatic brain injuries using GSC

The immediate identification of traumatic brain injuries is crucial for the positive-outlook prognosis of a patient, and injuries of this nature present in a spectrum of severities each consisting of unique clinical presentations. To simplify the spectra of severity, a classification system has been established that rates injuries in three categories: mild, moderate, and severe. This classification system is called the Glasgow Coma Scale (GSC), a system readily utilized for the classification of thousands of traumatic brain injury cases per year [12]. The Glasgow Coma Scale is used in evaluating a patient's level of consciousness based on a sum of several categories ranging between 3 and 15. Evaluation of patient consciousness is based upon his or her responsiveness to general verbal, visual, and motor stimuli [13]. The numerical score of this assessment will classify the severity of a patient's brain injury; a score closer to 15 demonstrates near-full neurological ability and consciousness, while a score closer to 3 demonstrates a case in which severe brain injury has occurred and the patient is in a deep coma. A Glasgow Coma Scale score of 13–15 indicates a mild brain injury, while a score of 9–12 indicates a moderate brain injury, and any patient scoring 8 or below is said to have incurred a severe brain injury [12, 14]. **Table 1** demonstrates the rating categories medical staff use to generate a Glasgow Coma Scale score.

To generate the Glasgow Coma Scale (GSC) score, a score will be determined for each category, followed by the summation of all three categories to generate a score between 3 and 15.

2.4. Introduction to intracranial pressure

An accumulation of pressure above the normal standard within the skull is denoted as elevated intracranial pressure (ICP), a severe condition that requires immediate remediation. While the

	1	2	3	4	5	6
Visual	Eyes closed	Eyes open to sharp stimuli	Eyes open to sounds	Eyes open without induced stimuli		
Motor	No movement	Movement to sharp stimuli	Muscle flexion to sharp stimuli	Muscle flexion and bodily movement	Able to localize touch	Appears to have normal movement
Verbal	No sounds	Slow intensity sounds	Incoherent words	Understandable words spoken	Normal conversation	

Table 1. The Glasgow Coma Scale (GSC) rating score sheet.

initial cause for the onset of ICP may vary greatly from patient to patient, the anatomical factors that play a role in intracranial pressure are simply the cranium, the brain, and the cerebrospinal fluid that fills the volume in between the cranium and the brain. Within medicine, the standard unit to measure pressure is "mm Hg," which stands for millimeters of mercury, the distance mercury travels in a closed system to indicate pressure. For a normal adult, at rest, and in good health, the intracranial pressure should remain between 6 and 16 mm Hg [15]. A unique high-order organismal advantage humans possess is the ability to maintain homeostasis, much like many functions of the body, and homeostasis is crucial to the long-term survival of the human. Within the brain, there are also many hemostatic mechanisms in place to maintain a healthy and acceptable pressure within the cranium. The management of intracranial pressure is in fact done through the regulation of the metabolism and production of cerebrospinal fluid (CSF). Since CSF is the only liquid occupying the volume between the cranium and brain, there is no other regulation factor that the body can maintain. The size of the brain and skull only grows slightly after birth and cannot be altered freely to reduce pressure; thus, the regulation of CSF metabolism and production are crucial in maintaining a healthy and acceptable intracranial pressure [16]. In the event that intracranial pressure rises to the limits of the normal and healthy range, immediate remediation is necessary. When the ICP reaches 17–18 mmHg, concern should be raised, and when ICP ranges between 19 and 25 mmHg, immediate relief of pressure is required to prevent damage to regions of the brain [17].

2.5. Monro-Kellie hypothesis

The Monro-Kellie hypothesis was proposed by Doctors Alexander Monro and George Kellie in correspondence to the impact cerebrospinal fluid (CSF) has on the pressure-volume relationship within the cranium. This particular hypothesis describes the intracranial volume-pressure relationship, which we briefly mentioned above. According to the Monro-Kellie hypothesis, the fixed volume of the cranium is comprised of the brain, cerebrospinal fluid, blood, and the pressure of blood flowing to the brain called cerebral perfusion pressure (CPP). Within the fixed volume, the cranium and all components within come to form a state of equilibrium, which we discussed as homeostasis. This hypothesis states that an increase in volume of any one of the cranial constituents results in an increase of pressure within the cranium unless there is an equal or greater reduction of volume in another cranial constituent [18]. Buffers within the cranium respond to increases in cranial constituent volume in hopes to reduce pressure to avoid brain damage. In the event that cranial pressure rises, typically due to an increase in lesion volume, a decrease in blood and cerebrospinal fluid is observed in hopes to reduce intracranial pressure [19].

2.6. Rise of intracranial pressure resulting from brain injury

The most common cause of increased intracranial pressure, also known as intracranial hypertension, is traumatic brain injuries. The neurological complication that is accompanied by a traumatic brain injury is the loss of pathophysiologic regulators of the brain that results in deregulation of intracranial pressure management [20]. The volume of an average adult's skull is approximately 1500 mL, in which over 85% is occupied by the brain, 10% by arterial blood, and 5% by cerebrospinal fluid [21]. Cerebral profusion pressure (CPP), which we briefly mentioned above as the pressure created by cerebral blood flow, is dependent on two factors: both mean systemic arterial pressure (MAP) and ICP. Mean systemic arterial pressure (MAP) and intracranial

pressure (ICP) are related to cerebral perfusion pressure (CPP) based on the relationship that: CPP = MAP – ICP. Mean systematic arterial pressure (MAP) is calculated by the summation of one-third systolic blood pressure (SBP) and two-thirds diastolic blood pressure (DBP), in abbreviated form as: MAP = 1/3 SBP + 2/3DBP [22]. In line with the Monro-Kellie hypothesis, an increase in intracranial pressure (ICP) is remediated physiologically by a decrease in cerebral perfusion pressure (CPP) [23]. Through the relationship we described above, a decrease in CPP dictates that there is a decrease in blood pressure and therefore autoregulation of intracranial pressure. While a decrease in CPP regulates ICP, it is also vital that a minimum CPP be maintained such that the brain can receive adequate amounts of blood. Normal CPP ranges between 50 and 165 mmHg, and in the event that CPP drops below 50 mmHg, the brain will not receive adequate amounts of blood, thus creating further complications with maintaining normal cerebral blood flow [24]. When the brain is subjected to injury, the physiological homeostatic functions of the brain may be deregulated or not functional at all. A normal pathology will maintain the normal CPP of 50–165 mmHg while regulating an appropriate ICP level as well. In the event that the intracranial pressure (ICP) rises past 16 mmHg, blood vessels within the brain will constrict to reduce the blood to flow to cranium thus lowering the intracranial pressure. Thus, when a traumatic brain injury occurs, to the extend where the brain's homeostatic functions are lost, intracranial pressure (ICP) increases and physiological regulatory functions are nonoperable [25].

2.7. Negative outlook of untreated increased intracranial pressure

The management of an increased intracranial pressure is vital for the successful outcome of the patient. In the event that an elevated intracranial pressure goes untreated, two major complications arise. The first is the temporary or permanent loss of vision (depending on severity) and the second is development of a severe headache that lasts for more than 48 hours [26]. Additionally, patients will also begin to exhibit irritability, lethargy, slow cognitive processes, as well as abnormal behavior. Untreated elevated intracranial pressure may subject the patient to enter a state of near-unconsciousness, coma, or even death [27]. Another concern for patients presenting with elevated intracranial pressure is the possibility for damage incurred through brain herniation. Brain herniation is a deadly condition that arises when the ICP is extremely high, and this condition presses the brain tissue against the hard cranium causing compression damage to the brain. This extreme pressure may also cause the brain to shift across vital structures that connect the brain to the spinal cord, such as the falx cerebri [28]. While brain herniation may also occur in the absence of an elevated ICP, it is more frequently seen in patients that do in fact present with a severely elevated ICP. High pressure within the cranium induces brain herniation and that can constrict or block arterial blood flow to various parts of the brain, proving to be fatal. In **Figure 1**, the CT scan of a patient presenting with left-side brain herniation of the parahippocampal gyrus, a structure of the brain that is paramount in memory encoding and memory retrieval, is shown, and damage to this area may result in memory disturbances and schizophrenia [29].

2.8. Clinical presentation of patients with ICP resulting from TBI

Patients presenting with elevated intracranial pressure (ICP) resulting from traumatic brain injury (TBI) exhibit symptoms very much similar to patients presenting solely with elevated ICP due to other factors. Traumatic brain injury patients, depending on severity, will present

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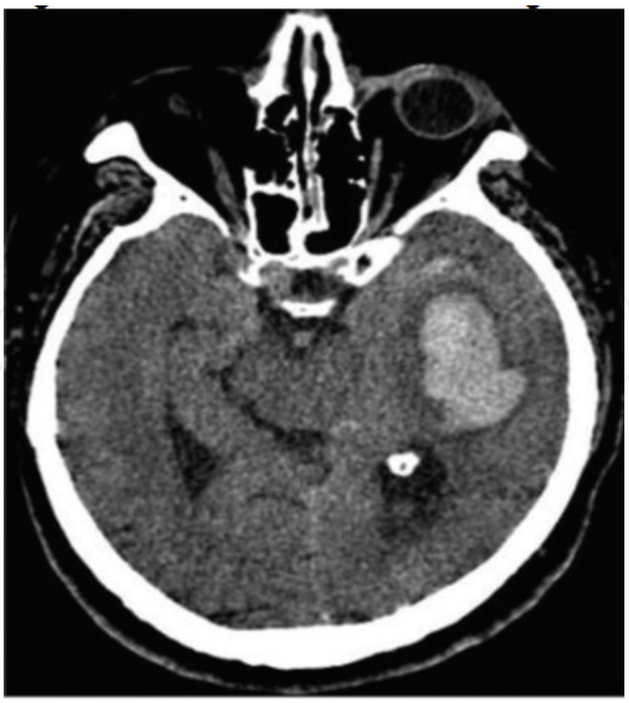

Figure 1. CT scan demonstrating brain herniation on left side due to temporal lobe hemorrhage [29].

with a series of symptoms that are generally along the lines of unconsciousness/coma, headache, vomiting, nausea, compromised motor function, blurred vision, headache, perception of noise that is not present (ringing sounds), as well as difficulty keeping balance. The one symptom we did not mentioned above that we will discuss in depth now is elevated intracranial pressure resulting from traumatic brain injury. Alongside all the symptoms a TBI patient will exhibit, ICP will also cause several symptoms to be present much similar to those already seen in TBI patients [30]. Typical elevated ICP patients present clinically with headache, vomiting, nausea, reduced state of consciousness, and vision blurriness. **Figure 3** demonstrates the overlap of the symptoms a TBI patient will exhibit versus symptoms an elevated ICP patient will exhibit. But do note that a common symptom of a TBI patient is in fact also elevated ICP, but elevated ICP in not only brought on my a TBI, and other factors may contribute to elevated ICP such as hydrocephalus and intracranial hemorrhage [33].

Now that we understand the clinical features of traumatic brain injury (TBI) patients as well as elevated intracranial pressure (ICP) patients, let us combine the two as one pathology, elevated intracranial pressure due to a traumatic brain injury. Referring back to **Table 2**, we know how similar the presenting symptoms may be; thus, let us briefly discuss how a diagnosis may be made. First and foremost, if the history of the patient prior to clinical presentation involves any form of trauma to the head, a TBI can be easily diagnosed. The next step will be to conduct a neurological evaluation, often using the Glasgow Coma Scale (GCS). A symptom of a TBI may be elevated intracranial pressure (ICP), which can be diagnosed primarily through a neurological exam conducted by a neurologist or neurological surgeon. Additionally, radiological imaging via computed topography (CT) scan and magnetic resonance imaging (MRI) can be utilized to determine the presence of the cause as well as the severity of the elevated intracranial pressure [34].

Clinical presentation	TBI patient	ICP patient
Coma—unconsciousness	Present	Present
Headache	Present	Present
Vomiting—nausea	Present	Present
Reduced motor function—balance	Present	Present
Vision deficits—blurriness	Present	Present
Ringing of ear	Present	Not Present
Elevated intracranial pressure (ICP)	Present	←**Symptom of TBI**

Table 2. Comparison of TBI and ICP symptoms.

Regions of common trends in radiological findings
Optic nerves
Bilateral venous sinus stenosis
Lesioned ventricles
Enlarged arachnoid
Cerebellar tonsil
Subdermal adipose tissue accumulation

Table 3. CT and MRI radiological findings for ICP resultant of TBI.

2.9. Radiology of intracranial pressure caused by TBI

Radiographic imaging to determine the presence of a traumatic brain injury that results in the onset of elevated intracranial pressure is crucial for the high-certainty diagnosis medical professionals seek to provide. Radiographic methods utilized in the diagnostic process are CT scans and MRI, and over the years, a series of common trends have been documented in regard to radiological findings, which we will discuss in this section. In **Table 3**, we highlight the core radiological findings that we will discuss in this section.

The second cranial nerve, also known as the optic nerve, transmits visual information from the retina through a complex nervous network to the brain. This cranial nerve develops from optic stalks during early embryonic development and is supported by nonneuronal glial cells [35]. In patients presenting with elevated intracranial pressure, the optic nerve region of the CT and MRI scan shows a clear area of prominence. Within this region, approximately 40% of elevated ICP patients present with optic nerve tortuosity, a condition in which optic nerve is twisted or alerted slightly from physiologically normal conditions [36, 37]. In approximately 45% of patients, the subarachnoid space surrounding the optic nerve is highly prominent and protrudes the space of the optic nerve [38]. This region is comprised of delicate connective tissue as well as channels that contain cerebrospinal fluid (CSF), and this region plays a role is creating channels for intercommunication between the arachnoid, the pia mater, and the CSF. The optic disk, also referred to as the optic nerve head, is the terminal point for ganglion cells leaving the eye. The region is a physiologically normal blind spot each eye possesses due to the lack of

photoreceptors, rods, and cones in that region [39]. A condition involving the swelling of optic disk is called papilledema, and in patients that present with elevated ICP, this is a common occurrence [40]. Due to the fact that the optic disk is continuous with the subarachnoid space, swelling of this region contributes greatly to elevated intracranial pressure. This swelling can manifest itself into two forms: either as an intraocular protrusion of the optic nerve or as seen in a vast majority of papilledema patients, a flattening of the posterior white of the eye, sclera [41]. In **Figure 2**, the nodular enhancement of the optic nerves is seen, from a case study on irregular papilloedema [42]. The last pathology we will discuss in relevance to the optic nerve is the MRI enhancement of the intraocular optic nerve, which is anterior to the sclera's lamina cribrosa. This pathology is seen in approximately 50% of elevated ICP due to TBI and adds to the intracranial pressure via degeneration of optic nerves, causing damage to axonal components leading to increased ICP and irreversible blindness in a vast majority of glaucoma patients [43].

Another pathology consistent with the increase in intracranial pressure is bilateral venous sinus stenosis, which may be prevalent in segments of the transverse sinus. Stenosis involves the constriction of a venous tract, in this case of the sinus [44].

Pseudotumor cerebri (PTC) is a clinically relevant pathology that presents with increased intracranial pressure, but the etiology is not entirely understood. This syndrome tends to target women over men, and specifically obese women. PTC, as mentioned above, results from increased ICP and presents clinically with headaches, nausea, as well as changes in vision. Through radiographic efforts, this pathology is linked with elevated CSF, a connection that was dismissed in the earlier years of medicine. Interestingly enough, patients with elevated ICP who also present with PTC have the following pathologies: an empty sella, an enhancement of the optic nerve head, and a tortuosity of the optic nerve [45]. An empty sella, otherwise known as empty sella syndrome (ESS), is where the pituitary gland is physically altered, and thus the sella turcica becomes filled with CSF [46]; see **Figure 3**. ESS is usually highly indicative of an increased intracranial pressure.

Thus far, we have discussed three of the most common radiological findings, in a nonspecific order. The next pathology we will discuss is the enlargement of the arachnoid resulting from an increase of ICP due to TBI. ESS, or empty sella syndrome, we just discussed also plays

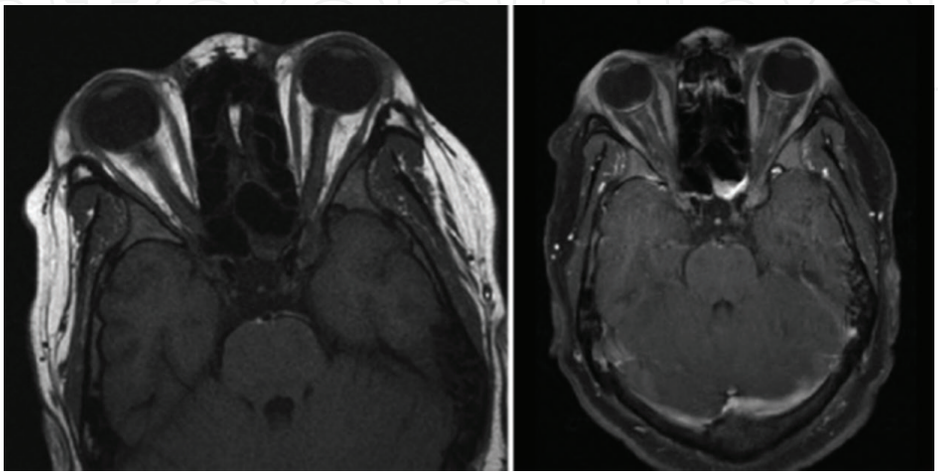

Figure 2. Papilledema, enhancement of optic nerve leading to increased ICP [42].

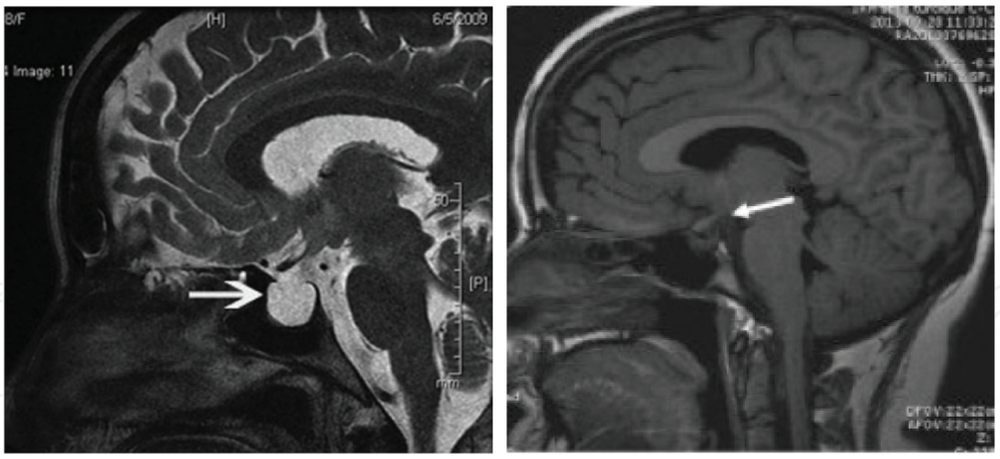

Figure 3. Sagittal T2-weighted image of ESS in an elevated ICP patient [31, 32]. (Left) Pathological findings of "empty sella syndrome" where the arrow points at a completely empty sella. (Right) Anatomically and pathologically healthy individual, for comparison.

a major role in the enlargement of the arachnoid. Actually, approximately 70% of enlarged arachnoid cases due to increased ICP are because of empty sella syndrome, replacing the void volume of the pituitary gland with CSF [47, 48]. Some cases of arachnoid enlargement are attributable to an enlarged Meckel cave [49]. The Meckel cave, previously known as the trigeminal cave, is a CSF-filled arachnoid pouch, which protrudes from the posterior cranial fossa, the most posterior segment of the cranium base where the cerebellum and brainstem reside. In the event of a TBI, cerebrospinal fluid (CSF) fills into this cavity, expanding the volume of this cavity, resulting in an increase of intracranial pressure due to the narrow and space-limited anatomy of this brain region [50, 51].

The next pathology that we will discuss that is commonly observed in radiological investigations of increased ICP in TBI patients is tonsillar ectopia, synonymous to cerebellar tonsils, a disorder of the papa-axial mesoderm. In this pathology, the cerebellar tonsils elongate due to pressure, leading the cerebellum to be pushed through the foramen magnum of the cranium resulting in additional increased intracranial pressure as well as tonsillar herniation [52]. This condition is life threatening, as cranial pressure is heavily diverted onto the medulla oblongata, a vital sector of the brain that controls cardiac and respiratory functions [53, 55].

As we have now discussed the most common radiological presentation for patients presenting with increased intracranial pressure due to a traumatic brain injury, we will begin our discussion on the treatment pathways to remediate these issues. Management and treatment for increased intracranial pressure can utilize both a nonsurgical and surgical intervention, and in the sections to follow, we will discuss both management options.

2.10. Nonsurgical care of increased intracranial pressure

Nonsurgical management of increased intracranial pressure can take on a multitude of forms. In this section, we will discuss the many medicinal options available to patients presenting with increased intracranial pressure resulting from a traumatic brain injury. The quickest and least

invasive method to reduce a patient's ICP to a normal range is by elevating the patients head to 30° with respect to the horizontal plane [56]. The elevation of a patient's head does not directly act toward lowering the ICP; in fact, an elevated head aims to reduce CPP, which in turn reduces ICP by increasing venous drainage (based on the relationship we discussed in an earlier section of this chapter) [57]. A common practice typically conducted by first responders to patients that show clear signs of a traumatic brain injury and increased intracranial pressure in the field is to medically induce a state of minimal hyperventilation. In 1970, hyperventilation of a patient with an increased ICP was a common practice and used readily, and it was not until a study came out stating the adverse outcomes of a prolonged state of hyperventilation; in fact, it was shown to have caused cerebral ischemia, a condition where an insufficient amount of blood is delivered to the brain. Today, hyperventilation is still used in the treatment of an increased ICP but only to a pCO_2 level of 25 mmHg. Immediate initial treatment may call for the hyperventilation of a patient to pCO_2 levels of 30–40 mmHg but for no greater than 2–5 min, before returning back to 25 mmHg. More often than not, hyperventilation is not necessary for the treatment of an increased ICP, and there are better methods that were developed compared to the hyperventilation that was introduced in the 1970s [58, 59]. An organic biological medication, mannitol, which is a sugar alcohol typically administered intravenously is used to decrease high blood pressure in the eyes as well as to decrease intracranial pressure [60]. With effects seen 10–20 min after administration and lasting up to 8–10 h, mannitol is metabolized by the liver and excreted mostly by the kidney. Mannitol has a biological half-life of 100 min and is mostly a synthetic drug, with only a 7% bioavailability [61]. The biochemical mechanism by which mannitol acts on human physiology is bimodal, meaning the drug can mechanistically act in two ways. The first pathway is to lower osmotic diuresis through the reduction of swelling within the cerebral parenchyma. The second pathway is to lower the viscosity of the blood, therefore allowing for more laminar blood flow through veins and arteries eventually causing a state of vasoconstriction, which decreases the intracranial volume of blood and thus a lowered intracranial pressure [62, 63]. The mechanistic bimodal action by mannitol makes the drug a popular choice among patients presenting with an increased ICP due to TBI [64]. A class of drugs known as barbiturates has been widely debated in its effects on lowering intracranial pressure, while this class of drugs can successfully complete the task at hand, the use of barbiturates often causes a state of decreased myocardial function and decreased CPP, which can cause higher rates of morbidity and mortality if left unmonitored. Thus, we do not advocate the use of barbiturates as a drug for the treatment of elevated ICP, unless as a last resort option. For your understanding, a barbiturate is a synthetic chemical drug that acts as a depressant of the central nervous system capable of producing a wide spectrum of effects [65]. While barbiturates are not a recommended class of drugs to utilize in the treatment of elevated ICP due to TBI, it in fact can and has been utilized as a last resort option for a procedure called "barbiturate-induced coma," which aims to immediately reduce intracranial pressure in patients that are unresponsive to any other form of nonsurgical medical treatment. The barbiturate of choice for this procedure is pentobarbital, which requires electroencephalogram (EEG) monitor during intravenous use. While using this drug, it is of key importance to monitor the blood pressure of the patient such that the patient does not slip into a hypotensive state. Hypotension resulting from the use of a barbiturate dramatically increases the rate of mortality two-fold, from 25 to 50% mortality [66]. In addition to the intravenous medication administered to patients presenting with increased ICP due to TBI, the administration of a

hypertonic solution to maintain a state of euvolemia, a condition in which bodily liquid volume, viscosity, and circulation are all normal, is also imperative. These hypertonic solutions depend on the results of a complete blood diagnostic panel and differ from patient to patient, and the most common intravenous hypertonic solution administered is a 0.8–8% NaCl solution [67]. In patients where intravenous euvolemia cannot be established and maintained, remediation of coagulopathy must be placed as a medical team's highest priority [68]. Coagulopathy is a state in which the blood's ability to coagulate is diminished or impaired, resulting in a variable viscosity of the blood and therefore further complications in the treatment of elevated intracranial pressure. Normal human physiology tightly regulates the viscosity of blood and its coagulating ability, and in patients that have suffered a traumatic brain injury, the release of biochemical pathway intermediate, thromboplastin, causes abnormal blood clotting. These abnormal clotting factors can be fatal if not remediated quickly; therefore, blood transfusions for these patients is the quickest and most preferred method in correcting blood coagulating abilities and eliminating coagulopathy. Patients on anticoagulating mediations due to high cholesterol, such as heparin or warfarin, who sustain a traumatic brain injury that results in elevated intracranial pressure are typically at risk and thus require immediate blood transfusion [69]. Approximately 1 in 10 TBI patients that present with elevated ICP also demonstrates a fever 24–48 hours after the initial injury, and this elevation in temperature is in fact part of the body's inflammatory response [70]. Often in patients where the hypothalamus has been damaged, elevated body temperature is noticed due to an underlying infection of the region. While the biochemical mechanism of this observation is not understood, what is understood is that immediate elimination of the infection is required for a successful recovery. This unexplained fever is often called "neurological fever," and unlike a fever caused by a cold or viral infection, neurological fever tremendously increases metabolic demand [71]. In patients with GCS scores less than or equal to 8 with imaging that demonstrates signs of cerebral edema, placement of external ventricular drain in a sterile fashion is recommended to allow CSF drainage. This procedure can be completed at bedside or in the operating room with the overall goal of decreasing cerebral edema by CSF drainage. In cases where intracranial pressure remains at or above 22 mmHg, despite all strategies discussed above, surgical intervention will need to be considered. In this section, we have discussed the nonsurgical and medicinal approach in resolving an elevated intracranial pressure; however, in any remedy for the management of ICP, it is important to remember that the overall goal is to reduce and prevent any agitation of the intracranial region. In the section to follow, we will discuss the surgical option for the management of intracranial pressure in the event that nonsurgical interventions do not remediate the issue.

2.11. Surgical care of increased intracranial pressure

Immediate and rapid surgical care of patients presenting with elevated intracranial pressure due to a traumatic brain injury is vital for the positive prognosis of the patient, especially if nonsurgical routes did not suffice in the remediation of intracranial pressure. Intracranial lesions resulting in an increased ICP typically present in patients as a state of reduced consciousness, and this pathology requires a surgical procedure called "rapid decompression." This surgery is self-explanatory at the elementary level, as the surgical efforts aim to reduce intracranial pressure ("decompress") and do so as soon as possible. Prior to operation, a complete patient

profile must be reobtained, meaning that radiological evidence alone is not enough to proceed with decompression surgery; a neurological examination coupled with radiological evidence that is convincing without any doubt is what surgical staff must aim to achieve. Similar to many forms of surgery, patient age plays a major role. For decompression surgeries, patients that are young (12 years old or younger) or elder (70 years old or older) pose a greater risk to surgical harm, and this harm is referred to as intracranial hemorrhage, excessive bleeding from tissue and venous tract within the intracranial region [72]. The first form of decompression surgery that we will discuss is decompressive craniotomy, a procedure in which a segment of the cranium is removed to relieve intracranial pressure and to create additional room for the brain swelling. This decompressive surgery evolved from a primitive form of surgery called trephining. Today, this surgical practice is a last resort option and has been more successful in younger patients rather than in older patients, another surgical option is craniotomy for evacuation of focal hemorrhage which can be subdural, epidural or intraparenchymal in nature [73]. The next surgical practice we will discuss that is used in the treatment of elevated intracranial pressure due to a TBI, which causes an over production of CSF, is a ventriculoperitoneal (VP) shunt. A VP shunt is a medical device that relieves pressure from the brain due to fluid accumulation, and this shunt drains the excess fluid and allows it to be metabolized and reabsorbed. Normally, CSF will coat the brain and spinal cord and be reabsorbed into the blood, and in a disrupted flow, the CSF can build up and create pressure on the brain causing damage. A common source for deregulation for CSF production and reabsorption is traumatic brain injury, and in these cases, it is common for CSF to cause damage to the brain [74]. Prior to surgery, a patient will be instructed to halt any consumption of food and water by mouth (PO) at least 8–12 h before surgery. Then a surgical nurse will prepare the area behind the ear for surgical incision. The shunt is a catheter, a thin flexible but heavy-duty tube that is used to drain excess liquid. A neurological surgeon will then make a small incision behind the ear and using a burr-drill will create a small hole within the patient's scalp [75]. With the hole in the cranium, surgical staff will insert one catheter into the brain and another subdermal catheter will be placed behind the ear. A thin tube will travel down the patient's torso and into the abdominal cavity. The excess CSF will drain into the abdominal cavity relieving intracranial pressure [76]. In patients that present with intracranial pressure regularly, a pump may be placed to activate this channel when ICP rises. For the context in which we have been discussing, this shunt will be used to relieve patients of increased ICP following a TBI.

2.12. Medication utilization

Management of elevated intracranial pressure due to a traumatic brain injury requires the utilization of many forms of medication. Thus far, we have discussed a few drugs that directly target elevated intracranial pressure, but often these pathologies require surgical intervention. For that to occur, a wide variety of drugs must be utilized to stabilize the patient from common complications that arise. In this section, we will touch upon the class of drugs utilized prior to neurological surgery. The most prevalent presurgical complication presenting in medical centers today is intracranial hemorrhages; see **Figure 4**. Intracranial hemorrhages denote bleeding of the brain; medical personnel often utilize prophylactic anticonvulsants, and the term prophylactic refers to the act of committing an action before hand and the term anticonvulsants refers to a set of

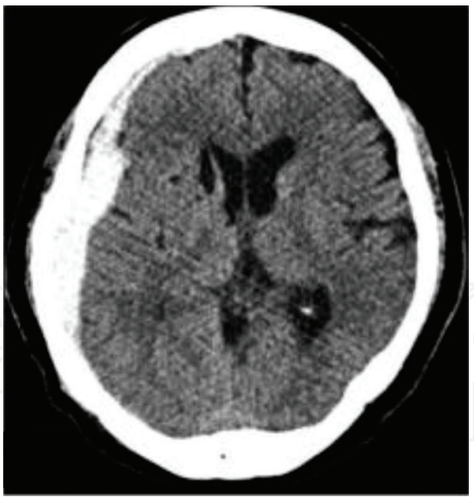

Figure 4. CT scan of subdural in a TBI patient presenting for surgical intervention [77]. Presentation of severe intracranial pressure buildup resulting from TBI. Images depict transverse CT images from ventral (left) to dorsal views (right), respectively, 5 h after injury with a Glasgow Coma Scale (GSC) score of 3 (an extremely severe form). This imaging depicts right focal subdural hematoma.

pharmacological drugs that block sodium channels or enhance GABA (gamma-aminobutyric acid) function [78, 79]. Physiologically, these drugs can save a life in the event of a seizure as well as reduce bleeding in the brain prior to surgery [80]. There are many drugs that can be used that are considered in this pharmacological class, and the first of its kind was discovered in 1882 (paraldehyde); today, the drug of choice is phenytoin or fosphenytoin [81]. In adults, a loading dose of phenytoin or fosphenytoin is administered, typically in adults 18 mg anticonvulsant per kilogram (kg) of patient body weight. Then, therapeutic levels of 20 milligram (mg) per deciliter (dL) are maintained until intracranial hemorrhage subsides [82]. Prolonged use of anticonvulsant drugs may result in gingival hyperplasia, an enlargement of one's gingiva (commonly known as gums) as well as randomized hair growth in men and unwanted male-hair growth in women, known as hirsutism [83–85]. In the unfortunate case where a pediatric patient is subjected to elevated intracranial pressure due to a traumatic brain injury that is accompanied by intracranial hemorrhage, the drug of choice changes to phenobarbital, where a 20 mg/1 kg body weight loading dose is given, followed by a therapeutic dose of 10–50 mg/dL.

2.13. Patient follow-up and future care

Many patients that present to medical centers for treatment of mild to severe traumatic brain injury and are subjected to an elevated intracranial pressure will tremendously benefit from numerous outpatient care options. As traumatic brain injury patients typically have difficulty with daily tasks, physical and occupation therapy is highly recommended as patients try to regain a normal lifestyle. Additionally, times following a traumatic incident can be hard emotionally and spiritually, and thus it is also beneficial for patients to receive counseling care from professional as well as from family and loved ones. The initial efforts to reestablish the life the patient once had is difficult and both mentally and physically taxing, and support and counseling are of key essence.

3. Conclusion

Throughout this chapter, we have discussed the many applications and forms of medical care pertaining to the presence of elevated intracranial pressure resulting from traumatic brain injury. Throughout this chapter, we hope that you have learned the key diagnostic characteristics, medical treatment, and future outcomes for patients experiencing this traumatic pathology. While we hope no patient has to suffer from TBI, we wish all medical staff best of luck in their efforts to remediate these conditions and for continual excellence in patient care.

Disclosure

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