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A Hypertensive Crisis is an Emergency Condition of Sudden Increase in Blood Presssure

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Abstract

A hypertensive crisis is a critical medical situation characterized by a rapid spike in blood pressure that may result in severe outcomes like stroke, heart attack, or damage to organs. Immediate action is necessary to safely decrease blood pressure, typically through a combination of medications and medical intervention. Symptoms can include intense headaches, dizziness, chest discomfort, breathing difficulties, nausea, vision alterations, and feelings of anxiety. It is crucial to differentiate between hypertensive emergencies and hypertensive urgencies, as they necessitate distinct treatment strategies.

Keywords: Hypertension, Hypertensive Crisis, SBP, DBP, Health

Introduction

Hypertension is a widespread issue, and data from populations suggest that its prevalence is on the rise around the world [1]. Approximately one billion people across the globe currently experience hypertension. Roughly one-third of adults in the United States have hypertension. In contrast to two-thirds a decade ago, now only half of those with this condition manage to keep their blood pressure under control. One-third of individuals with uncontrolled hypertension remain unaware of their condition. The precise likelihood of experiencing a hypertensive crisis is uncertain, but the majority of experts estimate it to be under 1%.

Hypertensive emergency is characterized by an increased blood pressure coupled with signs of acute damage to vital organs. The risk of morbidity is substantial within hours if no therapeutic measures are taken, particularly when key organs such as the kidneys, heart, and brain sustain acute injury. Both the height of the blood pressure reading and how quickly it rises are critical in determining whether an emergency situation has developed. Typically, in hypertensive emergencies, the diastolic blood pressure exceeds 120 mm Hg. However, in children, pregnant women, and individuals who typically have normal blood pressure, a hypertensive emergency may occur with lesser increases in blood pressure. Early identification of this condition is crucial to preventing organ damage and initiating proper treatment upon diagnosis. Malignant hypertension refers to a specific condition where significantly elevated blood pressure is associated with hypertensive neuroretinopathy.

Patients experiencing hypertensive urgency exhibit heightened blood pressure (with systolic blood pressure often reaching 180 mm Hg and diastolic pressure often around 110 mm Hg) but show no signs of acute damage to their organs. This condition may relate to long-standing, stable issues such as stable angina, history of heart attack, chronic heart failure, chronic kidney failure, previous transient ischemic attacks, or past strokes, without the immediate threat of an acute incident.

The leading triggering cause of a hypertensive crisis is often non-adherence to prescribed medications in those already diagnosed with hypertension [2]. It is believed that certain vasoconstrictors in the blood lead to a sudden rise in systemic vascular resistance. This condition can harm small blood vessel linings, resulting in the accumulation of platelets and fibrin, along with the disruption of vascular autoregulation. The problem primarily lies with afterload rather than cardiac output. In most situations, treatment should focus on reducing afterload. If renal failure is not present, persistent hypertension can lead to natriuresis and a decrease in blood volume. In specific cases, mild volume replenishment with saline may be necessary. Renal failure or rapid heart rate may or may not be observed. Diuretic treatment would exacerbate the vasoconstriction issue.

Patients exhibiting a systolic blood pressure (SBP) exceeding 180 or a diastolic blood pressure (DBP) surpassing 110 are typically recognized as experiencing a hypertensive crisis. Such elevated pressures may lead to acute damage affecting the heart, brain, lungs, kidneys, retina, aorta, and small blood vessels. When there is damage to these organs, the situation is referred to as a "hypertensive emergency. " The term malignant hypertension is considered obsolete and has been removed from current blood pressure guidelines. The normal mean arterial pressure (MAP) typically lies between 70 to 100 mm Hg. The value of MAP that is double the baseline is not a recognized measurement. A patient experiencing a hypertensive emergency requires admission to the ICU for management of blood pressure via intravenous means. Hypertensive urgency refers to high blood pressure occurring without organ damage. Such patients can be treated with oral medications with the intent of gradually stabilizing blood pressure over a period of days to weeks.

Pathophysiology

The exact pathophysiology underlying hypertensive emergencies remains unclear [1]. A sudden spike in blood pressure often marks the shift from regular hypertension or normal blood pressure levels to a hypertensive emergency. Blood pressure is influenced by the combination of cardiac output and peripheral vascular resistance. The initial rise in blood pressure is probably a consequence of heightened vascular resistance. Substantial evidence indicates that mechanical stress on the arteriolar walls triggers vascular myogenic responses and compromises the integrity of the endothelium. When the integrity of the vascular system is compromised, widespread microvascular injuries appear. Fibrinoid necrosis in arterioles and myointimal proliferation are noticeable in susceptible organs and are regarded as key histological features of hypertensive emergencies. Damage to the endothelium also leads to a decrease in nitric oxide production. These maladaptive reactions contribute to elevated peripheral resistance and establish a harmful cycle, where relative reduction in blood flow due to vasoconstriction results in an increase in vasoactive substances. For instance, reduced blood flow activates the renin-angiotensin-aldosterone system, and research indicates that angiotensin II may directly harm vascular walls by stimulating pro-inflammatory cytokine gene expression such as interleukin-6 and nuclear factor kB. Other factors detrimental to the vascular system could also raise peripheral vascular resistance, including increased blood viscosity, immunological factors, and other hormones like catecholamines, vasopressin, and endothelin. Ultimately, these alterations produce a notable rise in peripheral vascular resistance, resulting in ischemia affecting the heart, brain, and kidneys.

In assessing hypertensive emergencies and their management, the effect of blood pressure on cerebrovascular function is crucial. Hypertensive encephalopathy, for instance, is a unique clinical condition that arises when swiftly elevating central perfusion pressures (CPPs) surpass the central nervous system's (CNS) capacity to maintain autoregulation. The autoregulation of cerebral blood flow (CBF) denotes the brain's capability to sustain consistent CBF within a CPP range of 60 to 150 mm Hg. In cases of chronic hypertension, the autoregulation range expands to between 80 to 160 mm Hg.

Under typical physiological circumstances, the reverse flow within the cerebral venous system or venous pressure remains close to zero, and the cerebral perfusion pressure (CPP) is influenced primarily by arterial pressure. In cases of severe brain injury, such as those resulting from subarachnoid hemorrhage, stroke, and intracranial bleeding, the brain's capacity to self-regulate and sustain cerebral blood flow (CBF) is compromised. A failure to self-regulate CBF is also evident during hypertensive emergencies when the mean arterial pressure (MAP) exceeds 140 mm Hg. This breakdown in autoregulation causes elevated blood pressure to be transmitted, leading to endothelial injury, disruption of the blood-brain barrier, fluid and protein leakage, vasogenic edema, and additional cerebral vasoconstriction, which can cause brain infarcts.

Pre-operative Hypertension

Management of pre-operative hypertension is essential, and when feasible, elective surgical procedures should be postponed until blood pressure is lowered to below 180/110 mmHg [3]. It is believed that at or below this threshold, patients do not face an increased risk of cardiac issues during the perioperative period. Hence, discontinuing antihypertensive medication before surgery should be avoided, as rebound hypertension can arise. Administering sedative premedication may help decrease the levels of endogenous catecholamines. After confirming and addressing inadequate anesthesia, medications like esmolol, labetolol, hydralazine, nifedipine, sodium nitroprusside, and glyceryl trinitrate may be utilized to manage hypertension. These should be titrated with caution, as anesthetic agents may enhance their actions.

A hypertensive crisis can manifest as part of hypertensive diseases, during pregnancy, in the presence of pheochromocytoma, or as a complication after cardiac or vascular operations. If left untreated, initial symptoms such as confusion, headaches, and visual disturbances can escalate to encephalopathy, seizures, and coma. Potential related issues may encompass retinal hemorrhages, papilledema, kidney dysfunction, heart failure, and stroke. Oral medications are generally recommended since intravenous antihypertensives may lead to a rapid decrease in blood pressure, which could in turn trigger renal damage, strokes, or myocardial ischemia.

APH

Acute postoperative hypertension (APH) is commonly recognized as a notable spike in blood pressure during the early postoperative phase, which can lead to serious clinical outcomes, including strokes, intracranial bleeding,

myocardial ischemia, heart failure, acute kidney failure, and complications at the surgical site or anastomosis [4]. Blood pressure usually rises within less than two hours after surgery and can stay elevated for several hours, although instances of sustained elevation lasting up to 48 hours have been noted. APH is considered to arise from adrenergic activation, vasoconstriction, and a subsequent increase in systemic vascular resistance, with significant surges in plasma catecholamine levels being recorded. Factors related to anesthesia and procedures may affect the occurrence of APH, which tends to be most prevalent following cardiovascular, neurosurgical, and head and neck surgeries. APH is particularly frequent after carotid endarterectomy, potentially due to baroreceptor stimulation.

Even though APH is a widely acknowledged condition, there is ongoing discussion regarding several critical aspects, such as an exact definition, its clinical significance, the suitable threshold for starting treatment, and the level of aggressiveness required for management. Although there are limited prospective evaluations studies indicate that therapy can be beneficial clinically, particularly after noncardiac surgical procedures. Postoperative hypertension is linked to higher rates of morbidity and mortality, suggesting that managing severe hypertension could enhance patient outcomes. Consequently, treatment choices typically take into account individual factors such as the patient's current and preoperative blood pressure readings, the severity of any coexisting health issues, the nature of the surgery conducted, and an assessment of the likelihood of surgical complications.

Although research has not definitively established the best blood pressure threshold for starting treatment, various criteria have been utilized in clinical trials, including a reading of 160/90 mmHg, a mean arterial pressure exceeding 110 mmHg, or a relative increase of 20% in either systolic or diastolic blood pressure compared to preoperative measurements. Conversely, the relationship between acute postoperative hypertension and complications has been more clearly established in cardiovascular procedures, with a typical threshold for initiating therapy set at a blood pressure of 140/90 mmHg or a mean arterial blood pressure of 105 mmHg.

Signs

Acute severe hypertension can lead to rapid deterioration in organ function, particularly if it arises suddenly [4]. Acute postoperative hypertension has been linked to negative clinical outcomes, which may include encephalopathy, both hemorrhagic and ischemic strokes, myocardial ischemia and heart attacks, congestive heart failure, aortic dissection, acute kidney failure, and complications from surgical sites or anastomosis. The symptoms of end-organ damage are often concealed in the postoperative phase because of sedation, pain relief medications, and altered levels of consciousness. Severe hypertension may serve as a physiological response to conditions such as myocardial ischemia, stroke, or increased pressure within the skull. Therefore, it is essential that any patient exhibiting significant postoperative hypertension undergo a thorough physical examination to evaluate potential end-organ dysfunction.

While hypertensive emergencies are rare in non-surgical situations when diastolic blood pressure remains below 130 mmHg, the speed of blood pressure changes plays a crucial role in the risk of end-organ damage, given the limitations of the body's autoregulatory mechanisms. Consequently, hypertensive emergencies may occur more frequently during the perioperative period, where there are often numerous hypertensive triggers. In non-surgical circumstances, common indicators of end-organ damage involve chest pain, shortness of breath, cognitive impairment, and headaches. Stroke-like symptoms that present focal effects cannot be solely attributed to hypertension and necessitate a thorough neurological assessment. Indicators of organ damage can manifest as pulmonary rales, cardiac murmurs or gallops, ischemic changes in the electrocardiogram (ECG), cognitive decline, reduced urine output with the presence of blood or protein in urine, or signs of papilledema in a fundoscopic exam.

Organ Damage

Severe hypertension characterized by readings greater than 180/120 mmHg, regardless of the presence of acute end-organ damage, can occur due to chronic hypertensive conditions, may arise in the postoperative phase following cardiac or vascular procedures, or can be related to other disorders such as pre-eclampsia and pheochromocytoma [5]. Such conditions are indicative of end-organ dysfunction:

- myocardial infarction, heart failure, and cerebrovascular accident.
- kidney failure.
- encephalopathy: disorientation, headaches, vision issues, seizures, unconsciousness.
- eye bleeding, swelling of the optic disc, and nosebleeds.
- in the absence of damage to end organs, oral medications are administered (such as atenolol, labetalol or nifedipine), with the goal of progressively lowering blood pressure to below 160/100 mmHg within hours to days. High doses or intravenous medications can lead to rapid drops in blood pressure, potentially causing stroke, loss of vision, kidney dysfunction, or heart ischemia.
- severe hypertension (for instance, above 220/130 mmHg) is generally linked to critical damage to organs, necessitating transfer to an intensive care unit for continuous blood pressure monitoring and immediate medication adjustments (for instance, a 15% to 20% decrease in blood pressure during the first hour, followed by a reduction to 160/110 mmHg in the next 2 to 6 hours). Effective substances include sodium nitroprusside, esmolol, and labetalol. Patients frequently experience sodium and fluid depletion, thus careful administration of isotonic saline is needed.

Emergency

A hypertensive emergency is typically characterized by uncontrolled high blood pressure along with damage

to organs, and it requires immediate treatment with adjustable intravenous agents [6]. Individuals experiencing a hypertensive emergency should be referred for ICU assessment.

An essential aspect of managing any hypertensive crisis is grasping the principle of autoregulation. Autoregulation refers to an organ's capacity to keep its blood flow stable regardless of changes in pressure. This response is inherent to the specific blood vessels of the organ and operates independently from neural or hormonal influences. Instances of autoregulation can be found throughout the body, but are most notable in the blood vessels of the brain, heart, and kidneys.

Nonetheless, autoregulation can only function effectively within a specified range of pressures. When blood pressure exceeds or falls below this range, alterations in blood pressure directly affect the small blood vessels. This scenario can induce ischemia during low blood pressure and damage due to excessive blood flow during high blood pressure. Additionally, persistent high blood pressure can shift the autoregulatory range to a higher pressure threshold. Rapidly reducing high blood pressure in such individuals may inadvertently result in insufficient blood flow and ischemic damage.

Traditionally, numerous terms have been utilized to describe different types of acute hypertensive situations, including hypertensive crisis, accelerated hypertension, hypertensive emergency, malignant hypertension, hypertensive encephalopathy, posterior reversible encephalopathy syndrome, hypertensive urgency, and uncontrolled hypertension [7]. Certain expert recommendations have streamlined these numerous terms into two main classifications: uncontrolled hypertension and hypertensive emergency, distinguishing between the lack (uncontrolled hypertension) or presence (hypertensive emergency) of acute damage to organs resulting from hypertension.

The classification of a hypertensive emergency occurs when pronounced hypertension (often over 180/120 mm Hg) leads to injury in the heart, eyes, brain, kidneys, major arteries, or microcirculation. A sudden increase in blood pressure without clear evidence of end-organ injury is characterized by uncontrolled high blood pressure. This classification separates individuals whose blood pressure needs immediate management from those whose blood pressure can be regulated safely over a period of hours to days. In a hypertensive emergency, it is essential to admit the patient to the hospital to address the effects of organ damage and to meticulously oversee the blood pressure reaction to intravenous medications aimed at lowering blood pressure. Conversely, uncontrolled high blood pressure can be addressed with standard oral medications and can be monitored with limited observation and follow-up in an outpatient setting, making hospital admission unnecessary. For a hypertensive emergency, the specific profile of organ injury influences the selection of antihypertensive medication, the speed at which blood pressure should be decreased, as well as the timing and final blood pressure targets in response to treatment.

Management

Patients experiencing a hypertensive emergency should receive treatment via injections while being closely monitored in an intensive care environment, using either arterial cannulation or automated blood pressure measurement methods [1]. Generally, the necessity to lower blood pressure and the speed at which it happens rely on the clinical circumstances. Sudden drops in blood pressure should be avoided as they can lead to renal, cerebral, and coronary ischemia.

In most cases, blood pressure can be rapidly decreased by 20% to 25% over minutes to hours. Although the autoregulatory range of cerebral blood flow (CBF) may shift higher in chronic hypertension, the lower limit of autoregulation remains about 25% below the resting mean arterial pressure (MAP) for individuals with both normal and chronic hypertension. When blood pressure dips below this threshold, CBF reduces progressively, leading to symptoms associated with decreased CBF such as nausea, yawning, hyperventilation, clamminess, and fainting. To safeguard brain function, after an initial 20% blood pressure reduction in the first hour, further reductions should occur over the next 2 to 6 hours, aiming for a reading around 160/110 mm Hg, assuming the patient remains stable. If the patient continues to be stable, blood pressure can be further lowered to 140/90 mm Hg over the next 24 to 48 hours. Typically, these blood pressure reductions maintain CBF autoregulation. Several clinical situations may necessitate consideration of different concerns and strategies for lowering blood pressure. In cases of ischemic stroke, immediate blood pressure reduction is generally not advised unless measurements exceed 220/120 mm Hg or the patient is eligible for thrombolytic therapy. Recent research suggests that acute blood pressure lowering offers no advantage (according to the CATIS trial) and may even be detrimental (as indicated by the SCAST trial). For intracerebral hemorrhage (ICH), there is a recommendation to lower systolic blood pressure (SBP) to 140 mm Hg based on limited evidence. The findings from the INTERACT2 study support this guideline, although further reduction to 126 mm Hg did not show additional benefits in the ATACH2 trial. In scenarios of acute aortic dissection, if the patient can handle it, a swift blood pressure reduction to an SBP under 100 mm Hg over 15 to 30 minutes is clinically justified. Additionally, more rapid blood pressure decrease is advised for patients with unstable angina or congestive heart failure accompanied by pulmonary edema.

Exceptions for the quick reduction of blood pressure may apply to elderly patients suffering from carotid stenosis, as these individuals are especially vulnerable to central nervous system hypoperfusion. A considerable drop in blood pressure during an ischemic stroke may not yield positive outcomes. In general, the approach to managing blood pressure in individuals experiencing a stroke or intracranial

hemorrhage is debated, since the disruption of cerebral blood flow autoregulation and the existence of brain swelling necessitate elevated systemic pressures to ensure proper cerebral perfusion.

Conclusion

A hypertensive crisis is a grave health condition marked by a rapid and extreme rise in blood pressure. This phenomenon is important because it has the potential to cause severe complications that can endanger life, such as myocardial infarction, stroke, and damage to organs. It is vital for both patients and medical professionals to comprehend hypertensive crises, as prompt treatment can be lifesaving and help avert serious health issues in the future.

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