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Obstacles in the Treatment and Diagnosis of Hypertensive Emergency Cardiac Abnormalities

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Introduction

Systemic hypertension is the most common noncommunicable disease and the leading preventable cause of death worldwide, accounting for more than 50% of myocardial infarction, heart failure and stroke cases. Since 1990, the global prevalence of systemic hypertension has more than doubled, with low- and middle-income countries (LMICs) accounting for the majority of this increase. In 2010, there were approximately 1.4 billion people worldwide who had systemic hypertension and this figure is expected to rise to 1.6 billion by 2025. LMICS are home to approximately 1.04 billion (75%) of the world's hypertensive population. South Africa has the highest burden of uncontrolled hypertension in Sub-Saharan Africa, with a hypertension prevalence of 35%.

Hypertensive emergency is the most common acute complication of systemic hypertension that results in emergency room visits. Hypertensive emergencies are a diverse group of disorders characterised by acute severe blood pressure (BP) elevation, frequently above 180/120 mmHg, acute hypertension-mediated organ damage and the need for prompt but contextual, system-specific BP lowering to avoid catastrophic outcomes. Acute hypertension-mediated organ damage commonly affects the heart and aorta, as well as the brain, kidneys and retina. The presence of acute hypertension-mediated organ damage in multiple organs at the same time has been demonstrated, implying a common pathophysiologic mechanism across vascular beds.

Description

Patients with severe blood pressure elevation but no evidence of acute hypertension-mediated organ damage are classified as having hypertensive urgency, which, together with hypertensive emergency, constitutes the syndrome of hypertensive crisis. However, the European Society of Cardiology (ESC) Council on Hypertension recently proposed replacing the term hypertensive urgency with "uncontrolled hypertension," making the umbrella term hypertensive crisis (which had previously been used to describe both hypertensive emergency and hypertensive urgency) obsolete [1-3].

In hypertensive emergencies, cardiac complications are the most common acute hypertension-mediated organ damage. Acute heart failure/cardiogenic pulmonary oedema, acute coronary syndrome (ACS), and, less commonly, acute aortic syndrome are the three major cardiac acute hypertension-mediated organ damage syndromes. When compared to patients without hypertensive emergencies, mortality in hypertensive emergencies is significantly higher, particularly among patients admitted to coronary care units. Raised cardiac troponin levels, with or without proven ACS, are one of the prognostic factors for major adverse cardiac events (MACE) and cerebrovascular events in patients with hypertensive emergency.

Despite numerous studies and reports on the cardiac complications of hypertensive emergencies, there are still many unknowns. Various studies report varying prevalence rates, owing to the heterogeneity of the studies and, possibly, selection bias. Across studies, the use of terminology in the classification and reporting of hypertensive emergencies has been inconsistent (e.g., the use of acute heart failure and pulmonary oedema interchangeably in some studies and separately in others). Despite overwhelming evidence of the prognostic significance of elevated cardiac troponin and subclinical cardiac injury, acute myocardial injury is not regarded as acute hypertension-mediated organ damage [4,5]. There is currently no robust system for risk stratification that can quickly identify subgroups at high risk of adverse cardiovascular and renal outcomes and guide future management.

Despite the availability of effective and well-tolerated antihypertensive medications, the incidence of hypertensive emergencies remains unchanged. In their lifetime, an estimated 2-3% of hypertensive patients will experience a hypertensive emergency. Data on gender differences in hypertensive emergency patients have been inconsistent, with some studies showing a male predominance and others showing a similar prevalence in males and females. Similarly, age distribution reports in patients with acute severe hypertension but no acute hypertension-mediated organ damage have been contradictory.

Conclusion

The precise pathophysiologic mechanisms of hypertensive emergency remain unknown. A sudden increase in blood pressure, on the other hand, is a common denominator underlying the various forms of acute hypertension-mediated organ damage and the majority of hypertensive emergencies occur in people who already have hypertension. Although the causes of the rise in blood pressure are unknown, nonadherence to antihypertensive medications, stress and increased salt intake have all been identified as major risk factors. Three intrinsically intertwined processes that work together play an important role in pathophysiology. These include vascular autoregulation failure, endothelial dysfunction and renin angiotensin aldosterone system activation (RAAS).

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