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Case Report

Hypertensive Urgencies and Emergencies: Clinical Update

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Abstract

Hypertensive crisis is a common clinical situation characterized by symptomatic rise of the blood pressure that presents high rate of morbidity and mortality. It is classified in hypertensive emergency and urgency. The clinical picture of hypertensive emergency differs from that of hypertensive urgency by presenting an imminent death risk due to established or developing lesion in target-organs, especially heart, brain, kidneys, and arteries. This condition requires a sensible clinical approach that allows a correct and fast diagnosis of the compromised target-organ. The therapeutic intervention must be immediate, efficient, and individualized for each involved system, in general with anti-hypertensive drugs by endovenous route in an intensive care unit. On the other hand, the patient with hypertensive urgency does not present target-organ lesion nor finds himself/herself in a higher risk of death, therefore the treatment can be carried out with oral anti-hypertensive drugs in the appropriate urgency room. This article reviews the main causes of hypertensive crisis, its physiopathogenesis and epidemiology, as well as its clinical and therapeutic approaching.

Keywords: Hypertensive Crisis; Hypertensive Emergency; Hypertensive Urgency; Epidemiology; Treatment

Introduction

Systemic arterial hypertension (SAH) affects 70 million adults in the United States and about 36 million over 18-year-old Brazilians [1,2]. A hypertensive crisis (HC) is one presentation or complication of SAH; it comprises a wide variety of clinical conditions all of which involve a rapid, inappropriate, intense and symptomatic elevation in blood pressure (BP) that may be associated with risk of rapid deterioration of target organs (heart, brain, kidneys and arteries) and, consequently, immediate or potential risk to life [3-9]. However other conditions that are accompanied by elevations of BP need to be considered as part of the differential diagnosis of HC. These include pseudohypertension, difficult to control hypertension and malignant hypertension.

Classification and Epidemiology

Hypertensive crises may present in two distinct ways in respect to severity and prognosis. The first, hypertensive urgency (HU) is characterized by marked elevations in BP without evidence of target organ lesion (TOL) and without imminent risk to life. This allows slower reductions in BP levels over a period of 24-48 hours, generally using oral medications [3-9]. The other form of presentation, hypertensive emergency (HE), is characterized by marked elevations in BP leading to or associated with involvement and rapid deterioration of target organ function and imminent risk to life. It is a condition that requires rapid but controlled reductions in BP within minutes to a few hours. Usually this approach requires the use of intravenous drugs and monitoring in the intensive care unit [3-9]. In both situations, BP levels are high with the diastolic pressure, usually = 120 mmHg, being taken into account [3-9]. However, in cases of recent onset in non-hypertensive subjects, such as in individuals with acute glomerulopathy (nephritic syndrome) or eclampsia, crises can occur with relatively low diastolic pressures of between 100 and 110 mmHg [4-9]. Therefore, the severity of the condition is not determined by the absolute BP, but the magnitude of its elevation. It is

observed that the numerical definition of HC is conceptual and serves as a parameter for conduct, but should not be used as an absolute criterion. Tables 1 and 2 show the clinical conditions defined as HU and HE, respectively [3-9].

Table 1 – Types of Hypertensive Urgencies (adapted from references 3-6,8-10).

HIPERTENSIVE URGENCIES
Severe Hypertension associated to
Coronary Heart Disease
Heart Failure
Aortic Aneurysm
Severe Epistaxis
Extensive Burns
Hypocoagulable states
Systemic Vasculitis
Peri-operative
Pre-operative in emergency surgery
Intra-operative
Cardiac and vascular surgery, neurosurgery, pheochromocytoma, etc)
Hypertension stage III in the postoperative (organ transplant, cardiac and vascular surgery, neurosurgery, etc)
Mild/Moderate Adrenergic Crises
Rebound syndrome (sudden withdrawal of adrenergic inhibitors)
Drug-food interactions (tyramine vs MAO inhibitors)
Excessive consumption of stimulants (amphetamines, tricyclic, etc.)
In the Pregnancy
Preeclampsia
Hypertension stage III

MAO = Monoamine oxidase

Table 2 – Types of Hyper tensive Emergencies (adapted from references 3-6, 8-10).

HYPERTENSIVE EMERGENCIES
Severe Hypertension associated to acute complications
Cerebrovascular
- Hypertensive encephalopathy
- Intracerebral Hemorrhage
- Subarachnoid Hemorrhage
- Ischemic Stroke
Cardiocirculatory
- Acute Aortic Dissection
- Acute Pulmonary Edema with Left Ventricular Failure
- Acute Myocardial Infarction
- Unstable Angina
Renal
- Rapidly Progressive Renal Failure
Severe Adrenergic Crises
Pheochromocytoma
Illegal Drugs (cocaine, crack, LSD)
Hypertension in the Pregnancy
Eclampsia
Severe Preeclampsia
"HELLP" Syndrome
Severe Hypertension in Final Pregnancy

HELLP = hemolysis, increase of liver enzymes and plaquetopenia

Situations of elevated BP, whether acute or chronic, are very common in the clinical practice and the correct diagnosis of each is crucial for the best therapeutic approach and consequently the clinical outcome. Thus, the differential diagnosis of HC is important. Initially, we would like to highlight the pseudohypertensive crisis which courses to an acute elevation in BP and is common when attending HC cases [4-6,9,10]. The increase in BP, per se, causes great concern to physicians leading them to treat these cases aggressively, with 64.5% of hypertensive patients in the emergency unit being inappropriately treated as HC [11]. In these patients, independent of BP levels, there is no evidence of acute target organ injury or immediate risk to life during the workup of the patient using the usual exams (history, physical examination, fundus examination, biochemistry, electrocardiography, chest radiography and computed tomography). They are often uncontrolled hypertensive patients whether under treatment or not (uncomplicated severe chronic hypertension), who were referred to the hospital emergency room because of very high BP but who were asymptomatic or only mildly symptomatic. Thus, it is important to remember that patient counseling and re-examination are necessary. Another group of hypertensive individuals may have transient elevations of BP after an emotionally painful situation or some discomfort such as headache, vertigo, vascular headache of musculoskeletal origin or manifestations of panic disorder; this condition is defined as a pseudohypertensive crisis. Table 3 shows the diagnostic criteria of pseudohypertensive crises. Another differential diagnosis is represented by severe or difficult to control hypertension [systolic BP (SBP) = 180 mmHg and diastolic BP (DBP) = 110 mmHg] [10,12], characterized by the absence of rapidly evolving TOL, with an indication for gradual reduction and control of the BP. This condition can be confused with HC and pseudohypertensive crises. It is important to stress that the majority of individuals who have severe hypertension are not HE. So there is no proven benefit in the treatment and rapid reduction of the BP in this situation, on the contrary, there is substantial evidence of increased morbidity resulting from immediate and intensive decreases in BP in chronic hypertensive patients [13]. In asymptomatic individuals with severely elevated BP (= 180 x 110 mmHg), the diagnostic screening tests for acute hypertensive TOL revealed no abnormalities indicative of a HE [14]. We see in our day-to-day routine that the medical treatment decision is based on the severity of BP readings, and not on the symptomatology and presence of TOL. Another differential diagnosis is malignant or accelerated hypertension, characterized by severe hypertension, retinal exudates with or without papilledema, fibrinoid necrosis of renal arterioles and acute renal failure; this progresses rapidly to fatal clinical outcomes [15]. After the availability of new generation antihypertensive drugs that are better tolerated and long-acting, the incidence of this presentation has dropped from 7% to 1% [16,17].

Table 3 – Characters of individuals with pseudohypertensive crisis (adapted from references 3-6, 8-10).

1 - Very heterogeneous group.
2 - The diagnosis requires the exclusion of real crisis.
3 - Repeated measurements of BP help in reassessing and confirming the diagnosis.
4 - They are often uncomplicated hypertensive individuals or who stopped antihypertensive drugs + triggering factor.
5 - marked elevation in BP triggered by pain, discomfort, anxiety or treatment dropout.
6 - No signs of lesion in target organs.
7 - There is often agoraphobia or panic disorder.
8 - Assess whether migraine and rotational or emotional episodes can receive immediate symptomatic treatment, while continues the interview and clinical observation.
9 - If necessary, observe for a few hours the reduction of BP with symptomatic, analgesics and / or anxiolytics drugs.
10 - Treatment after diagnosis, only with symptomatic medications and of chronic use.
11 - Reports of severe hypotension in cases of pseudo crisis and use of sublingual nifedipine.

The incidence and prevalence of HC is rarely addressed in the literature. It is estimated that approximately 1% of hypertensive patients develop one or multiple episodes of hypertensive crisis [18]. In our department, HC accounted for 0.59% of all emergency hospital visits over a period of one year and 1.7% of emergency clinical consultations; HU was more common than HE [19]. Ischemic strokes and acute pulmonary edema were the most frequently encountered TOL during HE [19]. These patients had higher mean ages and DBP than patients with HU. Furthermore, individuals with HE were more sedentary and had lower rates of antihypertensive treatment than individuals with HU, which reflects in a high clinical morbidity [20]. In summary, untreated or inadequately treated chronic hypertension without BP control and patients with secondary hypertension have a higher risk of HE.

Pathogenesis of Hypertensive Crises

There is imbalance between cardiac output and peripheral vascular resistance, hence, a disproportionate increase in intravascular volume or peripheral vascular resistance or both may occur, which is reflected on the pressure-natriuresis curve. In general, these principles apply to uncomplicated chronic hypertension and HC. The renin-angiotensin-aldosterone system plays a central role in regulating BP under normal circumstances. In hypertensive crises, acute increases in systemic vascular resistance resulting from vasoconstrictors released into the circulation, course with marked elevations of vascular BP, causing endothelial injury and fibrinoid necrosis of arterioles. Vascular injury leads to platelet and fibrin deposition and impairment of the normal self-regulation of blood flow. The resulting ischemia stimulates the release of vasoactive substances, thus creating a vicious circle [21,22].

Autoregulation of cerebral blood flow

Understanding the self-regulation mechanism of the blood flow to target organs (cerebral, coronary and renal flow) is vital for better antihypertensive management in cases of HE. The autoregulation of cerebral blood flow (CBF) is maintained by the relationship between cerebral perfusion pressure (CPP) and cerebrovascular resistance (CVR), i.e., CBF = CPP / CVR = mean arterial pressure - mean venous pressure / CVR. CPP is the difference between the BP, which helps tissue irrigation, and the pressure return (venous). Under normal CPP, venous pressure is not important, so that the CPP is equivalent to BP. Reductions in CPP can be caused by reductions in systemic arterial pressure (SAP) or increases in intracranial pressure, which increases the venous pressure. Elevations in intracranial pressure (ICP) may occur as a result of arterial or venous occlusive disease or intracerebral hemorrhage. In normotensive individuals, a wide range of variations in BP of between 60-150 mmHg can occur without changing the CBF. An increase in CPP (or SAP) causes an increase in CVR thereby protecting the patient against brain edema, while reductions in CPP cause a drop in CVR thus protecting the patient from tissue ischemia. When the CPP exceeds the upper limit of self-regulation, CBF increases causing cerebral edema. On the other hand, when the CPP drops below the lower limit of self-regulation, the CBF will decrease causing cerebral ischemia [23,24].

In hypertensive patients, this relationship is altered such that the lower limit of self-regulation is higher than in normotensive individuals. Therefore inappropriate decreases in CPP can lead to worse tissue irrigation and consequently worsen the viable ischemic area. For this reason, it is advisable to reduce the mean BP by a maximum of 20 to 25%, as this is close to the lower limit of self-regulation [25]. One should be aware of this situation because most patients with HC are chronically hypertensive with a right deviation from the self-regulatory pressure/flow (cerebral, coronary and renal) curve and do not have acute target organ injury, which is why a sudden reduction in BP may be associated with significant morbidity [25-27].

Approach to Hypertensive Crisis

The controversy to the approach to HC is mainly related to the correct diagnosis, the differentiation between emergency and urgency, the difficulties in evaluating the patient and the selection of appropriate therapy. This is very important as the diagnosis and correct treatment prevent serious injuries that may result from this critical situation. Therefore, the evaluation and diagnosis of HC should be thorough and with objectivity. The approach to patients with HC requires a clinical evaluation and complementary tests performed at clinical emergency centers with hospital support. The following is the sequence of steps used in the management of patients with HC, such that the clinical and complementary investigation obtains the information necessary for the diagnosis and optimal treatment strategy [4,5,9,10]. It is important to investigate:

1. Triggering factors.
2. Symptoms or situations that simulate hypertensive crisis (headache, labyrinthitis, physical trauma, pain, emotional stress, family or professional problems) (Table 3).
3. History of SAH, evolution time, use of antihypertensive drugs (doses and adherence).
4. Previous episodes similar to the current situation.
5. Use of medications that interfere with the BP (anti-inflammatories, steroids, painkillers, antidepressants, appetite moderators).
6. Use or abuse of alcohol and toxics (cocaine, crack, LSD).
7. Sudden suspension adrenergic inhibitors (clonidine and ?-blockers).
8. Association of other diseases and risk factors (diabetes, heart disease, kidney disease, smoking, dyslipidemia).
9. Clinical investigation depending on the system:

- Central nervous system
- Headache, dizziness, visual and speech disturbances, level of consciousness, agitation or apathy, confusion, focal neurologic deficits, seizures, and coma.
- Cardiovascular system
- Chest pain, signs and symptoms of left ventricular failure, palpitations, heart rhythm, gallop rhythm, dyspnea, jugular venous stasis, carotid bruit, peripheral pulses and BP measurement (3 measures).
- Renal system
- Reduced urine flow, edema, hematuria, dysuria. Note: do not forget to examine the abdomen (abdominal pulsatile masses and abdominal bruit).
- Visual system (ocular fundus)
- Vasospasm, arteriovenous crossings, arteries as copper or silver wiring, hard and soft exudates, hemorrhage, papilledema.

Complementary tests (complementary exams are conducted as needed and focused on specific systems after the initial clinical investigation to characterize TOL):

- Central nervous system
- Computed tomography
- Cardiovascular system
- Electrocardiography, chest radiography, echocardiography, cardiac enzymes
- Renal system
- urinalysis, blood urea nitrogen, creatinine, electrolytes

Factors associated with hypertensive crisis

It is important to identify predisposing factors for HC for primary or secondary prevention of cerebral-cardiovascular events. Mean SBP and DBP during 24-hour ambulatory monitoring are predictors of the risk of HC [28,29]. Moreover, reduction or absence of nocturnal decline is associated to increased risk of endothelial dysfunction and a higher risk of TOL, a situation that may trigger acute elevations in BP [16,24,30,31].

Individuals with HU showed longer survival than those with HE during a 5-year follow-up period [19,32]. Age > 50 years, male gender, and smoking are predictors of HU while age > 62 years, SBP > 190 mmHg and DBP > 120 mmHg are predictors of HE [32]. Female gender, higher body mass index, presence of coronary artery disease and hypertensive heart disease, a higher number of antihypertensive drugs and lack of adherence to treatment are also risk factors significantly associated with HC [33]. These findings are important as HC risk markers can define patients who require careful monitoring.

Treatment of Hypertensive Urgency

Treatment should be initiated after a period of clinical observation in a quiet environment with low light conditions which may reduce BP without antihypertensive drugs thereby helping to exclude cases of pseudocrises (treated only with rest, analgesics or tranquilizers) 34. When the use of oral antihypertensive medications (captopril, clonidine and ß-blockers) is required, the BP should be gradually reduced over 24-48 h. [3,4]. Sublingual and oral use of quick-release nifedipine capsules should be proscribed in the treatment of HU, as this approach is neither safe nor effective, as it causes rapid and marked reductions in BP which can result in tissue ischemia [35]. The process of patients discharge from medical observation should follow these steps [10]: a) few hours of observation (4-6 h) after controlling the BP; b) recognition of reversible precipitating causes; c) clinical history of SAH previously controlled with medication that the patient stopped to use; d) initiate or reintroduce the prior treatment based on the guidelines of SAH [36] and e) refer the patient to ambulatory monitoring after a few days. Table 4 shows the major drugs used in the treatment of HU.

Table 4 – Antihypertensive Drugs Used in the Hypertensive Urgency (adapted from references 3-6, 8-10, 40).

Drugs	Administration and dosage	Start	Duration	Adverse Effects
Captopril (ACEI)	25-50 mg oral (repeat in 40-60 min)	15-20 min	4-6 h	Hypotension, acute renal failure (if bilateral renal artery stenosis) Don't use in pregnancy
Atenolol (β-blocker)	50-100 mg oral	6 min	6-8 h	Bradycardia, bronchospasm, AVB
Clonidine (α2 adrenergic agonist)	0,2 mg initial, after 0,1 mg/h until 0,8 mg total	30 min–2 h	6-8 h	Dry mouth, hypotension, rebound effect (chronic use)

ACEI = Angiotensin converting enzyme inhibitor
AVB = Atrioventricular Block

Treatment of Hypertensive Emergency

The treatment of HE should be based on the target organ involved and requires intensive care due to the unstable hemodynamic and neurologic conditions that may be a risk of imminent death. In most cases,

intravenously administered drugs are used. Oral antihypertensive medications should be administered along with the parenteral drugs to facilitate subsequent weaning and to achieve better BP control in less time. The BP should be reduced quickly but controlled (in minutes to hours) to levels up to 25% lower than the initial mean BP or, in practical terms, the DBP should be reduced to 100 to 110 mmHg. After these levels have been reached, further reductions can be slower until eventually reaching normal levels. Excessive drops in BP should be avoided because they may result in renal, cerebral or coronary ischemia. A typical example is found in stroke victims, for whom clinical trials do not recommend immediate use of antihypertensive drugs. Table 5 shows the BP therapeutic targets to be achieved in the treatment of HE. Table 6 presents the drugs of choice and those contraindicated in each type of HE and Table 7 shows parenteral drug use with dose, action, indications and main adverse effects. The treatment of the main clinical situations such as HE are discussed below.

Table 5 – Therapeutic Targets in Special Situations (adapted from references 3-6, 8-10).

Hypertensive Emergency	Therapeutic Targets
	Cardiovascular
APE / AMI	20 to 25% of MBP or DBP = 100 mmHg
Acute Aortic Dissection	SBP = 120 to 100 mmHg if well tolerated
Adrenergic Crisis	SBP = 120 to 100 mmHg if well tolerated
Malignant Hypertension	20 to 25% of MBP or DBP = 110 to 100 mmHg
	Cerebrovascular
Hypertensive Encephalopathy	20 to 25% of MBP or DBP = 100 to 120 mmHg
Intracerebral Hemorrhage	20 to 25% of MBP or DBP = 160 to 140 mmHg
Subarachnoid Hemorrhage	20 to 25% of MBP
Ischemic Stroke	SBP = 185 mmHg/DBP = 110 mmHg

APE = Acute Pulmonary Edema; AMI = Acute Myocardial Infarction; SBP = Systolic Blood Pressure; DBP = Diastolic Blood Pressure; MBP = Mean Blood Pressure

Table 6 – Drugs of choice and those contraindicated in each type of HE (Ref 6).

TYPES OF HE	INDICATED DRUGS	CONTRAINDICATED DRUGS
HYPERTENSIVE ENCEPHALOPATHY	■ SNP ■ Labetalol ■ Diazoxide	■ B-Blockers ■ Methyldopa ■ Clonidine
ISCHEMIC STROKE	■ NONE ■ SNP ■ Labetalol	■ B-Blockers ■ Methyldopa ■ Clonidine
INTRACEREBRAL AND SUBARACHNOID HEMORRHAGE	■ NONE ■ SNP ■ Labetalol	■ B-Blockers ■ Methyldopa ■ Clonidine
MYOCARDIAL ISCHEMIA	■ Nitroglycerine/Nitrates ■ B-Blockers ■ SNP	■ Hydralazine ■ Diazoxide ■ Minoxidil
ACUTE PULMONARY EDEMA	■ SNP ■ Loop Diuretics ■ Morphine ■ Nitroglycerine	■ Hydralazine ■ Diazoxide ■ B-Blockers ■ Labetalol
ACUTE AORTIC DISSECTION	■ SNP and B-Blockers ■ Trimetafan and B-Blockers ■ Labetalol	■ Hydralazine ■ Diazoxide ■ Minoxidil
ECLAMPSIA	■ Hydralazine ■ Methyldopa ■ Labetalol ■ Calcium channel blocker ■ SNP	■ Trimetafan ■ Diuretics ■ B-Blockers
ACUTE RENAL FAILURE	■ SNP ■ Labetalol ■ Calcium channel blocker ■ Dialysis	■ B-Blockers ■ Trimetafan
ADRENERGIC CRISIS	■ Phentolamine or Phenoxybenzamine, after B-Blockers ■ SNP + B-Blockers	
POS-OPERATIVE HYPERTENSION	■ SNP ■ Hydralazine ■ Diazoxide	

HE = Hypertensive Emergency; SNP =Sodium Nitroprusside

Table7 - Pharmacodynamic and pharmacokinetic properties of the antihypertensive drugs to use in hypertensive emergencies (adapted from references 3-6, 8-10, 40).

Drugs	Administration and dosage	Start	Duration	Advantages and indications	Disadvantages
Sodium Nitroprusside (Venous and arterial vasodilator)	Continuous infusion 0,5-10 µg/kg/min	Immediate	1-2 min	Titration	Thiocyanate intoxication, hypotension, nausea, vomiting, muscle spasm
Esmolol (cardioselective β-blocker)	Attack: 500µg/kg Intermittent infusion 25-50 mg/kg/min ↑ 25 mcg/kg/min 10-20 Maximum 300 mcg/kg/min	1-2 min	1-20 min	Acute Aortic Dissection	Nausea, vomiting, 1st degree AV block, bronchospasm, hypotension
Phentolamine (α-adrenergic blocker)	Continuous infusion 1-5mg Maximum 15mg	1-2 min	3-5 min	cathecolamine excess	Reflex tachycardia
Trimetafan (SNS and PSNS blocker)	Continuous infusion 0,5-1,0 mg/min increase 0,5mg/min until maximum 15mg/min	1-5 min	10 min		Tachyphylaxis
Nitroglycerine (Venous and arterial vasodilator)	Continuous infusion 5– 15mg/h	2-5 min	3-5 min	Coronary Perfusion	Headache, variable efficacy, tachyphylaxis
Hydralazine (direct-acting smooth muscle relaxant)	Attack: 10-20 mg EV or 10-40 mg IM, repeat every 4-6 h	10-20min	3-8 h	Eclampsia	Tachycardia, fluid retention, headache, angina, worsening of aortic dissection, nausea, flushing, rash, dizziness
Fenoldopam (D1-like dopamine receptors agonist)	Continuous infusion 0,1-1,6 µg/kg/min	5-10min	10-15 min	Renal Perfusion	Headache, nausea, flushing
Nicardipine (Calcium channel blocker)	Continuous infusion 5-15mg/h	5-10min	1-4 h	Central nervous system protection	Reflex tachycardia, phlebitis, avoid in patients with HF or myocardial ischemia
Labetalol (α and β-blocker)	Attack: 20-80mg 10-10min Continuous infusion 2mg/min maximum 300mg/24h	5-10min	2-6 h	B-blocker Vasodilator	Nausea, vomiting, AVB, bronchospasm, orthostatic hypotension
Enalapril (ACE inhibitor)	Intermittent infusion 1,25-5,0 mg 6/6h	15 min	4-6 h	HF, acute LVF	Hypotension, renal failure
Furosemide (Loop diuretic)	Infusion: 20-80 mg The same dose or an increased dose: 6-8 h later	5-10min	30-90 min	HF, acute LVF	Hypokalemia

SNP = sodium nitroprusside; SNS = sympathetic nervous system; PSNS = parasympathetic nervous system; HF = heart failure; LVF = left ventricular failure; AVB = atrioventricular block; EV = endovenous; IM = intramuscular

1. Ischemic and hemorrhagic stroke

In all cases of stroke, the BP should be lowered slowly and progressively, especially in patients with a history of coronary or cerebral atherosclerosis. We recommend a period of 1-4 h, trying to get the DBP to around 100-115 mmHg or decrease the mean BP by 25% from the baseline levels. These precautions are important in HE because the mechanisms of cerebral vascular self-regulation are altered. Thus, there is a loss of efficiency of circulatory adaptation mechanisms to hypotension and thus cerebral ischemic episodes may arise or worsen [37].

1.1. Ischemic stroke (guidelines 2013) [38]

1.1.2 - SBP >185 mmHg or DBP >110 mmHg:

- Metoprolol 5 mg IV over 2-5 min. Repeat until total dose of 15 mg. Maximum dose 45 mg / day
- Labetalol 10-20 mg IV in 1-2 min. repeat once or
- Nicardipine IV 5 mg/h and titration with addition of 2.5 mg/h every 5-15 min to a maximum dose of 15 mg/h. When desired BP is achieved, reduce to 3 mg/h.

Consider the possibility of using rTPA IV after BP is controlled.

During reperfusion therapy with rTPA, monitor the BP every 15 min for the first 2 h, every 30 min for the next 6 hours and then every hour for 16 h.

If the BP > 185/110 mmHg do not administer rTPA

1.1.2 - SBP >180-230 mmHg or DBP >105-120 mmHg:

- Metoprolol 5 mg IV over 2-5 min. Repeat until total dose of 15 mg. Maximum dose 45 mg/day or
- Labetalol 10 mg IV for 1-2 min + continuous infusion 2-8 mg/min or
- Nicardipine 5 mg/h IV and titration with the addition of 2.5 mg/h every 5-15 min until a maximum dose of 15 mg/h

1.1.3 - If there is no satisfactory control or DBP > 140 mmHg

- Sodium nitroprusside 0.5 mcg/kg/min IV with continuous monitoring of BP

1.2. Hemorrhagic stroke [39]

1.2.1 - SBP > 200 mmHg or DBP > 150 mmHg:

- Aggressive reduction of BP with IV antihypertensive drugs and BP monitoring every 5 min or even continuously

1.2.2 - SBP > 180 mmHg or DBP > 130 mmHg with evidence or suspicion of elevated ICP:

- Monitor ICP, continuous or intermittent IV infusion of antihypertensive drugs with maintenance of cerebral perfusion pressure between 60-80 mmHg.

1.2.3 - SBP > 180 mmHg or DBP > 130 mmHg without elevation of ICP:

- Continuous or intermittent IV infusion of antihypertensive drugs with moderate reduction in BP (DBP = 110 mmHg or BP 160/90 mm Hg), with reevaluations every 15 min

2. Acute Aortic Dissection [4-6,8-10,40]

Target BP: 120 mmHg in 20 min

- ß-blocker (Esmolol / Metoprolol) + sodium nitroprusside
- Metoprolol 5 mg IV over 2-5 min. Repeat until a total dose of 15 mg. Maximum dose 45 mg / day
- Labetalol 10 mg IV 1-2 min. Repeat in 10-20 min until maximum dose of 300 mg

Trimethaphan can be used when there is intolerance to nitroprusside or contraindication to the use of ß-blockers due to chronic obstructive pulmonary disease

3. Acute Lung Edema [4-6,8-10,40]

Target BP: Pressure reduction by 10-15%

- Nitroprusside and/or nitroglycerin 60 µg/min IV
- Loop diuretics
- ACE inhibitor (enalapril)
- Fenoldopam
- Morphine

4. Acute Coronary Syndrome and Arterial Hypertension [4-6,8-10,40]

- Nitroprusside and/or nitroglycerine 60 mg/min IV
- ß-blocker (Esmolol/Metoprolol)
- Nicardipine

5. Hypertensive emergency due to catecholamine excess [4-6,8-10,40]

- Phentolamine and/or associated ß-blocker

6. Preeclampsia and Eclampsia [41]

Preeclampsia is a hypertensive syndrome of pregnancy which occurs after the 20th week and is characterized by increased BP (= 140/90 mmHg) and proteinuria (> 300 mg/24 h) in previously normotensive women. It occurs in 5% to 8% of pregnancies; it is more frequent in primigravidae and represents an important cause of maternal and perinatal mortality in developing countries. HELLP syndrome is a severe variant of preeclampsia characterized by hemolysis, elevated liver enzymes and low platelet count (thrombocytopenia). Eclampsia is

characterized by seizures in gestational hypertensive or pre-eclampsia patients and courses with high maternal mortality.

6.1 Pre-eclampsia/eclampsia associated to chronic arterial hypertension

This should be suspected when microalbuminuria develops (albumin 30-300 mg/24 h urine or 30-300 mg/g albumin/creatinine ratio in chronic isolated urine sample) or when there is an increase in preexisting proteinuria, clinical or laboratory alterations characteristic of pre-eclampsia or increases in pre-existing BP levels after the 20th week of pregnancy in a patient with SAH. Antihypertensive drugs are recommended when the BP is elevated: agonist with central action (methyldopa), oral hydralazine, calcium channel antagonist (prolonged action nifedipine, amlodipine) or pindolol (ß-blocker with intrinsic sympathomimetic activity). Magnesium sulfate is the drug of choice both for treatment and for prevention of seizures. The recommended loading dose is 4-6g IV in infusion for 20-30 min, followed by continuous IV infusion of 1-2 g/h for 24 hours after the seizure or delivery. Recurrent convulsions are treated with the application of another 2g "bolus" IV infusion or by increasing the dose to 1.5 to 2 g/h (if the original dose is 1 g/h). The patient should be monitored in relation to urinary output, patellar reflexes, respiratory rate and oxygen saturation. Plasma magnesium should be maintained between 4-7 mEq/L and should be measured when renal failure is present. Calcium gluconate 1g IV (10 mL of 10% solution) should be administered within 2 minutes in cases of suspected magnesium sulfate poisoning.

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