Treatment of Oncologic Emergencies in the Neurocritical Care Unit

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Management of Intracranial Pressure

Overview

Both metastatic disease and primary brain tumors can lead to emergent cases of increased intracranial pressure (ICP). These tumors can cause cerebral edema, further increasing the ICP. Intracranial tumors are inherently dangerous due to the fixed volume of the skull of approximately 1400–1700 mL [1]. In a healthy adult, 80% of this volume will be brain parenchyma, 10% will be cerebrospinal fluid (CSF), and 10% will be blood. The Monro-Kellie doctrine states that when one of these components is altered or a new lesion is present, the remaining components are displaced and ICP increases if a volume threshold is reached [2].

In adults, ICP is normally ≤15 mm Hg. Increasing pressure above 20 mm Hg defines intracranial hypertension (ICH). Although the volume of the brain parenchyma is relatively fixed, CSF is able to move into the spinal arachnoid space and cerebral venous blood volume can be decreased through increased drainage or vasoconstriction. If these volume compensations cannot adequately account for the presence of a new lesion, ICP begins to increase [3]. The compensation of decreasing cerebral blood flow (CBF) is limited by its effect of reducing cerebral perfusion pressure (CPP). This decrease can eventually lead to an ischemic state, which can cause poor outcomes for the patient [1].

In addition to the increase of volume due directly to a mass, cancer can increase ICP through different mechanisms that require consideration. "Pseudoprogression" is the term used to describe the increase in ICP after a patient undergoes radiation for a mass lesion and vasogenic edema ensues [4]. This can be differentiated from actual tumor progression through serial imaging as pseudoprogression would not continue to increase in volume over time [4]. Furthermore, blockage of any CSF outflow tract will cause this fluid to build up, and its compensatory displacement cannot occur [3]. Both of these mechanisms mimic the mass effect of a lesion and can therefore lead to similar presentations.

One major consequence of acutely increased ICP beyond the compensation threshold is herniation of the brain parenchyma [5]. The three types of herniations that often occur are subfalcine, uncal, and tonsillar herniations. Subfalcine herniation refers to the compression of the anterior cerebral artery inferior to the falx cerebri, uncal herniation refers to the compression of the midbrain and ipsilateral oculomotor nerve by the uncus, and tonsillar herniation refers to the downward compression of the brainstem through the foramen magnum [3]. Each of these herniations is responsible for separate patient presentations and will be discussed in the following section.

Presentation

The most common presenting symptom of increased ICP in patients with brain tumors is a headache similar to the non-throbbing tension-type. Unlike tension-type headaches, headaches from tumors are often worsened when the patient bends over [6]. The severity of the headaches can vary, with some patients describing it as "the worst headache of my life" that was not relieved by common analgesics [7]. These acute onsets headaches could be due to hemorrhage or obstructive hydrocephalus caused by tumors and can be noted by phenomena called plateau waves. Plateau waves are defined as acute elevations in ICP that often surpass 40 mm Hg for over 5 minutes [8]. These severe headaches may also be accompanied by transient neurologic deficits that can falsely present as orthostatic hypotension since moving to the standing position can decrease level of con-

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sciousness [9]. Furthermore, plateau waves lasting more than 30 minutes can cause irreversible damage due to the lack of CBF [10].

Headaches may be accompanied by other symptoms of mass effect including nausea, vomiting, papilledema, abducens nerve palsy, and transient neurologic deficits [6]. Headaches that occur in a patient with no prior history of headaches or headaches that abruptly change pattern are particularly worrisome, although keep in mind that brain tumors are present in patients with isolated headaches less than 1% of the time [11, 12]. As previously stated, these deficits may be brought on from standing or other triggers that increase ICP such as coughing or sneezing. Impairments of consciousness often present as patient lethargy [13].

The most severe presentations of elevated ICP occur from herniation of the brain. These most often occur after acute increases in ICP when volume compensations are not adequate. A few signals that herniation is likely are the Cushing's reflex, Cheyne-Stokes respiration, or acute impairments in consciousness [14]. Cushing's reflex refers to hypertension, bradycardia, and abnormal breathing patterns as a response to increased ICP [15]. Cheyne-Stokes respiration is characterized by pronounced fluctuations in breathing that cycle between deep breathing and temporary pauses in breathing [16]. Both of these signs, as well as consciousness impairments, require emergency attention [13].

Compression of the anterior cerebral artery from subfalcine herniation can lead to contralateral leg weakness and potentially bladder incontinence [4]. Uncal herniation may cause compression of the oculomotor nerve and the midbrain. A patient with an oculomotor nerve palsy will likely present with a "blown pupil" as the parasympathetic fibers are located on the outer portion of the oculomotor nerve. Further compression can then cause a "down and out" eye accompanied by ptosis, both on the ipsilateral side. Ipsilateral hemiparesis may occur as the midbrain is pressed against the contralateral cerebral peduncle. The posterior communicating arteries may also be compressed, which could lead to a stroke in the occipital lobe [17]. Tonsillar herniations can lead to devastating outcomes when the brainstem is compressed, leading to reticular activating system damage or pontine hemorrhage. Levels of consciousness can be greatly impaired, and patients with pontine hemorrhage may lose all motor function except upward gaze and eyelid movements as in the classic locked-in syndrome [18].

Hospital Course and Management

When a patient's clinical presentation demonstrates the previously listed symptoms, the patient should be managed medically in the hyperacute setting with hyperventilation, steroids, and osmotherapy. Hyperventilation should be the initial treatment, and it is performed through intubation with the patient sedated. The goal is to reduce pCO_2 to 25–30 mm Hg in order to cause vasoconstriction in the cerebral vessels, although this decreases CBF, so the duration of hyperventilation must be balanced with the risk of ischemic damage [19].

Once hyperventilation has been initiated, the patient should be started on steroids and osmotherapy. Dexamethasone is the preferred corticosteroid and is used to reduce ICP by decreasing vasogenic edema. This mechanism is especially effective when ICP elevations are caused by mass lesions. Based on a level 3 recommendation, patients with acute ICP emergencies should receive a bolus of 10–20 mg and be maintained on 8–16 mg per day as seen in Table 14.1 [20]. This should then be tapered over a 2-week period unless the patient remains symptomatic, requiring a lengthened duration of the steroid [21]. One contraindication to note is when CNS lymphoma is also on the differential, as this corticosteroid may lyse the B lymphocytes and confound the biopsy. In this case, osmotherapy should be used without concurrent corticosteroid use until the biopsy is performed [12].

Osmotherapy is used with dexamethasone and can either be in the form of 20–25% mannitol or hypertonic (23.4%) saline [22, 23]. By increasing the osmolarity of the blood, a gradient is formed across the blood-brain barrier to reduce water in the intracranial space [13]. This therapy has the potential to acutely reverse ICP to avoid or alleviate brain herniation. However, caution should be used because a rebound effect has been observed after frequent doses are administered, which could exacerbate the elevated ICP [23]. Furthermore, osmotherapy is only effective until the brain builds up enough "idiogenic osmoles" to reverse the osmolarity gradient and draw fluid back across the blood-brain barrier [24].

Once these immediate treatments are given, imaging is needed to look for the cause of elevated ICP. In emergency situations, a non-contrast CT should be performed in order to rule out hemorrhage. Once this is ruled out or the patient is stable, magnetic resonance imaging (MRI) should be the next step to evaluate the cause of increased ICP. T2 fluid-attenuated inversion recover (FLAIR) images can demonstrate the vasogenic edema and mass effect present by suppressing the CSF signal, while T1-contrast-enhanced images represent the gold standard by providing clear and high-resolution images of the mass lesion present [12, 25].

If the cause of elevated ICP is found to be due to a mass lesion and immediate ICP-lowering treatments do not reverse the acute presentation, neurosurgical intervention becomes warranted [25]. This can involve an entire resection or a partial debulking to decrease mass volume, and the extent of resection will largely be due to the intracranial location of the tumor [13]. Furthermore, CSF can be reduced through the placement of a ventriculostomy, especially when the mass lesion is causing obstructive hydrocephalus [25].

Table 14.1 Common pharmacological agents used in the NCCU

Pharmacological agents			
Elevated intracranial pressure			
Drug	**Dosing**	**Route**	**Key side effects**
20% mannitol solution	Bolus of 1 g/kg, repeat with 0.25–0.5 g/kg as needed every 6–8 hours	Intravenous	"Rebound" increase in ICP Hypernatremia Pulmonary edema
Hypertonic (23.4%) saline	Bolus of 30 mL	Intravenous	Central pontine myelinolysis (rare) Acute heart failure Pulmonary edema
Dexamethasone (also used in intratumoral hemorrhage)	Loading dose of 10–20 mg, maintained on 8–16 mg per day, then tapered over a 2-week period	Oral or intravenous	Insomnia Essential tremor GI complications Steroid myopathy Opportunistic infections
Pituitary tumor apoplexy			
Drug	**Dosing**	**Route**	**Key side effects**
Hydrocortisone	100 mg IV or IM followed by 50–100 mg IM every 6 hours or 100–200 mg IV with 2–4 mg/hour through IV infusion	Intravenous and intramuscular	Nausea Headache Dizziness Opportunistic infections Hyperglycemia (rare)
Status epilepticus			
Drug	**Dosing**	**Route**	**Key side effects**
Lorazepam	4 mg fixed dose and repeat if no termination of seizure activity	Intravenous (use intramuscular midazolam if no venous access)	Drowsiness Cognitive impairment Respiratory depression Hypotension
Fosphenytoin	15–20 mg/kg phenytoin equivalents (PE) infused at 100 mg PE/minute	Intravenous or intramuscular if no venous access	Hypotension Arrhythmias CNS adverse effects Local dermatological reactions
Valproic acid	20–40 mg/kg infused at 5 mg/kg/min	Intravenous	Nausea/vomiting Drowsiness Thrombocytopenia Pancreatitis Hepatotoxicity
Lacosamide	200–400 mg IV bolus	Intravenous	Visual changes Nausea/vomiting Ataxia
Levetiracetam	40–60 mg/kg	Intravenous	Behavioral changes Somnolence Headache Stevens-Johnsons syndrome (rare)
Phenobarbital	20 mg/kg infused at 30–50 mg/min	Intravenous	Cardiorespiratory depression Visual changes Effects of CYP450 induction Paradoxical hyperactivity (children)

Pituitary Tumor Apoplexy

Overview

Pituitary tumor apoplexy (PTA) is the phenomenon that occurs when the blood supply to the pituitary is acutely interrupted. This blood supply includes the superior and inferior hypophyseal arteries for the anterior and posterior pituitary glands, respectively [26]. If a pituitary adenoma grows rapidly enough, it may physically disrupt the blood supply, and this can lead to either hemorrhage or necrosis of the tissue. Furthermore, pregnancy, peri- or postsurgical hypotension, vasospasm, and head trauma can lead to apoplexy [12, 27].

The incidence of these episodes is relatively rare and incidence ranges from 0.6% to 7% of pituitary adenomas [27–29]. The most common pituitary tumors associated with PTA are nonfunctioning macroadenomas, prolactinomas, and growth hormone-secreting macroadenomas [30]. These tumors may extend into the suprasellar region and impinge

upon the optic chiasm as well as laterally to affect the structures of the cavernous sinus [27]. These cavernous structures include cranial nerves (III, IV, V_1, V_2, VI) and the internal carotid artery [31].

Presentation

The most common presentation of PTA is acute headache. It is speculated that these severe headaches can be due to several factors, including trigeminal nerve involvement, dural stretching, or meningeal irritation. The headaches are often retro-orbital and deep and are usually unique to any previous headaches [27]. These headaches may also be severe enough to be accompanied by nausea and vomiting, or even mental disturbances in the most severe cases [12].

As mentioned previously, cranial nerves can be affected in PTA, which can present with visual disturbances. The oculomotor nerve (cranial nerve III) is the most common cranial nerve deficit and is present in approximately 50% of patients. This can be observed as ptosis and "blown" pupils [32]. Bitemporal hemianopsia may also arise in patients if the pituitary tumors grow superiorly enough to make contact with the optic chiasm [31]. However, the rates of visual alterations may be lower in modern practice than what the literature states since many studies include patients that were treated before MRIs were widely available, with more delayed diagnoses and larger tumors [33, 34].

Since PTA episodes are most often reported in cases of macroadenomas, endocrinopathies are often present in patients. Approximately 80% of patients with PTA have at least one sign of anterior pituitary hormone dysfunction; the most common is adrenocorticotropic hormone (ACTH) dysfunction, present in nearly 70% of PTA cases. This regulates the cortisol production axis, and deficiency can therefore present as hypotension or hyponatremia [32]. Less common hormone deficiencies can include thyrotropin and gonadotropin. Conversely, prolactin may be increased from either prolactinomas or stalk compression due to interruption of its dopamine-signaled constitutive inhibition [35]. While anterior pituitary dysfunction is relatively common in PTA, the posterior pituitary gland is rarely affected with only 5% of patients presenting with symptoms of diabetes insipidus [34].

Hospital Course and Management

Traditionally, CT scans were performed when patients presented with signs of PTA to look for evidence of sellar hemorrhage. However, a recent study found that hemorrhage was only identified in 42% of patients with PTA [34]. MRI was able to detect hemorrhage in 89% of patients while also allowing for the evaluation of the health of the surrounding tissue [34]. Furthermore, MRI allows subacute and chronic apoplexy to be observed to a much greater extent than CT [27]. Therefore, an urgent brain MRI is indicated when signs of PTA are present unless an MRI is contraindicated in a patient. In these cases, high-quality CT scans with and without contrast are warranted [27]. MR angiograms may be utilized when vasospasm or aneurysms are assumed to be contributing precipitants of PTA [27]. Furthermore, CT scans may be performed in order to exclude subarachnoid hemorrhage (SAH) and meningitis as these conditions can present with similar signs [33].

The greatest cause of morbidity and mortality after PTA episodes is acute adrenal insufficiency due to pituitary damage [33]. Therefore, 100 mg intravenous (IV) or intramuscular (IM) hydrocortisone should be given to combat this effect along with IV fluids to maintain electrolyte balance. This should be followed by hydrocortisone 50–100 mg IM every 6 hours or 100–200 mg IV bolus with 2–4 mg/hour continuous IV infusion [33]. Hydrocortisone should be continued orally after discharge until it is clear that adrenal function is stabilized [27]. An urgent blood endocrine panel should also be obtained in order to assess the need to supplement other hormone deficiencies [33, 34].

While early decompression has been the traditional treatment for PTA, there is growing evidence that conservative treatment involving sole medical management can lead to similar outcomes in select patients [33, 36, 37]. Early decompression is most often indicated when patients present with severe ophthalmological alterations. This is currently the preferred treatment when visual deficits are progressive and not improved with initial medical therapy [34]. In patients who did not present with visual symptoms or their symptoms decreased with medical therapy, conservative approaches of medical management can lead to complete resolution of symptoms. However, as many studies have pointed out, direct comparisons between these two treatments cannot be made since treatment decisions were based on the presentation of individual cases. Therefore, this selection bias should not be overlooked, and sole medical management should only be opted for when progressive neuro-ophthalmological symptoms are not present and symptoms are decreasing in severity [33, 34, 36, 37]. This treatment paradigm is in agreement with the Society for Endocrinology UK guidelines for the management of pituitary apoplexy [38].

Acute Tumor Hemorrhage

Overview

The incidence of intratumoral hemorrhage has been reported to range between 1% and 10% in previous studies, and this includes both primary and metastatic brain tumors [39, 40].

The vast majority of these hemorrhages occur in patients with previously discovered brain tumors, while only 4% of patients had intracerebral hemorrhages caused by unsuspected brain tumors [40].

The two primary brain tumors most associated with intracranial hemorrhage are glioblastoma multiforme (GBM) and oligodendrogliomas [41]. GBMs are the most common primary brain tumor in adults and are known for their very destructive nature. Although oligodendrogliomas are less invasive, these tumors contain retiform capillaries that are known to hemorrhage [42]. Of metastatic brain tumors, melanoma, lung, renal, choriocarcinomas, and papillary thyroid carcinomas have had the highest rates of intracerebral hemorrhages [23, 43].

Presentation

The presentation of intratumoral hemorrhage is very similar to the presentation common in intracranial hemorrhage patients without brain tumors [44]. The two most common presenting signs include hemiparesis and headaches, both occurring in nearly half of patients [43]. These are followed by encephalopathy, nausea and vomiting, seizure, and coma in the most severe cases [43]. However, one difference between brain tumor patients and the general population is that intracranial hemorrhages in tumor patients are primarily intraparenchymal instead of subdural or subarachnoid [26].

Hospital Course and Management

When hemorrhage is suspected in a patient, an urgent MRI is indicated unless one is not immediately available. In that case, an immediate non-contrast CT scan is indicated. This is supported by Class I, Level A evidence from the American Stroke Association [45]. Brain tumor hemorrhages are often suspected when an intracerebral hemorrhage (ICH) is present in an atypical location, there are multiple hemorrhages, or if an enhancing mass is seen near the bleeding site [23]. A baseline severity score, such as the original ICH score, must also be given when initially evaluating a patient with a suspected ICH (*Class I; Level of Evidence B*) [45]. This grading scale includes variables such as age, ICH volume, location of hemorrhage, presence of intraventricular hemorrhage, and score on the Glasgow Coma Scale [45]. If suspicion for the presence of a tumor remains, "a CTA, CT venography, contrast-enhanced CT, contrast-enhanced MRI, magnetic resonance angiography and magnetic resonance venography, and catheter angiography can be useful to evaluate for underlying structural lesions including vascular malformations and tumors" (*Class IIa; Level of Evidence B*) [45]. An MRI also allows one to search for alternative causes of hemorrhage such as ischemic stroke with hemorrhagic conversion, venous sinus thrombosis, or amyloid angiopathy [44].

Once an intratumoral hemorrhage is confirmed, corticosteroids can be given to alleviate any vasogenic edema present [23]. However, corticosteroids should not be given solely to reduce ICP after intratumoral hemorrhage (*Class III; Level of Evidence B)* [45]. Consideration should then be given to whether the tumor can be surgically removed [41, 43]. The International Surgical Trial in Intracerebral Hemorrhage (STICH) found that lobar hemorrhages trended toward better surgical outcomes, although this finding was nonsignificant [46]. However, if there are multiple tumors or a tumor is unresectable, whole-brain radiation may be the next option [44]. If an intratumoral hemorrhage bleeds excessively and a clot forms, the clot may be subject to surgical evacuation. However, this should only be performed if the clot is causing progressive symptoms or is superficially located because the STICH and STICH II trials found that early evacuation did not improve patient outcomes [46, 47].

Although short-term outcomes of patients with intratumoral hemorrhage are similar to non-cancer patients with intracerebral hemorrhage, long-term outcomes are often worse due to the underlying malignancy. Navi et al. observed a 78% mortality at 1 year, likely because intratumoral hemorrhages often occur late in the course of malignancy [43]. However, patients with intracerebral hemorrhage due to cancer-related coagulopathies had worse outcomes than patients with intratumoral hemorrhages, as the former patients often had larger hemorrhages and involvement of multiple intracranial compartments [43, 44].

Status Epilepticus in Tumor Patients

Overview

Patients with both primary and metastatic brain tumors have a relatively high rate of seizures, likely due to the mass effect of the surrounding cortex [12]. Seizures most often begin as partial seizures due to the location of the tumor, and only a portion of these will become generalized seizures. Dysembryoplastic neuroepithelial tumors (DNETs) and gangliomas are associated with the highest rates of seizures (80–100%), with these tumors most often arising in children [48]. These tumors likely carry a high risk of seizures due to their frequent localization in the temporal lobe, which is an epileptogenic area along with the insula and cortex [49]. Interestingly, 60–85% of lower-grade gliomas have been found to lead to seizures compared to only 30–60% of glioblastomas [50]. This may be due to the fact that the survival period in patients with low-grade gliomas is much longer [51]. Seizures from brain metastases occur at a lower frequency than each of the above-mentioned primary brain

tumors, carrying only a 20–35% seizure incidence [51, 52]. Metastases from melanomas have the highest incidence of seizures (67%), likely due to its hemorrhagic nature [51].

Although seizures in patients with brain tumors are most often self-limited and short-lasting, those that do transform into status epilepticus (SE) become emergencies [12]. SE is defined as a continuous seizure lasting greater than 5 minutes, or multiple seizures occurring consecutively without return to baseline [53]. SE is associated with a 20% 30-day mortality, and between 15% and 22% of brain tumor patients with epilepsy progress to SE [54, 55].

In addition to seizures being caused by mass effect, several cancer therapies have been known to cause seizures through a phenomenon known as posterior reversible encephalopathy syndrome (PRES). These therapies include bevacizumab, sorafenib, cyclophosphamide, l-asparaginase, cisplatin, and gemcitabine [56]. Therefore, PRES must be ruled out when brain tumors are being investigated as the cause of seizures.

Presentation

Patients may present with different subtypes of seizures, including simple partial seizures, complex partial seizures, and focal seizures with secondary generalization [23, 51]. Patients may also present with focal weakness in an extremity after they experience an epileptic episode, known as postictal paralysis (Todd's paralysis) [57]. Some patients progress to SE in the absence of convulsions, also called nonconvulsive status epilepticus (NCSE). Patients with NCSE may present with abnormal eye movements, nonspecific personality changes, myoclonic jerks, or altered mental status [23, 57]. Furthermore, SE may arise at different periods throughout the neoplastic process. Cavaliere et al. found that 29% of SE in brain tumor patients arises at tumor presentation, 23% during tumor progression, and 23% in patients with stable brain tumors [54].

Hospital Course and Management

When a brain tumor patient presents with SE, medical therapy should be immediately given and the airway must be secured. Patients should initially be given lorazepam 4 mg IV (a benzodiazepine) to terminate the SE unless IV access is not possible, and then IM midazolam is substituted [58]. If SE is not terminated, another dose of 4 mg lorazepam (or IM midazolam) should be given [3, 59]. Electrocardiogram and vital sign monitoring should also be initiated upon diagnosis of SE, and blood samples should be tested for glucose, electrolytes, antiepileptic drug (AED) levels, and toxic agents. If SE is not terminated after benzodiazepine administration, phenytoin (a second-line agent) should be given at 15–20 mg/kg. If phenytoin does not terminate SE, third-line agents include phenobarbital, valproic acid, lacosamide, and levetiracetam [3, 60]. These treatments are based on the Guidelines for Status Epilepticus by the Neurocritical Care Society and should be followed regardless of whether the cause of SE is a brain tumor [60]. Although epileptic attacks are more frequent with low-grade gliomas, high-grade gliomas are associated with AED refractoriness with a 60% failure rate of terminating after first-line benzodiazepines [55]. However, when seizures progress to SE in brain tumor patients, this tumor-associated form of SE is paradoxically easier to treat with first-line benzodiazepines than SE in the general population [55].

When treating SE in cancer patients, it is important to be aware of drug-drug interactions between SE therapies and chemotherapeutic agents. For example, several older-generation anticonvulsants are hepatic cytochrome P450 inducers (phenytoin, carbamazepine, phenobarbital) that can reduce levels of drugs that use the same metabolic pathway, such as dexamethasone [12]. For this reason, newer antiepileptic drugs (levetiracetam, lamotrigine, lacosamide) are often used as they carry less risk of drug interactions [61, 62]. The opposite can also occur where hepatic cytochrome P450 inhibitors (valproic acid) can increase the levels of chemotherapeutic agents (cisplatin, etoposide), which can lead to bone marrow toxicity [63]. Furthermore, although these AEDs are helpful in the situations previously discussed, there is a lack of evidence in their prophylactic use to decrease risk of new seizures [64].

Once SE is terminated, imaging should be performed to search for the cause of the seizures. A CT scan may be performed in an emergent situation to look for obvious hemorrhage or mass effect, but an MRI is optimal for a more detailed evaluation of the cause of SE. MRI can more accurately display the number and size of masses as well as whether progression of a known tumor has caused SE [23]. Seizures in brain tumor patients may not always be convulsive, and this makes electroencephalography (EEG) a useful tool in order to look for seizure activity when NCSE is suspected [65]. An EEG also allows one to observe if a patient returns to baseline after a seizure in order to rule in/out SE [7, 23, 49].

Lastly, once SE is terminated and a brain mass is identified as the cause, surgical resection may be performed if feasible. Chang et al. analyzed 332 patients who underwent surgical resection for low-grade gliomas and found that 67% were seizure-free after surgery and that gross total resections achieved the best seizure outcomes [66]. Therefore, surgical resection should remain an option in treating these patients, and this should be discussed with the multidisciplinary neuro-oncological care team.

Patient Flow Upon Admission

Upon admission to the NCCU, the vital signs of brain tumor patients need to be stabilized immediately. This should be followed by a physical examination and comprehensive laboratory studies. The patient should undergo appropriate monitoring including electrocardiogram, blood pressure, oxygen saturation, hematologic laboratories, liver and kidney function tests, blood and urine osmolality, and body temperature [67]. This should be followed by a detailed neurological examination assessing mental status, cranial nerves, sensorimotor function, reflexes, and coordination if possible.

As discussed in the previous sections, ICP, CPP, and CBF should be continually monitored, and the team should consider whether invasive ICP monitoring is necessary as this could also allow for CSF drainage if required [68]. EEG monitoring is also supported for patients in the NCCU based on Class II and III evidence, Type C recommendation from the American Society of Neurophysiological Monitoring [69]. In addition to these monitoring techniques, selection of MRI or CT scans should depend on the urgency of symptoms and the type of pathology expected.

Pain severity and degree of sedation should also be evaluated in the NCCU. Pain severity should be assessed according to the numerical ranking score from 0 to 10, with 0 representing no pain and 10 representing excruciating pain. However, the patient may be incapacitated and unable to give a response. Therefore, patient behaviors such as facial expressions must be evaluated and combined with the physiological functions already being monitored (heart rate, blood pressure) [67]. These parameters can also be used when assessing the degree of sedation and can be applied to several scoring systems including the Ramsay Score, Riker Sedation-Agitation Score, and the Bispectral Index Scale. These scores, combined with respiratory and cardiovascular functioning, can inform the type and quantity of analgesics required for the patient while a treatment plan is being formed and delivered [70].

Summary

This chapter outlines the most common presentations, hospital courses, and management routes of patients with elevated ICP, pituitary tumor apoplexy, acute tumor hemorrhage, and status epilepticus. An understanding of these emergencies, as well as others not discussed in this chapter, will allow the NCCU team to give the best care to patients by delivering the most efficient evaluation, diagnosis, and treatment. While several NCCU guidelines have been established to allow for better, more standardized patient care, more are needed to ensure optimal care is being given in each NCCU.

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