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Hypertensive Crisis: An Emergent Condition-Treatment and Management Plans in Emergency Departments.

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Abstract:

Background: Hypertensive emergencies are characterized by a rapid and significant rise in blood pressure, often exceeding 180/110 mmHg, which can lead to acute organ damage and high morbidity rates. These emergencies require immediate medical intervention to prevent further complications, such as stroke, heart failure, and renal failure. The aim of this article is to provide an updated overview of hypertensive crises, focusing on the latest findings in assessment and management strategies within emergency departments (EDs).

Aim: The article aims to synthesize recent research on hypertensive emergencies, focusing on the epidemiology, risk factors, diagnostic evaluation, and treatment options available for emergency care providers. It also highlights existing knowledge gaps and offers insights into improving patient outcomes.

Methods: A comprehensive review of literature published between November 2008 and October 2023 was conducted using Medline, Embase, and Google Scholar, prioritizing randomized controlled trials (RCTs) and professional guidelines. The studies reviewed included both observational and interventional research relevant to hypertensive emergencies such as intracerebral hemorrhage, aortic dissection, and hypertensive encephalopathy.

Results: Hypertensive emergencies are rare, accounting for 0.6% of emergency department visits. Factors such as medication non-adherence, chronic conditions (e.g., heart failure, stroke), and socioeconomic

determinants of health contribute to the occurrence of these crises. The key pathophysiological mechanisms include vascular damage, endothelial dysfunction, and autoregulatory dysfunction in organ systems. Effective management of these emergencies includes careful monitoring and controlled lowering of blood pressure, with an emphasis on organ protection.

Conclusion: Hypertensive emergencies require urgent and precise medical intervention to prevent severe organ damage. While there is a need for clearer definitions and more research, current management strategies focus on controlled blood pressure reduction and addressing the underlying causes. More research is needed to improve outcomes and minimize complications, particularly in high-risk populations.

Keywords: Hypertensive emergencies, acute organ damage, blood pressure management, emergency departments, stroke, aortic dissection, hypertensive encephalopathy.

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Introduction:

Rapid and substantial blood pressure increase along with acute organ damage and a high risk of morbidity are the hallmarks of hypertensive emergencies. These situations are frequently linked to blood pressure readings above 180 mmHg systolic and/or 110 mmHg diastolic, while there is no widely recognized threshold. Although the primary audience for this study is doctors who treat patients in hospital and emergency department settings, it also offers advice for those who provide outpatient treatment to patients who have severe hypertension. With the exception of hypertension disorders of pregnancy, which have recently been covered in other literature, the discussion offers a current summary of the epidemiology, diagnostic methods, and treatment options for adults experiencing hypertensive emergencies. The focus is on recent findings about assessment and management, emphasizing knowledge gaps in the area [1]. For publications published between November 2008 and October 2023, a thorough search of Medline, Embase, and Google Scholar was carried out using keywords such "hypertensive emergency," "malignant hypertension," "hypertensive crisis," "hypertensive urgency," and "hypertensive encephalopathy." Prioritizing randomized controlled trials (RCTs) pertinent to intracerebral hemorrhage, aortic dissection, posterior reversible encephalopathy, and hypertensive encephalopathy, the focus was on observational and interventional investigations. Professional guidelines were reviewed in the absence of primary research, and studies were chosen based on their design quality, relevance, and recentness. This review does not follow systematic review procedures, while being well-informed by the literature. The clinical viewpoint was also influenced by information gleaned from casual interviews with individuals who had encountered hypertensive crises.

Epidemiology

About 1.3 billion people worldwide suffer from hypertension, although less than 20% of them are able to control it effectively [2]. Hypertensive emergencies are uncommon, despite the fact that hypertensive crises are frequently seen in hospital and outpatient settings. Blood pressure values greater than 180/110 mmHg were present in 4.6% of more than two million outpatient visits in an integrated health system [3]. Hospitalizations for hypertensive events increased marginally in the United States from 101 per 100,000 in 2000 to 111 per 100,000 in 2007 [4]. According to 2013 ED data, hypertensive emergencies accounted for 0.6% of visits (618 per 100,000) [5]. Men present at a younger mean age (55 years versus 62 years) and make up a somewhat larger percentage of cases (52.5%) than women (47.5%) [6]. The majority of hypertensive emergency admissions are for Black patients (42.6%), followed by white patients (40.1%) and Hispanic patients (11.3%). Disparities are mostly caused by unfavorable socioeconomic determinants of health (SDoH) rather than race alone [7].

Risk Factors for Hypertensive Crisis

Heart failure, stroke, coronary artery disease, renovascular hypertension, chronic renal disease, alcoholism, and recreational drug use are among the cardiovascular disorders that share several risk factors with hypertensive crises [8]. Diabetes, hyperlipidemia, and chronic kidney disease are other correlations,

while uncommon illnesses including inflammatory vascular disorders and pheochromocytoma can also cause crises. Emergency room visits for severe hypertension are increased by medication non-adherence, lack of health insurance, and negative SDoH; however, there is conflicting evidence that these characteristics are associated with actual hypertensive emergencies [9].[10]. Although it only accounts for 0.5% of emergency visits, organ damage is commonly seen in individuals with hypertensive episodes. Ischemic stroke (28.1%), heart failure or pulmonary edema (24.1%), hemorrhagic stroke (14.6%), acute coronary syndrome (10.8%), renal failure (8%), subarachnoid hemorrhage (6.9%), encephalopathy (6.1%), and aortic dissection (1.8%) are among the disorders that are related with this condition [11]. At 9.9%, in-hospital mortality is still high.

Classification of Hypertensive Emergencies

The terms "malignant hypertension" and "hypertensive urgency" are frequently used inconsistent and may be deceptive when referring to hypertensive emergencies. Acute organ damage is a distinctive characteristic of hypertensive emergencies, which are usually defined by blood pressure levels more than 220/110 mmHg [4][12][13][14]. However, in situations when there are sudden rises from baseline, including in younger patients with acute renal injury, they may appear at lower pressures. It's critical to distinguish between asymptomatic chronic hypertension and hypertensive emergencies, as well as temporary blood pressure increases brought on by stress, discomfort, or physical activity. About 4-6% of cases involve severe hypertension without end-organ damage, while 0.6% to 1.0% involve hypertensive emergencies, which are characterized by acute end-organ damage necessitating a quick drop in blood pressure. Neurological disorders (such as encephalopathy and strokes), cardiovascular problems (such as myocardial ischemia and aortic dissection), obstetric complications (such as pre-eclampsia), and other disorders like acute kidney injury or severe hypertensive retinopathy are examples of symptoms that are exclusive to a particular organ. Deceptive phrases like "hypertensive urgency" and "hypertensive crisis" frequently convey an unwarranted sense of urgency for treatment. Furthermore, the term "malignant hypertension" is being reinterpreted to refer to microvascular damage that affects three or more target organs.

Acute end-organ injury mostly impacts the cardiovascular, ophthalmologic, hematologic, renal, and cerebrovascular systems. Acute kidney injury, hypertensive encephalopathy, acute heart failure with pulmonary edema, and intracerebral hemorrhage are important instances. When no major new or increasing organ damage is seen, patients with very raised blood pressures—often reaching 180/110 mm Hg—are diagnosed with considerably elevated or acute severe hypertension. When there is no immediate end-organ damage, even pressures higher than 220/110 mm Hg do not qualify as a hypertensive emergency. Patients with extremely high blood pressure have historically been referred to as having "hypertensive urgency" in numerous papers and guidelines [17, 18]. However, the word "urgency" suggests that therapy must be started right once, even when there is evidence that such measures are harmful and have little therapeutic benefit [3]. Furthermore, this language has drawn criticism for possibly making patients feel overly anxious [19]. In new guidelines, the European Society of Hypertension has called attention to this term's ambiguity [20]. Further complicating the language, the term "hypertensive crisis" has also been used to refer to episodes of extreme hypertension with or without immediate organ injury [20]. In the past, the phrase "malignant hypertension" described vascular damage brought on by the absence of regular autoregulatory blood flow processes, which resulted in structural damage including fibrinoid necrosis and onion skinning in small arterioles. On clinical examination, these findings frequently match hypertensive retinopathy [21, 22]. When the word "malignant" was initially used about a century ago, it reflected the bleak outlook for this illness prior to the development of efficient antihypertensive medications. The contemporary conceptualization of malignant hypertension places emphasis on microvascular destruction in conjunction with damage to three or more organ systems, but this condition is nonetheless linked to severe morbidity [23–26].

Due to the use of expert opinion, outdated terminology in clinical guidelines, and the scarcity of reliable randomized controlled trials, there are still significant disagreements regarding the definition of

hypertensive emergencies [27]. Although the exact causes are frequently unknown, acute organ damage continues to be the hallmark of hypertensive emergencies. Acute medical problems like myocardial ischemia, aortic dissection, or stroke, for example, can cause significant physiological stress and secondary blood pressure increases. In certain situations, it might be difficult to determine if the increase in blood pressure is a reactive phenomenon or a direct cause of the condition (a real hypertensive emergency). Reducing blood pressure immediately is clinically advantageous in real hypertensive emergencies, but it is useless in reactive situations like post-ischemic stroke. The necessity for more sophisticated definitions is highlighted by this complexity. Two essential requirements should be met by a hypertensive emergency: (1) The urgent medical condition is directly caused by or made worse by extreme hypertension, and (2) there is evidence that lowering blood pressure right away has inherent clinical benefits. An example of a condition where blood pressure control is crucial is hypertensive encephalopathy. On the other hand, diseases like acute ischemic stroke frequently have high blood pressure that is not the cause and does not require immediate medical attention. Additionally, there are situations in which end-organ damage and severe hypertension coexist, but since the hypertension is not directly linked to the organ damage, lowering blood pressure right away is not helpful.

Pathophysiology

The fundamental causes of hypertensive episodes are complex and yet poorly understood. A cursory overview is given, although a thorough mechanistic investigation is outside the purview of this discussion. Chronic endothelial damage, including oxidative stress and decreased nitric oxide production, is intimately linked to uncontrolled hypertension [28]. Vascular narrowing and decreased compliance, notably in the cerebral circulation, are caused by structural alterations including arteriole thickening and atherosclerosis. One of the main hypotheses is that an abrupt increase in systemic vascular resistance exacerbates the vascular bed's autoregulatory dysfunction. A feed-forward process of rising blood pressure is the result of this cascade, which also causes microcirculatory damage, increased renin-angiotensin

system activation, vasoconstriction, pressure natriuresis, and volume depletion.

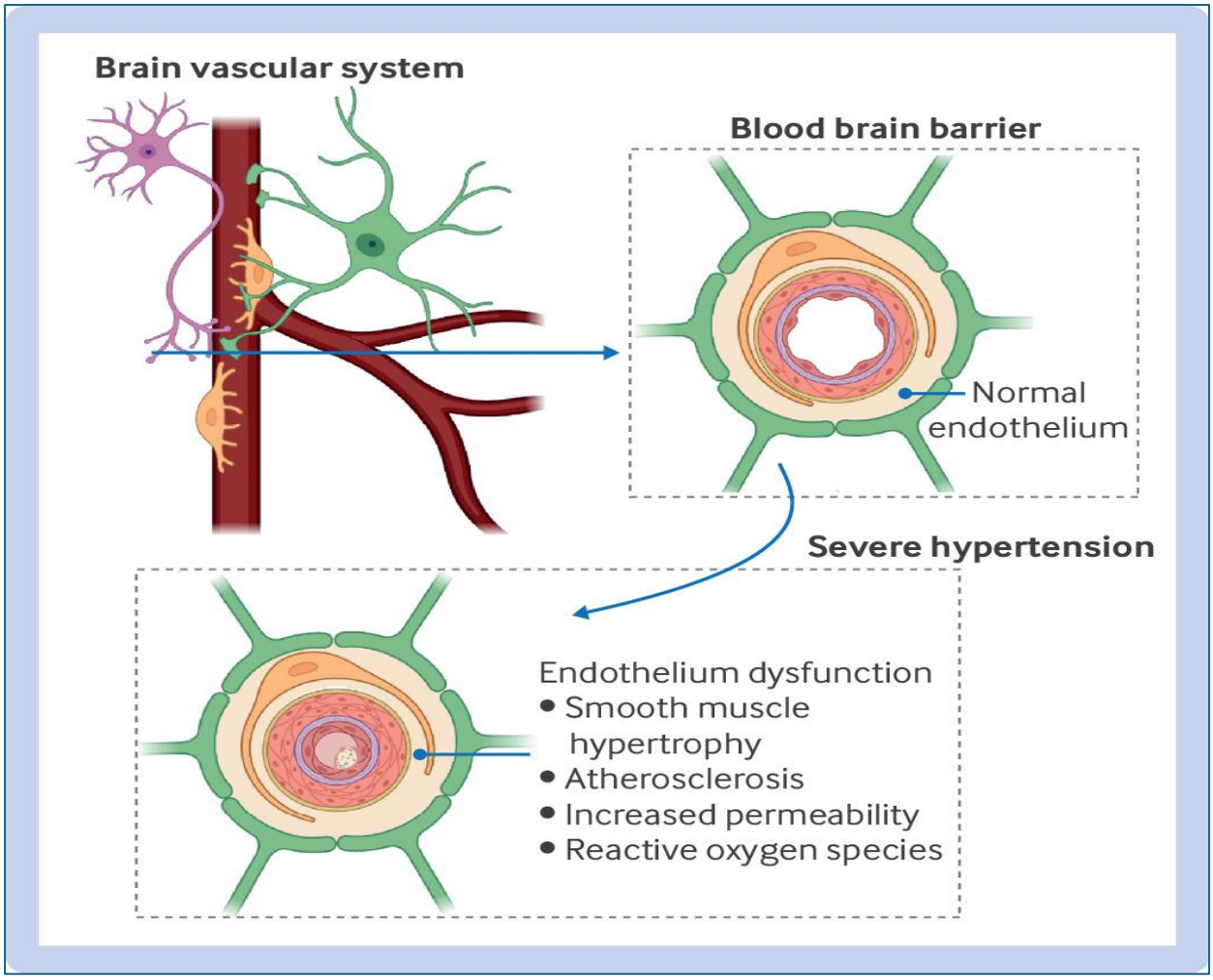

Figure 1: Neurovascular Remodeling in case of Severe Hypertension.

In order to manage hypertensive situations, it is essential to comprehend how hypertension affects brain circulation. Stable blood flow is maintained by cerebral autoregulation throughout a wide range of systemic arterial pressures. The highest limit of this range for people with normotension is roughly 150 mm Hg mean arterial pressure [29, 30]. This threshold is raised in patients with chronic uncontrolled hypertension, nevertheless, due to pathological vascular remodeling and compromised smooth muscle function [31]. As a result, patients experiencing hypertensive crises may be able to withstand mean arterial pressures well above 150 mm Hg [32]. Even in cases of extremely high blood pressure, these modifications frequently reduce the chance of immediate brain or end-organ damage. However, hazards such watershed ischemia and renal damage are associated with excessively severe blood pressure decrease [33–36]. Particularly in people with chronic hypertension, who may have reset cerebral autoregulatory limits, rapid drops in blood pressure may worsen negative cerebrovascular effects. Even though they can be reversed, these alterations need to be carefully managed in order to gradually return brain autoregulation to normal levels.

Diagnostic Evaluation of Suspected Hypertensive Emergencies

The assessment of important organ systems and the application of suitable diagnostic procedures are given first priority when evaluating suspected hypertensive crises. One essential but frequently disregarded initial step is taking an accurate blood pressure reading. It is typical for healthcare settings to deviate from best practices. The need for additional intervention can frequently be avoided by reassessing blood pressure using a cuff that is appropriately sized and positioned while making sure the patient is

seated and at ease [37]. Remeasurement following appropriate analgesia can often normalize high results in patients with severe pain. Continuous invasive arterial blood pressure monitoring is crucial in critical situations, such aortic dissection. Non-invasive automatic cuffs of the right size are adequate for titrating antihypertensive medication in less severe situations.

Clinical Evaluation

Clinical examination focuses on detecting symptoms and indicators of organ harm once severe hypertension has been diagnosed and found not to be reactive to stress or pain [38–41]. Prior diagnoses of pheochromocytoma, chronic kidney disease, cardiovascular and endocrine problems, and hypertension are all part of a comprehensive medical history. Adherence to prescribed medicine, usage of sympathomimetic drugs, and recent alcohol use or withdrawal are additional factors to consider [42]. Amphetamines, cocaine, and methamphetamines are among the substances that are known to cause hypertensive crises. Pregnancy-related factors must also be evaluated in females of reproductive age. When assessing severe hypertension, neurological and ocular evaluations are essential. Whereas focal neurological abnormalities point to a potential intracerebral hemorrhage or ischemic stroke, seizures or changed mental status point to hypertensive encephalopathy. Despite being common (affecting about 25% of those with severe hypertension), headaches are not always a sign of an emergency involving hypertension [39]. Fundoscopy and other ophthalmic evaluations are used to gauge the severity of hypertensive retinopathy. Arteriovenous pinching and arteriole tortuosity are chronic symptoms that point to mild retinopathy. On the other hand, moderate retinopathy is indicated by acute signs such as microaneurysms, cotton-wool patches, and flame hemorrhages. Severe retinopathy is indicated by papilledema, which calls for an urgent drop in blood pressure [43].

In hypertensive situations, cardiovascular symptoms such as palpitations, claudication, dyspnea, and chest discomfort are commonly noted (26%, 29%, and 29%, respectively) [44]. Signs of acute heart failure, such as peripheral edema, pulmonary rales, additional heart sounds, and jugular vein distension, or vascular abnormalities, such as abdominal bruits and pulse irregularities, may be discovered during a physical examination. Diaphoresis, recurrent headaches, and autonomic instability are among symptoms that may indicate pheochromocytoma. By evaluating five essential symptoms, discomfort, headache, dyspnea, visual abnormalities, and neurological symptoms methodical methodology has been put forth to distinguish between actual hypertensive emergencies [45]. With a negative predictive value of 99%, retrospective evaluations indicate that the lack of these symptoms successfully excludes out hypertensive emergencies [46]. The low positive predictive value (23%), however, for the presence of any symptom highlights how uncommon genuine crises are in asymptomatic people with severe hypertension. To improve diagnostic accuracy, predictive models must be further improved.

Diagnostic Testing: Laboratory and Imaging Evaluations

Laboratory Testing

There is little need for diagnostic testing in patients with acute severe hypertension who do not exhibit any symptoms. Nonetheless, laboratory testing should correspond with particular clinical presentations and the possible involvement of target organs when symptoms suggestive of hypertensive emergencies are present. A complete blood count (CBC) with differential, an electrocardiogram (ECG), and a metabolic panel that measures serum potassium, sodium, creatinine, and estimated glomerular filtration rate are all considered essential evaluations. Two important markers of hypertensive emergencies are the presence of fragmented red blood cells and acute renal damage. Thyroid function tests, protein urinalysis, and urine sediment examination for erythrocytes, leukocytes, and casts are examples of additional laboratory evaluations [37, 47]. Tests for secondary causes of hypertension, such as plasma renin activity, aldosterone levels, and catecholamine levels, are rarely performed in emergency situations, despite the advice of certain specialists. Results may be confused by renin-angiotensin system activation, secondary hyperaldosteronism, volume depletion, and increased sympathetic activity, all of which are frequently seen

in cases of severe hypertension. Although they contribute to hypertension, conditions such as primary aldosteronism and pheochromocytoma are usually poorly described in hypertensive situations.

As a result, diagnostic testing for secondary reasons is frequently postponed until the patient has stabilized, with the exception of hyperadrenergic states such pheochromocytoma or drug-induced crises, where certain treatments could be necessary [42, 48–51]. In addition to giving predictive information, circulating biomarkers such as brain natriuretic peptide (BNP), NT-proBNP, and high-sensitivity troponin provide important insights into heart failure, myocardial damage, and cardiovascular symptoms. Elevated lactate dehydrogenase (LDH), a sign of thrombotic microangiopathy, may also help detect hypertensive emergencies; observational studies have linked LDH levels above 190 U/L to these circumstances, but further study is required to confirm their usefulness [52]. Although they have been suggested, other possible indicators such urinary albumin-to-creatinine ratios, D-dimer, plasminogen activator inhibitor-1, and C-reactive protein have not yet been proven to have clinical uses in hypertensive situations [54].

Imaging Evaluations

Imaging approaches for hypertensive crises are based on presumed organ involvement and are symptom-driven. Computed tomography (CT) and plain radiography are common components of standard imaging. Non-contrast head CT is essential for identifying intracerebral bleeding in patients who report with altered mental status and significantly high blood pressure, but it is insufficient for diagnosing hypertensive encephalopathy. Because of its higher sensitivity, magnetic resonance imaging (MRI) is the preferable method for detecting vasogenic edema, which is frequently seen in parieto-occipital patterns that are consistent with posterior reversible encephalopathy syndrome (PRES). In around 65% of cases, MRI results may include microhemorrhages and hyperintense lesions on T2-weighted or FLAIR sequences [55]. Ultrasonography is becoming more and more useful in hypertensive situations. Evaluation of left ventricular hypertrophy, systolic and diastolic dysfunction, atrial dilatation, and possible aortic coarctation is made easier with echocardiography. Acute aortic dissection can be diagnosed with good specificity using bedside ultrasonography, especially if the abdominal aorta is involved [56]. By seeing B-lines, lung ultrasonography is a useful technique for identifying acute pulmonary edema [57]. Understanding the long-term vascular effects of hypertension is aided by further sophisticated ultrasonographic methods, such as renal artery duplex scanning for stenosis and carotid ultrasonography to evaluate stenosis or plaques. The ERIDANO observational trial, which examines arterial stiffness after ED visits for severe hypertension, shows how pulse wave analysis devices are becoming useful for measuring arterial stiffness and subclinical vascular damage [58,59].

Management of Severely Elevated Blood Pressure without Emergencies

Whether a hypertensive emergency requiring immediate care is present determines how to treat significantly raised blood pressure. Most individuals who are evaluated for hypertensive emergencies are later diagnosed with acute severe hypertension after it is discovered that they do not have acute organ damage. Usually, an outpatient setting can be used to care for these patients. Their short-term risk of adverse cardiovascular events is still low, despite their high long-term cardiovascular risk [38]. 4.6% of 59,535 patients in outpatient clinics had blood pressure readings higher than 180/110 mmHg, according to a retrospective investigation. Of them, 58.2% were using two or more antihypertensive drugs, and 72.9% had a history of hypertension. For this population, referral to the ED or hospitalization did not result in better results [3]. A short observation period might be suitable for acute severe hypertension cases in the emergency room where it is still unclear how to distinguish between hypertensive urgency and emergency. Evidence indicates that calm relaxation by itself can lower blood pressure in persons at low risk of hypertensive emergencies in a way that is equivalent to pharmaceutical therapies. After two hours, the mean blood pressure drop for 138 patients in a randomized trial was almost the same for both rest and telmisartan treatment (32.2 mmHg vs. 32.8 mmHg; non-significant difference, P=0.065) [60]. Similarly, mindfulness practices that have been reported to lower systolic blood pressure include number counting and pursed-lip breathing. Within three hours, the intervention group in a 110-patient trial showed a mean systolic pressure drop of 9.8 mmHg more than the usual care group (95% CI, 4.1 to 15.5) [61]. In certain

situations, anxiolytic drugs have also shown promise. For example, a pilot study comparing captopril with oral diazepam showed similar drops in systolic blood pressure (from 208 to 181 mmHg and from 213 to 170 mmHg, respectively) [62][63]. It makes sense to start taking oral drugs in accordance with guidelines when there is no previous antihypertensive regimen in place while awaiting more diagnostic testing [17][18]. To ensure successful outpatient management, it is imperative to address adherence hurdles such budgetary limitations, gaps in education, and logistical difficulties. Six months following an ED visit for severe hypertension, up to 65% of patients still have uncontrolled hypertension in spite of these efforts [3].

In-Hospital Management of Acute Severe Hypertension

Acute severe hypertension can develop in hospitalized patients who were treated for non-hypertensive emergencies for a variety of reasons, such as pain, anxiety, insomnia, or fluid overload. As-needed oral antihypertensives (such as clonidine or labetalol), slow titration of long-acting oral medicines, or, less frequently, intense intravenous therapy are some of the many different management strategies used in these situations. The use of as-needed drugs and intense intravenous treatments without obvious signs of a hypertensive emergency, however, may have negative effects, according to observational studies, and they should be avoided [64][65][66]. Intravenous antihypertensive medication was linked to increased risks of adverse events, such as acute renal injury and transfers to the intensive care unit, according to a retrospective analysis of 66,140 patients admitted for non-cardiovascular reasons. About 15% of individuals who received treatment had systolic blood pressures higher than 180 mmHg [64]. A research that included almost 23,000 patients from 10 hospitals found that individuals receiving therapy for acute hypertension had higher incidence of myocardial infarction and acute renal damage [65]. Furthermore, an analysis of 2,189 cases revealed that only 3% of patients fulfilled the rigorous requirements for intravenous antihypertensive therapy; yet, these patients remained in the hospital for longer than those who did not get treatment [66]. Clinical guidelines urge the gradual and careful titration of oral antihypertensive medications as the preferred in-hospital treatment technique. Except in rare cases, intravenous treatment is rarely necessary. Blood pressure regulation frequently improves significantly when contributory causes like pain, anxiety, or withdrawal symptoms are addressed [40]. Long-term outpatient techniques must be used to effectively control persistent hypertension and lower the risk of future problems, even though prompt therapies are required for acute end-organ damage [38][67].

Management of Hypertensive Emergencies

Clinical guidelines for managing hypertensive emergencies consistently advocate for immediate intervention when organ damage is evident. The recommended approach involves a phased strategy: an initial moderate reduction in blood pressure, continuous monitoring of clinical status, and gradual subsequent adjustments toward baseline levels. While general recommendations are outlined, treatment must be tailored to the specific etiology, comorbid conditions, and unique clinical scenarios of the patient. In most cases, rapid blood pressure reductions are discouraged to prevent ischemia in areas with impaired vascular autoregulation due to chronic hypertension. The intensity of blood pressure-reduction is proportionate to the risk of further injury. Treatment recommendations prioritize rapid acting, titratable intravenous antihypertensive agents, with continuous infusions preferred for initial management. Intravenous hydralazine and nitroprusside are avoided due to their unpredictable effects and the potential for abrupt blood pressure decreases [66, 76].

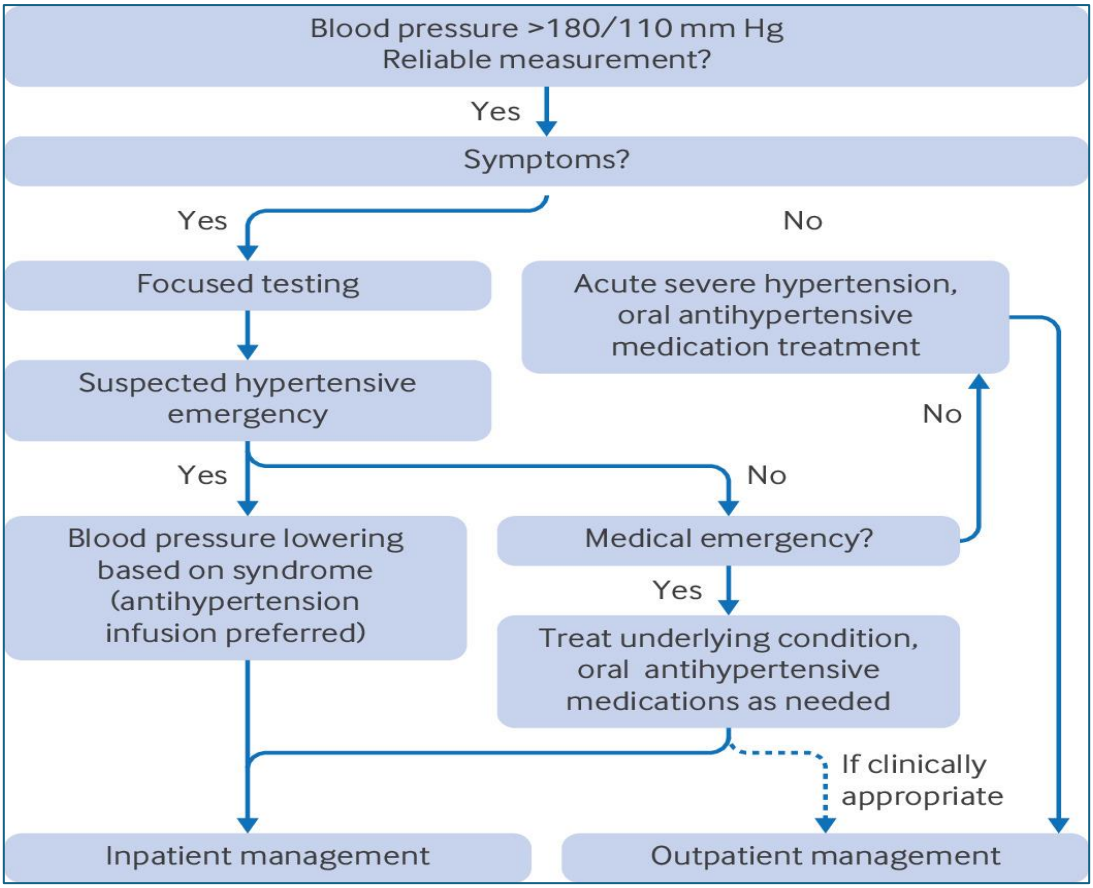

Figure 2: Management of Hypertension Crisis.

Hypertensive Encephalopathy

Changes in mental status linked to significantly increased blood pressure that beyond cerebral autoregulation thresholds are the hallmark of hypertensive encephalopathy. Seizures, headaches, tiredness, and vision abnormalities are possible symptoms, and blood pressure frequently rises above 220/110 mmHg. Those who are younger or have no history of persistent hypertension may experience these spikes at comparatively lower thresholds. Microangiopathic hemolytic anemia and acute hypertensive retinopathy frequently occur together. Clinical evaluation and ruling out other neurological emergencies, like ischemic or hemorrhagic stroke, are the mainstays of diagnosis; MRI often shows patterns linked to posterior reversible encephalopathy syndrome. In order to achieve tolerance, management entails a rapid 20–25% drop in mean arterial pressure (MAP) within the first hour, followed by stability over the course of two to six hours. First-line medications such as labetalol, clevidipine, and nicardipine are all effective; nicardipine is preferred due to its simplicity of titration. If intravascular depletion from pressure natriuresis causes MAP drops to be greater than expected, fluid resuscitation is required. Further progressive reductions to about 160/110 mmHg over 48 hours are suitable after initial stabilization [37, 77].

Aortic Disease

Dissection and other acute aortic syndromes require prompt afterload reduction, with a goal of systolic blood pressure below 120 mmHg and heart rate below 60 beats per minute. As a first-line treatment, current U.S. and European guidelines recommend β-blockers in combination with calcium channel blockers; esmolol, nicardipine, or clevidipine are recommended because of their quick onset and ease of titration [77, 78]. Historically used, nitroprusside has lost favor because of its negative side effects, which include cyanide poisoning and reflex tachycardia. More drastic MAP decreases (>25%) are necessary for aortic emergencies in order to reduce the chances of dissection advancement and death. Excessive decreases, however, could increase the risk of cerebral hypoperfusion, particularly in those with

persistently high autoregulatory limits. Increased rates of cerebrovascular damage were seen in a retrospective cohort study involving 112 patients who had type B aortic dissection, with MAP decreases above 25% [35].

Hemorrhagic Stroke and Subarachnoid Hemorrhage

There is strong evidence to support the therapy of ischemic and hemorrhagic stroke in hypertensive situations. Professional organizations such as the European Society of Cardiology and the International Society of Hypertension advise prompt systolic blood pressure drops to less than 130 mmHg in cases of hemorrhagic stroke. In order to prevent hypoperfusion injury, the American Heart Association/American Stroke Association (AHA/ASA) recommends a target of 140 mmHg, with higher thresholds permissible for first readings surpassing 220 mmHg [47]. INTERACT-2, ATACH-2, and INTERACT-3 are important experiments that helped shape these recommendations. In contrast to less demanding aims (180 mmHg), the INTERACT-2 trial, which included 2,839 participants, showed the safety of intense blood pressure targets (140 mmHg) but not their considerable efficacy [68]. Similarly, there was no discernible difference in results based on target ranges in the ATACH-2 study [80]. Intensive management as part of a comprehensive care approach is supported by the INTERACT-3 study, which included blood pressure control as part of a multimodal therapy bundle and reported lower death and disability [69]. A target systolic blood pressure of 140 mmHg is suitable for the majority of patients, and short-acting calcium channel blockers like clevidipine and nicardipine are recommended. AHA/ASA recommendations indicate gradual reductions for systolic pressures surpassing 180 mmHg [81], although blood pressure is usually less significant in cases of aneurysmal subarachnoid hemorrhage. In these situations, medications like nicardipine continue to work well.

Ischemic Stroke Management:

In the acute management of ischemic stroke with hypertensive emergency, blood pressure targets are guided by the necessity for reperfusion therapy (thrombolysis and/or endovascular thrombectomy). For patients with ischemic stroke who are not receiving acute reperfusion therapy, immediate blood pressure reduction is generally not recommended unless the systolic pressure exceeds 220 mmHg, or the diastolic pressure exceeds 120 mmHg. In such cases, a gradual reduction over several days is typically appropriate. Two major trials conducted in China investigated the effects of immediate versus delayed blood pressure treatment in ischemic stroke. The first trial, CATIS, randomized 4,071 patients to either a 10-25% reduction in blood pressure within the first 24 hours or to withholding antihypertensive treatment. The trial found no significant difference in mortality or disability rates following hospital discharge (odds ratio 1.00; 95% CI 0.88 to 1.14). The subsequent CATIS-2 trial involved 4,810 patients with mild to moderate stroke and systolic pressures between 140 and 220 mmHg. It tested a 10-20% reduction within the first 24 hours, followed by gradual reduction to below 140 mmHg, versus allowing higher pressures. Again, there was no significant difference in outcomes at 90 days (odds ratio 1.18; 95% CI 0.98 to 1.41). For patients receiving acute reperfusion therapy, international guidelines recommend blood pressure goals of systolic <185 mmHg and diastolic <110 mmHg. Recommended antihypertensive agents for this group include continuous nicardipine or intermittent intravenous labetalol. Additionally, the AHA/ASA guidelines suggest maintaining blood pressure <180/105 mmHg for the first 24 hours post-reperfusion therapy. Recent studies, such as the ENCHANTED2 trial, have explored blood pressure management in thrombectomy patients. This trial randomized 821 patients to either intensive management (systolic BP <120 mmHg) or a moderate approach (systolic BP between 140-180 mmHg) following thrombectomy. The trial revealed that intensive management was associated with a greater likelihood of poor functional outcomes (odds ratio 1.37; 95% CI 1.07 to 1.76). The OPTIMAL-BP trial in South Korea also randomized 306 patients with large vessel occlusion stroke to intensive or conventional blood pressure management. It demonstrated potential harm from more aggressive blood pressure lowering (adjusted odds ratio for functional independence 0.56; 95% CI 0.33 to 0.96). Similarly, the BEST-II trial found no significant differences in infarct volumes or functional outcomes between patients with different systolic blood pressure targets post-endovascular therapy [82].

Cardiac Emergencies:

Cardiac hypertensive emergencies, including acute myocardial infarction (MI) and cardiogenic pulmonary edema with blood pressures exceeding 180/110 mmHg, necessitate careful management. Elevated afterload in these situations increases myocardial oxygen demand and strain, potentially exacerbating ischemic symptoms or leading to pulmonary edema. Pulmonary edema is more frequently observed than MI in these settings and is often associated with type 2 myocardial infarction, where ischemia arises from a mismatch in oxygen supply and demand, rather than from coronary artery disease. Diagnosis is confirmed by a rise and fall in cardiac troponin above the 99th percentile. Elevated troponin levels that do not show this characteristic rise and fall pattern are not indicative of a hypertensive emergency or type 2 myocardial infarction. It is important to note that chronic elevations in cardiac troponin may be present in patients with acute severe hypertension, but these do not necessarily require immediate blood pressure reduction. Blood pressure treatment is indicated when acute pulmonary edema or type 2 myocardial infarction is present. The goal of treatment is typically a moderate reduction of mean arterial pressure (15-25%) to relieve symptoms. Nitroglycerin is often the preferred agent due to its ability to reduce both preload and afterload, with titration based on symptom relief and mean arterial pressure. In cases where beta-blockade is not contraindicated, agents such as esmolol or intravenous labetalol are second-line options. For patients with acute cardiogenic pulmonary edema, nitroglycerin provides venodilation and afterload reduction at higher doses, while nicardipine and clevidipine can effectively reduce afterload. These agents, when used alongside loop diuretics like furosemide, can rapidly alleviate symptoms associated with pulmonary edema [83].

Conclusion:

Hypertensive crises are critical conditions that demand immediate medical intervention to avoid severe and potentially fatal complications. These emergencies typically present blood pressure readings exceeding 180/110 mmHg, accompanied by acute organ damage. While hypertensive emergencies are rare, accounting for only 0.6% of emergency department visits, the associated morbidity and mortality rates remain high. Notably, the underlying pathophysiology of these crises often involves complex interactions between endothelial dysfunction, vascular narrowing, and impaired autoregulation, which exacerbates the damage caused by extreme hypertension. A thorough diagnostic approach is essential in managing hypertensive emergencies. Accurate blood pressure measurements, along with a detailed clinical evaluation of potential organ damage, are critical. Common complications associated with hypertensive crises include ischemic and hemorrhagic strokes, heart failure, acute kidney injury, and hypertensive encephalopathy. Neurological and ophthalmic assessments are particularly important in diagnosing and managing these conditions. In addition, a comprehensive medical history, including the use of medications, lifestyle factors, and pre-existing conditions like chronic kidney disease and cardiovascular disorders, plays a crucial role in the diagnostic process. Management strategies focus on the controlled reduction of blood pressure to prevent further organ damage while avoiding rapid drops that may exacerbate cerebrovascular events, particularly in patients with chronic hypertension. Pharmacologic interventions, including intravenous antihypertensive medications, are commonly used in these critical scenarios. However, careful monitoring is required to adjust treatment based on individual patient needs and responses. Despite advancements in the understanding and treatment of hypertensive emergencies, significant gaps remain in the literature. The current terminology, including terms like "hypertensive urgency" and "malignant hypertension," has been criticized for its ambiguity and potential to misguide clinical decision-making. Additionally, the precise mechanisms by which hypertension induces acute organ damage are still not fully understood. As such, more high-quality, randomized controlled trials are necessary to establish clear guidelines and improve management protocols for hypertensive crises. In conclusion, hypertensive emergencies require rapid, yet carefully considered, interventions to prevent irreversible organ damage. Emergency care providers must remain vigilant in assessing risk factors, monitoring clinical symptoms, and adjusting treatment strategies to optimize patient outcomes. Enhanced research and clearer definitions are essential for refining the approach to managing these high-risk conditions.

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أزمة ارتفاع ضغط الدم: حالة طارئة - خطط العلاج والإدارة في أقسام الطوارئ

الملخص:

الخلفية :تتميز الطوارئ المرتبطة بارتفاع ضغط الدم بارتفاع سريع وكبير في ضغط الدم، وغالبًا ما يتجاوز 110/180 مم زئبق، مما قد يؤدي إلى تلف الأعضاء الحاد وزيادة معدلات الممراضة. تتطلب هذه الحالات تدخلاً طبيًا فوريًا لمنع حدوث المزيد من المضاعفات مثل السكتة الدماغية، وفشل القلب، وفشل الكلى. يهدف هذا المقال إلى تقديم نظرة محدثة حول الأزمات المرتبطة بارتفاع ضغط الدم، مع التركيز على أحدث النتائج في التقييم واستراتيجيات الإدارة في أقسام الطوارئ.

الهدف :يهدف المقال إلى تجميع الأبحاث الحديثة حول الطوارئ المرتبطة بارتفاع ضغط الدم، مع التركيز على الوبائيات، وعوامل الخطر، والتقييم التشخيصي، وخيارات العلاج المتاحة لمقدمي الرعاية الطارئة. كما يسلط الضوء على الفجوات المعرفية الحالية ويقدم رؤى لتحسين نتائج المرضى.

الطرق :تم إجراء مراجعة شاملة للأدبيات المنشورة بين نوفمبر 2008 وأكتوبر 2023 باستخدام قواعد البيانات Medline و Embaseو Google Scholar، مع إعطاء الأولوية للتجارب السريرية العشوائية (RCTs) والإرشادات المهنية. شملت الدراسات التي تمت مراجعتها الأبحاث الملاحظة والتداخلية المتعلقة بالطوارئ المرتبطة بارتفاع ضغط الدم مثل النزف الدماغي، وتمزق الشريان الأورطي، واعتلال الدماغ المرتبط بارتفاع ضغط الدم.

النتائج :تعتبر الطوارئ المرتبطة بارتفاع ضغط الدم نادرة، حيث تمثل 0.6% من زيارات أقسام الطوارئ. تساهم عوامل مثل عدم الالتزام بالأدوية، والحالات المزمنة (مثل فشل القلب والسكتة الدماغية)، والعوامل الاجتماعية والاقتصادية للصحة في حدوث هذه الأزمات. تشمل الآليات الفيزيولوجية المرضية الرئيسية تلف الأوعية الدموية، واضطراب وظائف البطانة الوعائية، واضطراب التنظيم الذاتي في أنظمة الأعضاء. تشمل الإدارة الفعالة لهذه الطوارئ مراقبة دقيقة وتقليص ضغط الدم بشكل محكوم، مع التركيز على حماية الأعضاء.

الخاتمة :تتطلب الطوارئ المرتبطة بارتفاع ضغط الدم تدخلًا طبيًا عاجلاً ودقيقًا لمنع تلف الأعضاء الحاد. على الرغم من الحاجة إلى تعريفات أكثر وضوحًا ومزيد من البحث، تركز استراتيجيات الإدارة الحالية على خفض ضغط الدم بشكل محكوم ومعالجة الأسباب الكامنة. هناك حاجة إلى مزيد من البحث لتحسين النتائج وتقليل المضاعفات، خاصة في الفئات السكانية عالية المخاطر.

الكلمات المفتاحية :الطوارئ المرتبطة بارتفاع ضغط الدم، تلف الأعضاء الحاد، إدارة ضغط الدم، أقسام الطوارئ، السكتة الدماغية، تمزق الشريان الأورطي، اعتلال الدماغ المرتبط بارتفاع ضغط الدم.