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Review Article

Critical assessment and intervention in young patients with neurological emergencies

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ABSTRACT

Neurological emergencies in young patients require prompt recognition and intervention to avert irreversible damage or death. These emergencies encompass acute conditions such as strokes, traumatic brain injuries, status epilepticus, and infections like meningitis. Recent advancements in neuroimaging, including portable MRI, have significantly improved diagnostic capabilities. Management protocols are evolving, with notable progress in treatments such as intravenous thrombolysis and mechanical thrombectomy for acute ischemic stroke. For status epilepticus, the timely administration of antiepileptic drugs is vital. Furthermore, addressing conditions like myasthenic crisis and acute intracranial hypertension involves specific interventions such as plasmapheresis, IV immunoglobulin, and targeted ventilation strategies. Delirium management in hospitalized patients highlights the importance of a multidisciplinary approach, incorporating both pharmacological and non-pharmacological treatments. Education and continuous professional development are essential to keep healthcare providers abreast of the latest diagnostic and therapeutic advancements. This review synthesizes current practices in the assessment and intervention of neurological emergencies, emphasizing the importance of timely, evidence-based management to improve patient outcomes.

Keywords: Neurological emergencies, Young patients, Neuroimaging, Professional education

INTRODUCTION

Neurological emergencies represent some of the most time-sensitive and potentially life-threatening circumstances encountered in medical practice. They encompass a wide range of acute disorders affecting the central and peripheral nervous systems, requiring immediate recognition and intervention to prevent irreversible damage/ death. Rapid evolution of neurodiagnostic techniques and therapeutic strategies has significantly enhanced the ability to manage these critical conditions effectively.[1]

Neurological emergencies often present with severe symptoms such as sudden loss of consciousness, severe headaches, acute neurological deficits, and seizures. These presentations can result from various underlying conditions including strokes, traumatic brain injuries, status epilepticus, and infections like meningitis or encephalitis. The diverse aetiologies and presentations necessitate an elevated level of clinical acumen and the use of advanced diagnostic tools to ensure accurate and prompt diagnosis.[2]

One of the critical components in the management of neurological emergencies is neuroimaging. Modalities like computed tomography (CT) and magnetic resonance imaging (MRI) are indispensable in the initial assessment of these patients. Recent advancements have introduced portable MRI (pMRI) machines, which allow for

immediate bedside imaging, thereby expediting diagnosis and management in emergency and intensive care settings. These portable devices are particularly beneficial in scenarios where traditional MRI access is limited or transport is risky for the patient.[3]

Management protocols for neurological emergencies are continually evolving, integrating the latest evidence-based practices. For instance, the treatment of acute ischemic stroke has seen significant advancements with the advent of intravenous thrombolysis and mechanical thrombectomy, both of which are crucial in the early hours post-symptom onset. The development and refinement of clinical guidelines ensures that healthcare providers can deliver the most effective interventions in a timely manner.[4,5]

Another aspect of managing neurological emergencies involves advanced therapeutic interventions. For conditions like status epilepticus, which is characterized by prolonged or repetitive seizures, timely administration of antiepileptic drugs is critical. Benzodiazepines are often the first line of treatment, followed by other medications like fosphenytoin or levetiracetam if seizures persist. The goal is to terminate the seizures quickly to prevent long-term neurological damage.[2] The role of education and continuous professional development cannot be overstated in neurological emergencies. Training programs and continuing medical education courses, such as those offered by institutions like the world federation of neurology, equip healthcare professionals with the latest knowledge and skills required to manage these complex cases. These programs emphasize practical approaches to clinical reasoning, diagnostic strategies, and therapeutic interventions, ensuring that practitioners are well-prepared to handle the high-stakes scenarios associated with neurological emergencies.[5]

LITERATURE SEARCH

This study is based on a comprehensive literature search conducted on June 3, 2024, in the Medline and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed critical assessment and intervention in young patient with neurological emergencies. There were no restrictions on date, language, participant age, or type of publication.

Managing neurological emergencies is a crucial part of an emergency physician's skill set, regardless of their practice's focus.[6] Neurological issues account for approximately 5% of annual emergency department visits.[7]

NEUROLOGICAL EMERGENCIES

Acute ischemic stroke

It is a medical emergency characterized by the sudden onset of neurological deficits due to the abrupt loss of blood circulation to a brain region (Figure 1). This condition is primarily caused by thrombotic or embolic blockage of a cerebral artery, leading to symptoms such as motor deficits (hemiparesis, monoparesis, quadriparesis), sensory deficits, monocular or binocular vision loss, visual field deficits, diplopia, dysarthria, facial droop, ataxia, aphasia, and a sudden decrease in consciousness.[8]

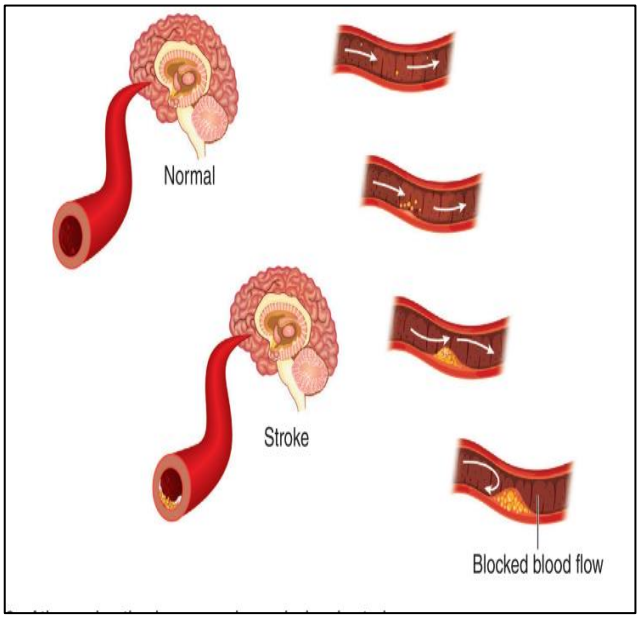

Figure 1: Atherosclerotic plaque causing an ischemic stroke.[23]

Assessment

The initial assessment of a patient with suspected acute ischemic stroke involves several critical steps. Continuous pulse oximetry and supplemental oxygen are administered to maintain SpO_2>94%. Cardiac and blood pressure monitoring is essential. A brief but thorough neurological examination is performed to identify deficits and localize the lesion, followed by an evaluation of stroke severity using the national institutes of health stroke scale (NIHSS). Establishing the time of symptom onset or when the patient was last known to be normal is vital for determining eligibility for treatments like thrombolysis.[8]

Intervention

Treatment of acute ischemic stroke begins with stabilizing the patient and addressing underlying conditions. Hypotension and hypovolemia are corrected to ensure adequate systemic perfusion. Blood pressure is

carefully managed in hypertensive patients eligible for IV alteplase or mechanical thrombectomy, with specific medications used to lower BP to <185/110 mmHg before treatment. Rapid administration of IV alteplase, ideally within 60 minutes of hospital arrival, is critical for dissolving the thrombus and restoring blood flow. Mechanical thrombectomy is indicated for large vessel occlusions within a specific period. Additional interventions include managing blood glucose levels, initiating antiplatelet therapy, and addressing complications such as brain swelling. Secondary prevention measures, including screening for diabetes, hyper-lipidemia, and hypertension, as well as promoting smoking cessation, are crucial for reducing the risk of future strokes.[9]

Acute haemorrhagic stroke

It primarily affects patients with vascular comorbidities such as prior ischemic or hemorrhagic stroke, hypertension, diabetes mellitus, and those who smoke (Figure 2). Symptoms and signs include a sudden focal neurological deficit that worsens over minutes to hours, headache, vomiting, decreased level of consciousness, elevated systolic blood pressure >220 mmHg, and coma. Procedures like carotid endarterectomy or carotid stenting can increase hyper perfusion, subsequently raising the risk for hemorrhagic stroke.[8]

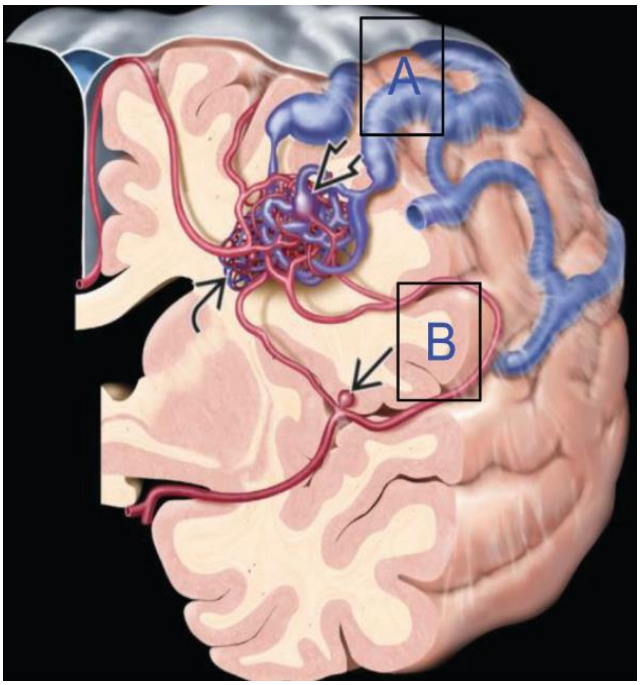

Figure 2 (A and B): Vascular malformations causing hemorrhagic strokes. Arteriovenous malformation and intracranial aneurysm.[23]

Assessment

Initial assessment of a patient with suspected acute hemorrhagic stroke involves continuous pulse oximetry and supplemental oxygen to maintain SpO_2>94%. Cardiac and blood pressure monitoring with a cycled automated BP cuff is essential. Establishing the time of symptom onset or when the patient was last known to be normal is crucial. A brief and accurate neurological examination helps identify neurological deficits and localize the lesion. Assessing the severity of the stroke using the intracerebral haemorrhage (ICH) score or NIHSS is necessary, as the initial ICH score and NIHSS predict functional outcomes. Diagnostic work-up includes labs (CBC, BMP, coagulation studies, troponin, toxicology screen, urinalysis, and urine pregnancy test in women of childbearing age), EKG, and imaging studies such as head CT scan or MRI to distinguish ischemic stroke from intracranial haemorrhage.[8]

Intervention

Treatment of acute hemorrhagic stroke involves several urgent steps. Discontinue all anticoagulants and antiplatelet agents and administer reversal agents as needed. For hypertensive patients, blood pressure should be reduced acutely to a goal of 140 mmHg using medications like nicardipine drip. Monitoring glucose levels to avoid both hyperglycemia and hypoglycemia is essential. Fever should be treated with antipyretics such as rectal acetaminophen. High-risk patients for seizures, especially those with cortical involvement, should be treated with antiseizure medications, and an EEG is recommended for patients with depressed mental status. Thromboprophylaxis using intermittent pneumatic compression and elastic stockings is recommended, with low-dose anticoagulation being considered post-bleeding cessation in consultation with a neurosurgeon. Patients with a Glasgow coma scale (GCS) of 3-8 should be intubated, while those with a GCS >8 should be assessed by a neurosurgeon. Intracranial pressure (ICP) monitoring is indicated for patients with significant neurological deterioration, and elevated ICP should be treated as per guidelines. Decompressive craniectomy with hematoma evacuation may be necessary for patients with cerebellar hemorrhage or significant midline shift and refractory elevated ICP. Finally, a formal screening protocol for dysphagia should be implemented before initiating oral intake.[10]

Seizure

It is a life-threatening neurological emergency, defined by the Epilepsy Foundation working group as seizure activity that persists for more than 10 minutes.[11] Presentations can include new onset seizure disorders or exacerbations of preexisting ones. While easily recognized in patients with general convulsions, nonconvulsive presentations require a higher index of suspicion. The most common trigger in patients with known seizure disorders is a change in medication, but other precipitating factors include infection, brain tumors, metabolic disorders, withdrawal, sleep deprivation, illicit drugs, or toxic ingestions.[8]

Assessment

Initial assessment for seizure includes continuous pulse oximetry and supplemental oxygen to maintain SpO_2>92% or PaO_2 >60-65 mmHg, with a transition to high flow oxygen if needed. Continuous cardiac monitoring and seizure/fall precautions are necessary, and large-bore IV access should be established if not already in place. Diagnostic work-up involves ABG to assess hypoxia and metabolic acidosis, along with labs (CBC with differential, procalcitonin, BMP, magnesium, liver panel, toxicologic screening, serum pregnancy test in women of childbearing age, and antiepileptic medication levels).[8] Additional tests include UA, urine, blood, and sputum cultures if infection is suspected, EKG to identify potential cardiac arrhythmias, and EEG to monitor seizure activity.[12] A noncontrast head CT scan may be ordered to evaluate for structural lesions, and lumbar puncture is reserved for patients with persistent fever, altered mental status, headache, and for immunocompromised patients.

Intervention

Seizure treatment involves immediate management of glucose levels, administering a 50 mL bolus of 50% dextrose IV if hypoglycemic.[8] Thiamine 100 mg IV may also be considered. The American epilepsy society's 2016 guidelines outline three phases of treatment: first-line agents include lorazepam, diazepam, and midazolam.[13,14] If seizures persist for ≥5 minutes despite aggressive benzodiazepine treatment, second-line agents such as fosphenytoin, valproic acid, levetiracetam, and phenobarbital are administered.

For patients who continue to seize ≥20 minutes, third-line agents should be given after intubation, including propofol infusion, midazolam infusion, and pentobarbital. Continuous monitoring and adjustments based on patient response are critical throughout the treatment process.[8]

Acute intracranial hypertension

It is a medical emergency characterized by a sudden increase in ICP, normally ranging from 5 to 15 mmHg.[8] This condition can arise in patients with acute intraventricular, intraparenchymal, subarachnoid, epidural, or subdural hematomas, severe hydrocephalus, severe brain trauma, brain tumors, and severe ischemic strokes. Symptoms include headache, nausea, vomiting, somnolence, blurry or double vision, focal neurological deficits, and potentially herniation syndrome with pupillary changes and cranial nerve palsies.

Patients may also exhibit Cushing's triad-bradycardia, irregular respirations, and widened pulse pressure, and typically have a low GCS score. Papilledema is a reliable sign of intracranial hypertension in the comatose patients.[15]

Assessment

Initial measures for assessing a patient with suspected acute intracranial hypertension include continuous pulse oximetry and supplemental oxygen to maintain SpO_2≥94%. Continuous cardiac and blood pressure monitoring is also crucial. Elevating the head of the bed to 15-30 degrees and ensuring the head is in a neutral and midline position can significantly reduce ICP by keeping the jugular veins open. Early consultation with a neurosurgeon is recommended to plan for potential interventions. The diagnostic work-up involves monitoring vital signs and neurological status closely, along with appropriate imaging and laboratory tests to identify the underlying cause and extent of the condition.[8]

Intervention

Treatment of acute intracranial hypertension involves several urgent interventions. Pain should be managed with opioid analgesics like IV morphine, and agitation controlled with benzodiazepines like IV lorazepam, as both can increase ICP. Fever should be treated with antipyretics such as rectal acetaminophen and cooling blankets. Hypertension is managed with medications like esmolol or labetalol, which reduce systemic blood pressure without affecting ICP.[8] In patients with brain tumors, steroids like dexamethasone may be used, while antiepileptics are administered for seizure control. Neurosurgical consultation for ventricular catheter placement is essential for monitoring and draining cerebrospinal fluid (CSF) to manage ICP.[16] Hyperventilation can temporarily reduce ICP by decreasing pCO_2 and inducing cerebral arteriolar constriction. Mannitol and hypertonic saline are used to reduce ICP, and their administration requires careful monitoring of serum osmolarity and fluid-electrolyte balance. In cases of intractable symptoms, neuromuscular blockade and intubation may be necessary, and surgical interventions, such as decompressive craniectomy, might be considered for patient's refractory to medical management.[8]

Delirium

It is an acute cognitive impairment characterized by a range of neuropsychiatric symptoms, often leading to long-term cognitive dysfunction. It is particularly prevalent in hospitalized elderly patients, those with dementia, mechanically ventilated patients, and individuals with a history of alcohol abuse. Other risk factors include living alone, smoking more than 10 cigarettes per day, severe acute illnesses such as sepsis, and the use of certain medications like antihistamines, anticholinergics, corticosteroids, metoclopramide, opiates, and benzodiazepines, as well as malnutrition.[17] Delirium can be hyperactive (agitated and restless), hypoactive (lethargic and apathetic), or mixed type, with hypoactive delirium being more frequent in ICU patients and associated with higher mortality rates. Symptoms can

include disorientation, reduced attentiveness and awareness, memory impairment, dysarthria, hallucinations, and delusions, with fluctuations in severity.[18]

Assessment

Initial measures for assessing a patient with suspected delirium include evaluating the level of consciousness using the Richmond agitation sedation scale (RASS) and screening for delirium with tools like the confusion assessment method for the intensive care unit (CAM-ICU) or the intensive care delirium screening checklist (ICDSC).[17] Discontinuing drugs associated with delirium is crucial. Diagnostic work-up involves labs (CBC with differential, procalcitonin, BMP, liver function tests, thyroid function tests, urinalysis, thiamine level, B12 level, and urine drug screen). Chest X-rays assess for pneumonia, pleural effusion, or pulmonary vascular congestion, while CT or MRI scans of the head evaluate for ischemic or hemorrhagic strokes or structural lesions. EEGs are ordered for suspected subtle seizures or nonconvulsive states, and lumbar punctures are reserved for patients with suspected CNS infections.[8]

Intervention

Treatment for delirium includes the use of antipsychotics at the lowest effective dosages, especially in elderly patients, with the goal of tapering and discontinuing them once the precipitating condition is managed. Haloperidol lactate can be administered IV or IM for agitation, with careful monitoring for side effects like QT prolongation and extrapyramidal symptoms.[8] Atypical antipsychotics, such as quetiapine, olanzapine, and risperidone, have fewer extrapyramidal side effects.[19] Benzodiazepines are used specifically for alcohol withdrawal delirium, alongside thiamine administration.[20] Continuous observation by one-to-one sitters and the use of soft mechanical restraints may be necessary to prevent self-harm. Nonpharmacological measures include correcting dehydration, preventing sleep deprivation, providing systematic orientation, early mobilization, therapeutic activities, music therapy, and engaging family members. Identifying and treating underlying causes of delirium, such as infection, hypoxia, hypoglycemia, urinary retention, constipation, or fecal impaction, is essential for effective management.[8]

Myasthenia gravis

It is an autoimmune neuromuscular disorder characterized by the weakness of skeletal muscles due to an antibody-mediated attack on acetylcholine receptors at the neuromuscular junction. Myasthenic crisis, a severe exacerbation of myasthenia gravis, can be triggered by infections, medication noncompliance, certain medications (e.g., antibiotics like fluoroquinolones or macrolides, antipsychotics, β-blockers, calcium channel blockers, muscle relaxants, and contrast media), thyroid disorders, surgical procedures, and emotional stress. Patients experience painless muscle weakness that worsens with use and improves with rest, often involving upper airway and respiratory muscles. Symptoms may include ptosis, diplopia, slurred or nasal speech, dysarthria, difficulty chewing and swallowing, dyspnea, and difficulty sitting up or walking.[8]

Assessment

Initial measures for assessing a patient in myasthenic crisis include continuous pulse oximetry, supplemental oxygen to maintain SpO_2 >92% or PaO_2 >60-65 mmHg, and continuous cardiac and capnography monitoring. forced vital capacity (FVC) should be measured at the bedside using a spirometer, with an FVC of 30 mL/kg indicating myasthenic crisis with airway compromise, necessitating ICU transfer.[21] Early consultation with a neurologist is crucial. Diagnostic work-up includes ABG to assess the severity of hypoxia or hypercapnia, labs (CBC for leukocytosis, BMP for electrolyte abnormalities or impaired renal function, and magnesium levels), and a chest X-ray to check for aspiration, lobar collapse, and pneumonia.

Intervention

Management of myasthenic crisis involves several urgent interventions. Cooling measures and antipyretics should be used to lower temperature and improve muscle strength. Positioning the jaw and tongue and suctioning pulmonary secretions are important initial steps. For patients with mild-to-moderate dyspnea who can clear secretions and protect their airway, BiPAP is recommended. Bag-valve mask ventilation should be used while preparing to intubate patients with worsening respiratory status, FVC<15 mL/kg, or worsening hypoxia.[22] Intubation with nondepolarizing agents like rocuronium or vecuronium is preferred due to resistance to succinylcholine. Initial ventilator settings should include assist control mode, FiO_2 100% titrated to maintain SpO_2 90-94%, PEEP 5 cm H_2O, tidal volume 8-10 mL/kg of predicted body weight, RR 12-16 breaths per minute, I/E ratio of 1:3, and Pplat ≤30 cm H_2O. Once the airway is secured, investigate the causes of the crisis. Plasmapheresis or immunotherapy with IV gamma globulin is recommended to remove circulating antibodies and improve symptoms.[8]

Future recommendations

Future recommendations include developing standardized protocols for rapid assessment and treatment, increasing the use of portable MRI in emergency settings, and promoting continuous professional development for healthcare providers. Emphasis should be placed on training in the latest diagnostic and therapeutic advancements, such as intravenous thrombolysis and mechanical thrombectomy. Further research on the efficacy and safety of these emerging treatments is

necessary. Implementing multidisciplinary approaches to conditions like delirium, combining pharmacological and non-pharmacological interventions, is crucial. Additionally, innovative educational tools and CME courses should be developed to keep healthcare professionals updated on best practices and new findings in the field.

CONCLUSION

Timely recognition and intervention in neurological emergencies are crucial to improving patient outcomes. Continuous advancements in diagnostic and therapeutic strategies, along with ongoing professional education, are essential for effective management.

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