Hypertensive crisis

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Background and definition

Hypertension is a common medical problem. It affects approximately one in four adults worldwide, with evidence that the prevalence is rising. In the USA, approximately 30% of adults have some form of hypertension.[1] It is estimated that 1-2% of the hypertensive population will present with an acute and severe elevation of blood pressure at some stage, i.e. hypertensive crisis: systolic blood pressure > 180 mmHg, or diastolic blood pressure > 120 mmHg.[2]

A hypertensive crisis is further divided into:[3]

- *Hypertensive emergency*: The elevated blood pressure poses an immediate threat to the integrity of the cardiovascular system, and there is end-organ damage or evidence of ongoing end-organ damage, e.g. hypertensive encephalopathy, intracerebral haemorrhage, acute myocardial infarction, acute left ventricular failure with pulmonary oedema, unstable angina (acute coronary syndrome), dissecting aorta aneurysm and eclampsia. Malignant hypertension, an abrupt rise in blood pressure associated with necrotising vasculitis, arteriolar thrombi and myointimal proliferation with fibrinoid necrosis in arterioles of the kidney (deteriorating renal function, proteinuria and haematuria), brain, retina (haemorrhages, exudates, papiloedema) and other organs, including microangiopathic haemolytic anaemia, is now referred to as a hypertensive emergency.[4]
- *Hypertensive urgency*: The elevated blood pressure is not associated with end-organ injury, but patients can complain of a severe headache, dyspnoea, epistaxis and severe anxiety. The majority of these patients are hypertensives, who were either inadequately treated, or who became non-compliant with prescribed therapy.

Common associations with hypertensive crisis

Several blood pressure elevations can develop de novo, or complicate essential hypertension or secondary hypertension. Common causes of a severe elevation in blood pressure can be seen in Table I. The exact pathophysiology and the exact reasons why some hypertensive patients will develop myointimal-proliferation fibrinoid necrosis, and a hypertensive crisis, are unknown.[5] An abrupt rise in blood pressure causes endothelial injury, activation of platelets and coagulation and fibrinoid necrosis. The renin-angiotensin system is often stimulated and contributes to vasoconstriction. Other contributing factors are volume depletion due to pressure natriuresis, and catecholamine excess leading to vasoconstriction. The end result is end-organ hypoperfusion, ischaemia, and organ dysfunction presenting as a hypertensive emergency.[6]

Treatment of hypertensive emergency

These patients must be treated in intensive care with a continuous infusion of a short-acting titratable antihypertensive drug. [6,7] The goal is to reduce blood pressure rapidly (minutes to an hour) by no more than 25%, not to normalise the blood pressure. Then in the next two to six hours, the blood pressure should be reduced to 160/100-110 mmHg. Beware of an excessive fall in blood pressure, as it can precipitate organ ischaemia (renal, cerebral and coronary). Reduction of the blood pressure should be carried out in a controlled manner. Gentle volume expansion with intravenous saline can restore organ perfusion, and prevent an abrupt decline in blood pressure when the parenteral antihypertensives are initiated. Oral therapy can be started as the intravenous agents are slowly titrated down.

Table I: Cause of hypertensive emergencies

Essential hypertension
Renal parenchymal disease Acute glomerulonephritis Vasculitis Haemolytic uraemic syndrome Thrombotic thrombocytopenic purpura
Renovascular disease Renal artery stenosis (atheromatous or fibromuscular dysplasia)
Pregnancy Eclampsia
Endocrine Phaeochromocytoma Cushing's syndrome Renin-secreting tumours Mineralocorticoid hypertension (rarely causes hypertensive emergencies)
Drugs Cocaine, sympathomimetics, erythropoietin, cyclosporine, and antihypertensive withdrawal Interactions with monoamine-oxidase inhibitors (tyramine), and amphetamines, Lead intoxication
Autonomic hyperreactivity Guillain-Barré syndrome, acute intermittent porphyria Central nervous system disorders Head injury, cerebral infarction/haemorrhage, brain tumours

There are exceptions to this rule:

- *Acute ischaemic stroke*: There is no real evidence that immediate antihypertensive therapy is beneficial. In most patients, after an acute stroke, blood pressure falls by itself by ± 28% in the first 24 hours, without therapy.
- *Aortic dissection*: In these patients, the systolic blood pressure should be lowered to below 100 mmHg.
- Patients who urgently need *thrombolytic therapy* must have their blood pressures lowered sufficiently, and it should be lowered to 160/110 mmHg before initiating thrombolytics.

Drugs used for hypertensive emergencies:[7]

- *Sodium nitropruside:* This is an arterial and venous vasodilator. The dose is 0.25-10 µg/kg/minute intravenous infusion, which works within one to two minutes of starting. This drug should only be used when other intravenous agents are not available, due to its severe toxic potential.
- *Nitroglycerine:* This is a potent venodilator, and when given in high doses, affects arterial tone. The dose is 5-100 µg/minute as intravenous infusion.
- *Labetolol:* This is a combined selective alpha-1-adrenergic and non selective beta-adrenergic receptor blocker. Start with a loading dose of 20 mg intravenous as a bonus. The drug works in five to 15 minutes, and the effect lasts for two to four hours. After the bonus dose, give 20-80 mg intravenous bolus every 10 minutes to achieve the desired effect. The effect begins within 5-10 minutes.
- The use of sublingual and oral short-acting nifedipine should be abandoned.

Treatment of hypertensive urgency

Start treatment with two oral agents, with the goal to lower diastolic blood pressure to 100 mmHg over 24-48 hours. Use drugs that block the renin-angiotension-system [angiotensin-converting enzyme (ACE) inhibitors/ angiotensin-receptor blockers (ARB)], or long-acting calcium-channel blockers, and/or diuretics. The goal is to reach 160/80 mmHg over hours to days, with low doses of combination drugs. Aggressive parenteral antihypertensive drugs may cause too rapid lowering of blood pressure, which may be associated with significant morbidity.

Conclusion

Approximately 1% of the hypertensive population will develop severe blood pressure elevations, or a hypertensive crisis. Hypertensive emergencies present with end-organ damage, and should be treated with parenteral drugs. Hypertensive urgencies, without end-organ damage, can be treated with oral agents, usually combinations of drugs. Uncontrolled blood pressure lowering that is too rapid in severe hypertension without end-organ damage is potentially harmful.

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