

Perioperative hypertensive crisis - the anaesthetic implications. A Review of Literature

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Abstract

Hypertensive emergencies involve a series of clinical presentations where uncontrolled blood pressure (BP) leads to progressive end-organ dysfunction affecting the neurological, cardiovascular, renal, or other organ systems. In these situations, the BP should be controlled over minutes to hours. Many causes are involved in severe elevation of blood pressure; inadequate treatment of hypertension, renal diseases, head trauma and pre-eclampsia. Intraoperative hypertension is also common and has many causes. It is usually successfully controlled by anaesthetists. However, there is a lack of agreement concerning treatment plans and appropriate therapeutic goals, making common management protocols difficult. A wide range of pharmacological alternatives are available to control blood pressure and reduce the risk of complications in these patients. This article reviews the perioperative hypertensive crisis and the common strategies used in management. Perioperative hypertension commonly occurs in patients undergoing surgery. Accurate adjustment of treatment and monitoring of patient's response to therapy are essential to safe and effective management of perioperative hypertension.

Keywords: Hypertension, crisis, perioperative, anaesthesia.

Abbreviations: blood pressure (BP), mean arterial pressure (MAP)

Introduction

Hypertension is the most common risk factor for perioperative cardiovascular emergencies. Acute episodes of hypertension may arise due to the aggravation of a pre-existing chronic hypertensive condition or as de novo phenomena¹.

Emergency, anaesthesia, intensive care and surgery are among the clinical settings where proper recognition and management of acute hypertensive episodes is of great importance. Many surgical events may induce sympathetic activity, leading to sudden elevations in BP².

The long term end-organ effects add to patient morbidity and mortality. Ensuring cardiovascular stability and pre-optimization of BP allows safe manipulation of physiology and pharmacology during anaesthesia². Different medications are available for the management of hypertensive emergencies. The greatest challenge is the acute care setting where the need for proper and sustained control of BP exists.

Definition

Acute severe elevations in BP have several terms. The syndrome characterized by a sudden increase in systolic and diastolic BPs (equal to or greater than 180/120 mmHg) associated with acute end-organ damage that requires immediate management otherwise it might be life-threatening was defined as malignant hypertension³. The international blood pressure control

guidelines removed this term and replaced it with hypertensive emergency or crisis⁴.

Criteria for hypertensive emergencies (crises) include: dissecting aortic aneurysm, acute left ventricular failure with pulmonary oedema, acute myocardial ischemia, eclampsia, acute renal failure, symptomatic microangiopathic haemolytic anemia and hypertensive encephalopathy⁵.

While they suggest 'hypertensive urgency' for patients with severe hypertension without acute end-organ damage³. The difference between hypertensive emergencies and urgencies depends on the existence of acute organ damage, rather than the absolute level of blood pressure⁵.

Causes of hypertensive crises

Cessation of antihypertensive medications is one of the main causes. Other common causes are autonomic hyperactivity, collagen-vascular diseases, drug use (stimulants, e.g. amphetamines and cocaine), glomerulonephritis, head trauma, pre-eclampsia and eclampsia, and renovascular hypertension⁶.

Signs and symptoms of hypertensive crisis include severe chest pain, severe headache accompanied by confusion and blurred vision, nausea and vomiting, severe anxiety, shortness of breath, seizures and unresponsiveness.

Pathogenesis

Humoral vasoconstrictors released in the hypertensive crises episodes result in a sudden increase in systemic vascular resistance. Endothelial injury accompanies severe elevations of BP resulting in fibrinoid necrosis of the arterioles with the deposition of platelets and fibrin, and a breakdown of the normal autoregulatory function. The resulting ischemia speeds the further release of vasoactive substances completing a vicious cycle⁷.

Perioperative hypertension

At least 25% of hypertensive patients who undergo noncardiac surgery develop myocardial ischemia associated with the induction of anaesthesia or during the intraoperative or early post-anaesthesia period⁸. Previous history of diastolic hypertension greater than 110 mmHg is a common predictor of perioperative hypertension. The level of risk depends on the severity of hypertension⁹.

Sympathetic activation during the induction of anaesthesia increases the BP by 20 to 30 mmHg and the heart rate by 15 to 20 beats per minute in normotensive individuals⁸. These responses may be more obvious in patients with untreated hypertension in whom the systolic BP can increase by 90 mmHg and heart rate by 40 beats per minute.

Intraoperative hypertension is associated with acute pain induced sympathetic stimulation besides certain types of surgical procedures like carotid surgery, intrathoracic surgery and abdominal aortic surgery. Paix et al, analysed 70 incidents of intraoperative hypertension and reported that drugs were the precipitating cause (inadvertent vasopressor administration by the anaesthetist or surgeon, intravenous adrenaline with local anaesthetic and failure to deliver a volatile agent or nitrous oxide) in 59% of the cases. Light anaesthesia and excessive surgical stimulation represented 21% of incidents, while equipment related causes (ventilation problems e.g. stuck valve, hypoventilation, soda lime exhaustion and endobronchial intubation) were 13% of incidents. Awareness under general anaesthesia, myocardial infarction and pulmonary oedema represented 7% of incidents¹⁰.

In the early postanaesthesia period, hypertension often starts within 10 to 20 minutes after surgery and may persist for 4 hours. Besides pain induced sympathetic stimulation, hypoxia, intravascular volume overload from excessive intraoperative fluid therapy and hypothermia can promote postoperative hypertension. If untreated, patients are at high risk for myocardial ischemia, cerebrovascular accidents and bleeding¹¹. Hypertension might happen 24 to 48 hours postoperative due to fluid mobilisation from the extravascular space, besides cessation of antihypertensive medication in the early postoperative period¹².

The absolute level of BP is as important as the rate of increase. For example, patients with chronic hypertension may tolerate systolic BPs (SBP) of 200 mm Hg without developing hypertensive encephalopathy, while pregnant

women and children may develop encephalopathy with diastolic BPs of 100 mm Hg¹³.

Preoperative general considerations for hypertensive patients

During preoperative assessment we have to review associated medical problems such as ischaemic heart disease, cerebrovascular disease and renal failure. This can assess the risk for anaesthesia and so the hypertensive end-organ damage. Some patients with hypertension are asymptomatic and accidentally discovered during preoperative assessment. Incidental hypertension may suggest long standing hypertensive disease¹. Idiopathic hypertension comprises about ninety percent of hypertensive patients⁶.

Management of perioperative hypertension crises

The treatment plan of perioperative hypertension differs from treatment of chronic hypertension. Hypertensive patients undergoing elective surgery are at risk for increased perioperative hypertensive attacks. Postponement of elective surgery is recommended in chronic hypertensive patients if the diastolic BP is ≥ 110 mm Hg until the BP is controlled¹⁴. We have to determine if it is a hypertensive emergency or urgency, besides the underlying causes of the patient's BP elevation.

The most appropriate medication for management of hypertensive emergency should have a rapid onset of action, a short duration of action, be rapidly titratable, allow for dosage adjustment, have a low incidence of toxicity, be well tolerated and have few contraindications^{2,15}. A parenteral antihypertensive agent is preferred due to rapid onset of action and ease of titration⁵.

The goal of therapy is to halt the vascular damage and reverse the pathological process, not to normalise the BP. Guidelines by the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High BP for treating hypertensive emergencies include starting intervention with reducing systolic BP by 10 to 15%, up to 25% within the first hour. Followed

by gradual reduction of the absolute BP to 160/110 mmHg over the following two to six hours^{5,16}.

Hypertension that occurs with tracheal intubation, surgical incision and emergence from anaesthesia is best treated with short-acting β -blockers, calcium channel blockers, vasodilators, or angiotensin-converting enzyme inhibitors. Postoperative hypertension is best managed by correction of precipitating factors (pain, hypothermia, hypervolemia, hypoxia and hypercarbia)¹⁷.

Unintentional hypotension and associated organ hypoperfusion happens with aggressive attempts to lower BP since the homeostatic mechanisms depend on higher blood pressure for adequate organ perfusion. While inadequate lowering of BP may result in increased morbidity and mortality. However, the alteration between overshooting BP and severe hypotensive states and using vasopressors to get the normotensive levels may

damage end-organs and the vasculature - precise control of BP in a hypertensive crisis is a challenge¹⁸.

Since chronic hypertension shifts cerebral and renal perfusion autoregulation to a higher level, the brain and kidneys are prone to hypoperfusion with rapid decrease in blood pressure. So control of blood pressure to baseline levels should take 24 to 48 hours⁵.

In cases of aortic dissection, the systolic BP should be reduced to less than 120 mmHg within twenty minutes. In ischemic stroke, BP must be lowered to less than 185/110 before administration of thrombolytic therapy¹⁹. Gentle volume expansion with intravenous saline solution will maintain organ perfusion and prevent sudden drop in BP with using antihypertensive medications⁵. Preoperative hypertension is a hypertensive urgency, not an emergency, as it rarely involves end-organ damage with adequate time to reduce the BP¹⁸. Longer acting oral medications such as Labetalol and Clonidine may be more suitable²⁰.

Common antihypertensive medications used in hypertensive crises

Sodium Nitroprusside is a combined venous and arterial vasodilator which decreases both afterload and preload. The onset of action is within seconds and duration of action lasts for one to two minutes, so continuous BP measurement is recommended. If the infusion is stopped, the BP rises immediately and returns to the pretreatment level within one to ten minutes. Prolonged intravenous administration with infusion rates more than 2 mcg/Kg/min may result in cyanide poisoning. Thus, infusion rates greater than 10 mcg/Kg/min should not be continued for prolonged periods²¹.

Labetalol, an alpha- and beta-blocking agent has proven to be beneficial to treat patients with hypertensive emergencies. Labetalol is preferred in patients with acute dissection and patients with end-stage renal disease. The onset of action is five minutes and lasts for four to six hours. The rapid fall in BP results from a decrease in peripheral vascular resistance and a slight fall in cardiac output²². A reasonable administration protocol is to give an initial intravenous bolus of Labetalol 0.25 mg/Kg, followed by boluses (0.5 mg/Kg) every 15 minutes until BP control or a total dose 3.25 mg/Kg. Once an adequate BP level is achieved, we can start oral therapy with gradual weaning from parenteral agents²².

Fenoldopam, a peripheral dopamine-1-receptor agonist, induces peripheral vasodilation; administered by intravenous infusion. Duration of action from 30 to 60 minutes. Gradual decrease in blood pressure to pretreatment values occurs without rebound once the infusion is stopped because of short elimination half-life. A starting dose of 0.1 g/kg/min, titrated by 0.05 to 0.1 g/kg/min up to 1.6 g/kg/min. Fenoldopam provides rapid decline in blood pressure with reflex tachycardia so beware in patients at risk of myocardial ischemia²³.

Clevidipine, a dihydropyridine calcium channel blocker, produces rapid and precise BP reduction. It has a short half-life of about one to two minutes with potent arterial vasodilation without affecting venous capacitance, myocardial contractility or causing reflex tachycardia²⁴. Start intravenous infusion of Clevidipine at 1-2 mg/h; titrate the dose at short intervals (90s) initially by doubling the dose. Systolic pressure decreases by at least 15% from baseline within 6 minutes post-infusion²⁴. A 1-2 mg/h increase in infusion rate produces an additional 2-4 mmHg reduction in SBP¹⁴. Clevidipine is an ideal agent to manage acute severe hypertension moreover safe for patients with hepatic and renal dysfunction².

Rational approach to the management of hypertensive crises

Neurological emergencies

Subarachnoid haemorrhage, acute intracerebral haemorrhage, hypertensive encephalopathy, and acute ischemic stroke require rapid BP reduction. In hypertensive encephalopathy, reduce the mean arterial pressure (MAP) 25% over 8 hours. Labetalol, Nicardipine and Esmolol are the preferred medications; Nitroprusside and Hydralazine should be avoided²⁵.

For acute ischemic stroke, the preferred medications are Labetalol and Nicardipine. The target BP is < 185/110 mm Hg especially if the patient is receiving fibrinolysis²⁵.

In acute intracerebral haemorrhage, Labetalol, Nicardipine and Esmolol are preferred; avoid Nitroprusside and Hydralazine. If signs of increased intracranial pressure (ICP) exist, keep SBP < 180 mm Hg, while maintain SBP < 160 mm Hg in patients without increased ICP for the first 24 hours after onset of symptoms²⁵. Early intensive BP control is recommended to reduce hematoma growth^{26,27}.

In subarachnoid haemorrhage, Nicardipine, Labetalol and Esmolol are also the preferred agents; while Nitroprusside and Hydralazine should be avoided. Maintain the SBP < 160 mm Hg until the aneurysm is treated or cerebral vasospasm happens²⁵.

Cardiovascular emergencies

Rapid BP reduction is also indicated in cardiovascular emergencies such as aortic dissection, acute heart failure, and acute coronary syndrome. Labetalol, Nicardipine, Nitroprusside (with beta-blocker), Esmolol, and Morphine are preferred in aortic dissection. Beta-blockers should be avoided if there is aortic valvular regurgitation or suspected cardiac tamponade. Keep the SBP < 110 mmHg unless signs of end-organ hypoperfusion exists²⁸.

In acute coronary syndrome if the BP is >160/100 mm Hg, Nitroglycerin and beta blockers are used to lower the BP by 20-30% of baseline but, thrombolytics are avoided if the BP is >185/100 mm Hg²⁸. In acute heart failure use intravenous Nitroglycerin and intravenous Enalaprilat. Give vasodilators (besides diuretics) when SBP is 140 mm Hg²⁸.

Cocaine toxicity/Pheochromocytoma

Diazepam, Phentolamine and Nitroglycerin/Nitroprusside are the preferred drugs. In cocaine toxicity, tachycardia and hypertension rarely require specific treatment. Phentolamine is proper for cocaine-associated acute coronary syndromes. In pheochromocytoma, beta blockers can be added after alpha blockade for BP control²⁹.

Pre-eclampsia/eclampsia

The proper medications are Hydralazine, Nifedipine and Labetalol however avoid Nitroprusside, Esmolol and angiotensin-converting enzyme inhibitors. The BP should be <160/110 mm Hg in the antepartum period and during delivery. The BP should be maintained below 150/100 mm Hg if the platelet count is less than 100,000 cells mm³. Intravenous Magnesium Sulphate should also be used to prevent seizures³⁰.

Perioperative hypertension

Nitroprusside, Nitroglycerin and Esmolol are used. Target the perioperative BP to within 20% of the patient's baseline pressure. Perioperative beta blockers are best to use in patients undergoing vascular procedures or at risk of cardiac complications²⁸.

CONCLUSION

Perioperative hypertension commonly occurs in patients undergoing surgery. The permitted value is based on the patient's preoperative BP. It is approximately 10% above that baseline however more reduction in BP may be warranted for patients at high risk of bleeding or with severe cardiac problems. Accurate adjustment of treatment and monitoring of patient's response to therapy are essential to safe and effective management of perioperative hypertension.

Competing Interests

None declared

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