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Review Article

Treatment of Hypertensive Urgencies

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Abstract

Hypertensive urgency can be expressed as a systolic blood pressure of at least 180 mmHg and/or diastolic blood pressure of at least 110 mmHg, without correlated end-organ damage. Elevated blood pressure causes endothelial injury by elevating the endothelial permeability and local activation of the clotting cascade (platelet and fibrin deposition), resulting in fibrinoid necrosis and intimal proliferation. In hypertensive urgencies, blood pressure should be lowered over a period of hrs to days with slower reductions in elderly patients to avoid and elevated risk of cerebral or myocardial ischemia resulting from excessively rapid reduction of blood pressure. Captopril is an angiotensin-converting enzyme inhibitor. Captopril administration is well tolerated and known to be able in decreasing blood pressure in hypertensive urgencies. Its hypotensive effect, when it is taken orally, is evident within 15 to 30 minutes.

Keywords: hypertensive urgencies; treatment

Introduction

Hypertensive urgency is characterized as a blood pressure values that are equal to or greater than grade 2 (systolic blood pressure 160–179 mmHg or diastolic blood pressure 100–109 mmHg), are severe migraine, epistaxis, dyspnea, or severe anxiety [1, 2]. Hypertensive urgency can be expressed as a systolic blood pressure of at least 180 mmHg and/or diastolic blood pressure of at least 110 mmHg, without correlated end-organ damage [3-5]. Patients with Hypertensive urgency perhaps completely asymptomatic or may present with symptoms such as headache, epistaxis, faintness, malaise, psychomotor agitation, nausea, or vomiting [6]. The prevalence of Hypertensive urgency in emergency room or office settings is estimated at 3–5% [7, 8].

Renal (renal artery stenosis, glomerulonephritis); vascular (vasculitis such as hemolytic-uremic syndrome, thrombotic thrombocytopenia purpura); pregnancy related (preeclampsia, eclampsia); pharmacologic (sympathomimetics, clonidine withdrawal, beta-blocker withdrawal, cocaine, amphetamines); endocrine (cushing's syndrome, conn's syndrome, pheochromocytoma, renin-secreting adenomas, thyrotoxicosis); neurologic, central nervous system trauma, intracranial mass); autoimmune (scleroderma renal crisis) are the common cause of hypertensive urgency [9, 10].

Pathophysiology

During the hypertensive episode, there is an abrupt elevate in the systemic vascular resistance owing to humoral vasoconstriction. Elevated blood pressure causes endothelial injury by elevating the endothelial permeability and local activation of the clotting cascade (platelet and fibrin deposition), resulting in fibrinoid necrosis and intimal proliferation. The endothelium is then unable to compensate or auto-regulate for alters in blood pressure. A vicious cycle ensues with further elevates in resistance and endothelial damage. High blood pressure also accelerates the stretch on the vessel wall which activates the renin-angiotensin system. Renin-angiotensin system plays a significant part in severely increased blood pressures. Nitric oxide is released under the influence of endothelial agonists such as acetylcholine, and norepinephrine. Nitric oxide is also released by the endothelium in response to mechanical forces such as shear stress. The combined process of endothelial injury, loss of auto-regulation, activated renin-angiotensin system, reduce in vasodilators (nitric oxide, prostacycline), and sustained blood pressure elevation can lead to tissue ischemia and end-organ damage. Major organ systems included the central nervous, cardiovascular, renal, and gravid uterus. Activation of the renin-angiotensin aldosterone system seems to be significant in the pathophysiology of severe hypertension [11-17].

Management

The objective of hypertensive urgency management is to lower the blood pressure to equal to or less than 160/100 mmHg over several hrs to days, not quickly. Unlike hypertensive emergency, hypertensive urgency can be resolved spontaneously or within 24–48 hrs by using oral or intramuscular medications [18]. The intention of treatment in hypertensive urgency is to slowly lower the blood pressure over a period of 24 hrs using oral antihypertensive agents. The mean arterial blood pressure should not be decreased by more than 25% in the first 24 hrs. Rapid or excessive reductions in blood pressure can have deleterious effects, involving hypotension. A reduction in blood pressure to 160/110

mmHg is all that is required in the first 24 hrs. In hypertensive urgencies, blood pressure should be decreased over a period of hours to days with slower reductions in elderly patients to avoid and elevated risk of cerebral or myocardial ischemia resulting from excessively rapid reduction of blood pressure. Systolic/diastolic blood pressure should often be reduced to less than 160/ less than 100 mmHg and MAP should not be reduced by more than 25 to 30% over the first two to four hr [19—22]. Hypertensive urgencies can be treated with oral antihypertensive agents such as angiotensin converting enzyme inhibitors, calcium-channel antagonists, beta-blockers, alpha-blockers, or a combination of such drugs.

Captopril: Captopril is an angiotensin-converting enzyme inhibitor. Captopril administration is well tolerated and known to be capable in lowering blood pressure in hypertensive urgencies and its hypotensive effect, when it is taken orally, is evident within 15 to 30 minutes. Captopril administration can be repeated after 1 to 2 hrs, in relation to response. Such a drug can be administered also sublingually using a dose of 25 mg and in this case the onset of its action is within 10 to 20 minutes, showing its maximal effect within 1 hr. The side effects of captopril are dry cough; acute renal failure, especially in patients with renal artery stenosis; severe hypotension in hypovolemic patients; cause renal agensia signifies to a congenital absence of one or both kidneys/failure in the fetus sequencing in oligohydraminosis; angioneurotic edema, swelling of the nose, throat, tongue, larynx (caused by prevention of bradykinin metabolism which concentrates in bronchial mucosa); 1st dose outcome (severe hypotension) [23, 24].

Nifedipine: Nifedipine is a dihydropyridine calcium-channel antagonist extensively studied for a rapid control of BP elevation. It determines peripheral vasodilatation with only mild negative inotropic and chronotropic effects. The side effects of nifedipine are flushing, headache, tachycardia, nausea, ankle oedema [25].

Clonidine: Clonidine is a drug that stimulates alpha-2 adrenergic receptors in the brain, resulting in prevention of efferent sympathetic activity and subsequent reduces of systemic vascular resistances (SVR). Its onset of action is within 30-60 minutes after oral administration; the maximal effect being seen within 2-4 hrs. Frequently, a loading dose of 0.1-0.2 mg is followed by 0.1 mg hrly for several hours until a suitable blood pressure level is reached. Abrupt withdrawal perhaps leads to rebound hypertension, dry mouth (xerostomia), bradycardia (in patients with SA nodal abnormality), and withdrawal syndrome upon abrupt discontinuation (elevated BP, headache, tachycardia, apprehension, and tremors [26]

Labetalol: Labetalol is a combined selective alpha-1 and nonselective β-adrenergic receptor blocker with α to β blocking ratio of 1:7. Labetalol is metabolized by the liver to form an inactive glucuronide conjugate. The hypotensive effect of labetalol begins within 2–5 minutes after its intravenous administration, reaching a peak at 5–15 minutes after administration and lasting for about 2–4 hrs. Because of its β-blocking effects, the heart rate is either maintained or slightly decreased. Unlike selective β-adrenergic blocking agents, which reduce cardiac output, labetalol maintains cardiac output [27]. Labetalol decreases the systemic vascular resistance without lowering total peripheral blood flow.

Other oral agents: Other oral agents such as sublingual nitroglycerin, losartan, methyldopa, valsartan, phenoxybenzamine, and direct vasodilators, involving minoxidil and hydralazine, in management of hypertensive urgencies are also effective.

Conclusion

Hypertensive urgency is characterized as a blood pressure values are equal to or greater than grade 2 (systolic blood pressure 160–179 mmHg or diastolic blood pressure 100–109 mmHg), are severe migraine, epistaxis, dyspnea, or severe anxiety. Activation of the renin-angiotensin aldosterone system seems to be significant in the pathophysiology of severe hypertension. The objective of hypertensive urgency treatment is to slowly lower the blood pressure over a period of 24 hrs using oral antihypertensive agents. Captopril is an angiotensin-converting enzyme inhibitor and its administration is well tolerated and known to be able in decreasing blood pressure in hypertensive urgencies. Its hypotensive effect, when it is taken orally, is evident within 15 to 30 minutes.

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