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HYPERTENSIVE ENCEPHALOPATHY: THERAPEUTIC STRATEGIES AND CHALLENGES IN ACUTE MANAGEMENT

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Abstract: Hypertensive encephalopathy (HE) is a medical emergency characterized by acute neurological dysfunction triggered by severe elevation of blood pressure, with the potential to cause vasogenic cerebral edema and, in severe cases, seizures and coma. This study, conducted as a narrative review of the literature, aims to discuss current therapeutic strategies, the main pharmacological agents used, and the challenges in the acute management of HE. The failure of cerebral autoregulation mechanisms and the disruption of the blood-brain barrier are central elements of the pathophysiology. The diagnosis is predominantly clinical and based on exclusion, and neuroimaging is essential to differentiate it from other neurological causes. Treatment requires a controlled reduction in mean arterial pressure by up to 25% in the first hour, using intravenous agents such as nicardipine, labetalol, and sodium nitroprusside. Factors such as therapeutic adherence, use of NSAIDs, and comorbidities directly influence the evolution of the condition. Nursing care is crucial, from initial screening to continuous monitoring, safe administration of drugs, and patient education. It is concluded that therapeutic success depends on a multidisciplinary approach, rigorous monitoring, and prevention of recurrence through education and adequate clinical follow-up.

Keywords: Hypertensive encephalopathy; Medical emergency; Blood pressure; Clinical management.

INTRODUCTION

Hypertensive encephalopathy (HE) is a medical emergency characterized by acute cerebral dysfunction triggered by a severe increase in blood pressure (Saalim and Ali, 2023). Although it is one of the less common manifestations of hypertensive crises, its severity requires immediate recognition and treatment to prevent permanent neurologi-

cal damage and other systemic complications (Saalim and Ali, 2023). The clinical presentation of HE is often progressive, beginning with symptoms such as severe headache, nausea, and vomiting, and progressing to mental status changes, visual disturbances, seizures, and, in severe cases, coma (Saalim and Ali, 2023; Wiraboonchai et al., 2025).

The pathophysiology of the condition is based on the failure of cerebral self-regulation mechanisms (Saalim and Ali, 2023). Under physiological conditions, the brain maintains a constant blood flow despite variations in systemic blood pressure (Wiraboonchai et al., 2025). However, a sudden and marked increase in blood pressure can exceed the upper limit of this autoregulatory capacity, resulting in hyperperfusion, disruption of the blood-brain barrier, and consequent vasogenic cerebral edema (Wiraboonchai et al., 2025; Saalim , and Ali, 2023). This condition is often reversible with proper blood pressure management, leading to complete resolution of symptoms (Saalim and Ali, 2023).

It is known that hypertensive encephalopathy is triggered by an increase in blood pressure that can occur intensely and in an unregulated manner. Cerebral perfusion is maintained through cerebral autoregulation, which in cases of hypertension leads to a compensatory increase in cerebral perfusion of this autoregulatory band, which can lead to cerebral edema. The brain operates within certain distinct limits, which involve the pressure that facilitates blood flow (50-150 mmHg) and the pressure considered standard for arteries (60-160 mmHg). As a result, this sudden increase in perfusion due to this increase in pressure can lead to the stimulation of vasoactive and inflammatory messengers that will further contribute to the destabilization of the blood-brain barrier (BBB). As a consequence of this destabilization, vasogenic edema may occur, a common component of hypertensive encephalopathy, which will present as headaches, changes in the level of consciousness, and potentially convulsive episodes. (Saalim and Ali, 2023).

Hypertensive encephalopathy, although rare, is one of the most serious complications of high blood pressure, associated with high morbidity and mortality rates if not identified and treated properly. It is estimated that up to 15% of patients with hypertensive crises may develop acute neurological symptoms, making early recognition of the syndrome an essential determinant for the best prognosis (Saalim and Ali, 2023).

The diagnosis of HTN is often a diagnosis of exclusion, confirmed by clinical improvement after controlled blood pressure reduction and exclusion of other cerebrovascular pathologies (Saalim and Ali, 2023). Acute management is challenging, requiring a delicate balance between rapid pressure reduction to protect target organs and caution to avoid an abrupt drop that could precipitate cerebral ischemia (Saalim and Ali, 2023). This review aims to discuss current therapeutic strategies, the pharmacological agents used, and the main challenges in the acute management of hypertensive encephalopathy.

METHODOLOGY

This study was conducted as a narrative review of the literature, with the aim of consolidating and analyzing therapeutic approaches and challenges relevant to the acute management of hypertensive encephalopathy. The search for articles was performed in the PubMed database, using the descriptors "Hypertensive Encephalopathy," "Diagnosis," and "Treatment," aligned with the Medical Subject Headings (MeSH) vocabulary. The search was optimized by combining the terms with the Boolean operators AND and OR. Review articles, case reports, and cohort studies that directly addressed the central theme were

included. The time criterion covered publications from the last five years. Works without direct relevance, duplicate articles, and publications with low methodological rigor were excluded. The selection of studies followed a two-phase process: initial screening by titles and abstracts, followed by a full analysis of the selected texts for the extraction and synthesis of information.

RESULTS

The approach to hypertensive encephalopathy is based on rapid diagnosis to rule out other causes of neurological dysfunction and the implementation of immediate, titrated intravenous antihypertensive therapy.

DIAGNOSTIC APPROACH

The diagnosis of HTN is established based on the clinical presentation of acute neurological symptoms in a patient with severely elevated blood pressure, typically above 220/120 mmHg (Saalim and Ali, 2023). The medical history is crucial for identifying potential triggers, such as non-adherence to antihypertensive medications or the use of substances that can raise blood pressure (Saalim and Ali, 2023; Priyanka et al., 2024). Neuroimaging is mandatory for differential diagnosis. Computed tomography (CT) of the skull is often the initial test due to its speed and availability, and is useful for ruling out intracranial hemorrhage (Saalim and Ali, 2023). Magnetic resonance imaging (MRI) is more sensitive for detecting vasogenic cerebral edema, particularly in the posterior regions, which is characteristic of Posterior Reversible Encephalopathy Syndrome (PRES), a common radiological manifestation of EH (Saalim and Ali, 2023; Priyanka et al., 2024). Laboratory tests, including complete blood count, metabolic panel, and renal function, are performed to assess the extent of target organ damage (Saalim and Ali, 2023; Plitman et al., 2024).

Imaging tests are essential for recognizing and evaluating the process of hypertensive encephalopathy. The tests of choice are usually computed tomography (CT) and magnetic resonance imaging (MRI), which are used both to evaluate possible brain abnormalities and to rule out other diagnoses such as strokes and other causes of bleeding. Although MRI is more sensitive in detecting cerebral edema, it is less accessible and takes longer to provide results when compared to CT, for example. However, both are fundamental neuroimaging techniques for the evaluation of cases of hypertensive encephalopathy, as they allow the identification of brain lesions and the differentiation of specific subtypes based on their findings. (Saalim and Ali, 2023).

THERAPEUTIC STRATEGIES AND BLOOD PRESSURE TARGETS

The primary treatment for HS consists of controlled reduction of blood pressure using intravenous antihypertensive agents (Saalim and Ali, 2023). The initial goal is to reduce mean arterial pressure (MAP) by no more than 20-25% in the first hour (Plitman et al., 2024). Excessively rapid reduction should be avoided to prevent hypoperfusion and cerebral ischemia (Saalim and Ali, 2023). Continuous monitoring of blood pressure, ideally every 5 to 15 minutes, is essential during the acute phase (Saalim and Ali, 2023).

PHARMACOLOGICAL AGENTS

The choice of intravenous antihypertensive agent is individualized based on the patient's comorbidities:

- Vasodilators: Sodium nitroprusside and nitroglycerin are options, the former being a potent arteriovenous vasodilator and the latter predominantly venous, useful in patients with concomitant myocardial ischemia (Saalim and Ali, 2023).

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- Calcium Channel Blockers: Nicardipine and clevidipine are effective and widely used. Clevidipine, with its ultra-short half-life, allows for precise titration but may induce reflex tachycardia (Saalim and Ali, 2023).
- Alpha and Beta-Adrenergic Blockers: Labetalol is a fast-acting agent that is safe in coronary artery disease but contraindicated in patients with asthma or significant bradycardia. Esmolol has a very short half-life, allowing for rapid dose adjustment (Saalim and Ali, 2023).
- Other Agents: Hydralazine, enalaprilat, phenoldopam, and phentolamine are other options used in specific clinical settings, such as in pregnant women or in catecholaminergic crises (Saalim and Ali, 2023).

ANTI-INFLAMMATORY AGENTS

Adverse effects of NSAIDs on blood pressure

- A meta-analysis shows that NSAIDs increase mean blood pressure by ~5 mmHg, even in patients treated for hypertension (Plitman et al., 2024).
- This effect occurs because NSAIDs inhibit prostaglandins, reducing natriuresis, increasing vasoconstriction, and decreasing renal flow and glomerular filtration rate. (Plitman et al., 2024)

Interaction with antihypertensive drugs

- The risk is higher in patients treated with **ACE inhibitors** (such as trandolapril used by the patient) compared to calcium channel blockers (such as amlodipine). (Plitman et al., 2024)
- Studies have shown that indomethacin can increase blood pressure by an average of 10/5 mmHg in patients taking enalapril, but does not significantly alter blood pressure in patients taking amlodipine. (Plitman et al., 2024)
- This is because ACE inhibitors limit the compensatory mechanisms to sodium retention and volume expansion induced by NSAIDs. (Plitman et al., 2024)

Clinical consequences

- In the case described, the headache was caused by uncontrolled hypertension. The use of indomethacin worsened the condition, leading to **hypertensive encephalopathy**.
- Discontinuation of NSAIDs and blood pressure control led to complete resolution of symptoms. (Plitman et al., 2024)

Management considerations

- It is not appropriate to increase the dose of RAS blockers to counterbalance the effects of NSAIDs, as this increases the risk of kidney damage.
- It is recommended to substitute alternative agents, such as calcium channel blockers. (Plitman et al., 2024)

Aspects of hypertensive encephalopathy

- This is a hypertensive emergency characterized by cerebral edema due to a sudden rise in pressure beyond cerebral autoregulation.
- Typical symptoms include headache, confusion, visual deficits, seizures, and, in severe cases, coma. Urgent treatment with pressure reduction usually resolves the symptoms. (Plitman et al., 2024)

PHARMACOLOGICAL DETAILS

The most up-to-date recommendations indicate that the choice of antihypertensive drug must take into account the patient's clinical condition and the presence of comorbidities, and therefore, individualized treatment should be established. Nicardipine and

clevidipine have gained prominence due to the benefits of rapid titration and favorable safety profile, especially in pediatric and geriatric settings (Saalim and Ali, 2023). On the other hand, labetalol is preferred when there is a risk of aortic dissection or concomitant cardiac injury, due to its combined alpha and beta-blocking action (Saalim and Ali, 2023). In cases associated with the use of drugs that alter renal function or potentiate sodium retention, such as nonsteroidal anti-inflammatory drugs, extra caution is recommended due to the risk of precipitating HE, as reported by Plitman et al. (2024).

NURSING CARE IN THE ACUTE MANAGEMENT OF HYPERTENSIVE ENCEPHALOPATHY

Nursing plays a strategic and indispensable role in the care of patients with hypertensive encephalopathy, especially in the acute phase. According to Potter, Agarwal, and Schaefer (2024), "blood pressure should be carefully normalized within 24 to 48 hours" and the reduction should be gradual, avoiding ischemic complications such as cerebral hypoperfusion. In this context, it is up to the nursing team to continuously monitor blood pressure and neurological status, ensuring an immediate response in case of clinical instability, in addition to collaborating with patient safety.

In addition, the authors emphasize that "excessively aggressive hypertensive therapy can cause MAP to fall below the self-regulating range [...] increasing the risk of stroke" (POTTER; AGARWAL; SCHAEFER, 2024). Nurses, therefore, must safely administer intravenous antihypertensive drugs in accordance with the medical prescription and local health unit protocol, strictly observing clinical effects and preventing abrupt reductions that compromise cerebral perfusion, in addition to ensuring a rapid and coordinated response with the multidisciplinary team to deal with adverse events immediately.

Another relevant aspect is clinical support. According to the referenced article, "continuous monitoring of complications such as seizures, cerebral edema, and multiple organ dysfunction is essential in the acute treatment of hypertensive encephalopathy" (POTTER; AGARWAL; SCHAEFER, 2024). Thus, nurses must maintain a clear airway, ensure adequate oxygenation, and prevent bronchoaspiration during seizures.

Finally, continuity of care must also be valued. As highlighted, "patient education is essential to prevent the recurrence of hypertensive encephalopathy" (POTTER; AGARWAL; SCHAEFER, 2024), which places nursing as a protagonist in providing guidance on treatment adherence and lifestyle changes.

DISCUSSION

Successful management of EH transcends simple blood pressure reduction, requiring an in-depth understanding of pathophysiology, precipitating factors, and the particularities of different patient populations.

The pathophysiology of HE, centered on cerebral autoregulation failure, dictates the therapeutic strategy (Saalim and Ali, 2023; Wiraboonchai et al., 2025). In patients with chronic hypertension, the cerebral autoregulation curve is shifted to the right, meaning that the brain is adapted to higher pressures to maintain perfusion (Saalim and Ali, 2023). An abrupt reduction in blood pressure to normal levels may, paradoxically, induce cerebral ischemia (Saalim and Ali, 2023). This fact reinforces the importance of the goal of gradually reducing MAP by 20-25% in the first hour, an approach that aims to restore blood-brain barrier function without compromising cerebral blood flow (Plitman et al., 2024).

Identifying triggering factors is crucial for preventing recurrence. Non-adherence to an-

tihypertensive treatment is a prominent cause of hypertensive crises and HE (Priyanka et al., 2024). In one case study, discontinuation of antihypertensive drugs for one week was sufficient to precipitate PRES (Priyanka et al., 2024). In addition, drug interactions can destabilize blood pressure control. The use of nonsteroidal anti-inflammatory drugs (NSAIDs), such as indomethacin, can antagonize the effect of antihypertensive drugs, especially renin-angiotensin-aldosterone system (RAAS) inhibitors, such as trandolapril, leading to severe hypertensive decompensation (Plitman et al., 2024). This risk underscores the need for careful review of medication and patient counseling (Plitman et al., 2024).

EH in pediatric populations has distinct characteristics. Kidney disease is the most common cause of hypertension and HE in children (Wiraboonchai et al., 2025). One study showed that children with kidney disease were older at the time of HE diagnosis and developed the condition more rapidly after diagnosis of the underlying disease, compared with children with other etiologies (Wiraboonchai et al., 2025). Generalized tonic-clonic seizures are the most common manifestation in this population (Wiraboonchai et al., 2025). Recurrent episodes of EH have been observed in patients with conditions involving endothelial injury, such as vasculitis, or in those using calcineurin inhibitors after hematopoietic stem cell transplantation (Wiraboonchai et al., 2025). Despite the severity of the acute condition, the prognosis in children is generally favorable with immediate treatment, with a high survival rate and recovery without neurological deficits (Wiraboonchai et al., 2025).

The complexity of the differential diagnosis and therapeutic management of HE reinforces the need for a multidisciplinary approach, involving intensivists, neurologists, and cardiologists, to optimize outcomes and prevent long-term complications (Saalim and Ali, 2023).

Prevention of HE depends, above all, on strict adherence to antihypertensive treatment. In a case report, Priyanka et al. (2024) described that in , after only seven days of drug discontinuation, a severe episode of PRES associated with AH was triggered. This observation reinforces the importance of patient education programs and systematic outpatient follow-up to reduce the risk of recurrence.

It is extremely important that a rapid diagnosis and appropriate management are carried out as early as possible in order to avoid possible neurological damage and associated complications. Adequate monitoring is essential for assessing the patient's blood pressure levels, which should be done gradually to avoid possible cerebral ischemia. In addition to the use of antihypertensive drugs to control the acute condition, it is also important to assess the possible existence of underlying causes such as renal dysfunction, electrolyte imbalances, or even the patient's lack of adherence to medication. (Saalim and Ali, 2023).

CONCLUSION

Hypertensive encephalopathy is a critical condition characterized by acute neurological dysfunction resulting from severe elevation of blood pressure. Although less frequent among hypertensive crises, it is a potentially fatal complication associated with vasogenic cerebral edema and generally reversible lesions of the white matter, especially in posterior regions.

Rapid diagnosis and immediate treatment are essential to prevent further neurological damage and systemic complications. Management should prioritize gradual and controlled reduction of blood pressure, avoiding abrupt drops that could precipitate cerebral ischemia. For this purpose, titratable intrave-

nous antihypertensive agents such as sodium nitroprusside, nicardipine, and labetalol are used, the choice of which should consider the patient's comorbidities and clinical context. In addition to blood pressure control, it is essential to identify and treat underlying causes, such as renal dysfunction, electrolyte imbalances, pheochromocytoma, renal artery stenosis, or even discontinuation of antihypertensive therapy. Close monitoring of neurological status, including changes in consciousness, visual function, and the presence of focal deficits, plays a central role in clinical follow-up.

Finally, therapeutic success and prevention of recurrence depend not only on acute treatment, but also on a multidisciplinary approach, combined with continuous monitoring and strict adherence to antihypertensive treatment, which are essential measures for reducing morbidity and mortality associated with this syndrome (SAALIM; ALI, 2023).

Hypertensive encephalopathy is a serious medical emergency that requires rapid recognition and careful treatment. According to Potter, Agarwal, and Schaefer (2024), gradual reduction of blood pressure, limited to up to 25% in the first hour and with normalization in 24 to 48 hours, is the main therapeutic strategy, as it minimizes the risk of ischemic and neurological complications. The use of intravenous antihypertensive agents, under strict monitoring, is the preferred method for achieving these blood pressure goals, preventing both the progression of brain damage and the risks of hypoperfusion.

In this process, nursing plays a decisive role, acting in the continuous monitoring of vital signs, the safe administration of drugs, and the maintenance of airways. In addition, as emphasized by the authors, "continuous monitoring of complications such as seizures, cerebral edema, and multiple organ dysfunction is essential in the acute treatment of hypertensive encephalopathy" (POTTER; AGARWAL; SCHAEFER, 2024). Health education is also fundamental, since "patient education is essential to prevent the recurrence of hypertensive encephalopathy" (POTTER; AGARWAL; SCHAEFER, 2024), which reinforces the responsibility of nursing in providing guidance on therapeutic adherence and healthy lifestyle habits.

From this perspective, it is important to highlight that the screening performed by the nursing team at the first moment of contact with the patient is a critical step in addressing this condition. The ability to identify early signs such as sudden onset of severe headache, changes in mental status, visual disturbances, persistent vomiting, and seizures represents a differential for the clinical outcome of affected patients. The trained eye of the nursing staff in screening not only speeds up diagnosis and treatment, but also strengthens the culture of patient safety, reducing risks associated with delays in recognizing clinical severity. Thus, screening should be understood not only as an initial admission process, but as a strategic mechanism for preventing adverse events and ensuring quality of care.

Thus, the management of hypertensive encephalopathy requires a multidisciplinary approach, supported by clinical protocols and effective communication. In this context, nursing is consolidated as a fundamental and indispensable axis for the safety, continuity, and humanization of care, contributing decisively to the reduction of morbidity and mortality and to better health outcomes.

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