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Hypertensive emergencies

Emergências hipertensivas

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ABSTRACT

Emergencies and hypertensive crises are clinical situations which may represent more than 25% of all medical emergency care. Considering such high prevalence, physicians should be prepared to correctly identify these crises and differentiate between urgent and emergent hypertension. Approximately 3% of all visits to emergency rooms are due to significant elevation of blood pressure. Across the spectrum of blood systemic arterial pressure, hypertensive emergency is the most critical clinical situation, thus requiring special attention and care. Such patients present with high blood pressure and signs of acute specific target organ damage (such as acute myocardial infarction, unstable angina, acute pulmonary edema, eclampsia, and stroke). Key elements of diagnosis and specific treatment for the different presentations of hypertensive emergency will be reviewed in this article. The MedLine and PubMed databases were searched for pertinent abstracts, using the key words “hypertensive crises” and “hypertensive emergencies”. Additional references were obtained from review articles. Available English language clinical trials, retrospective studies and re-

view articles were identified, reviewed and summarized in a simple and practical way. The hypertensive crisis is a clinical situation characterized by acute elevation of blood pressure followed by clinical signs and symptoms. These signs and symptoms may be mild (headache, dizziness, tinnitus) or severe (dyspnea, chest pain, coma or death). If the patient presents with mild symptoms, but without acute specific target organ damage, diagnosis is hypertensive urgency. However, if severe signs and symptoms and acute specific target organ damage are present, then the patient is experiencing a hypertensive emergency. Some patients arrive at the emergency rooms with high blood pressure, but without any other sign or symptom. In these cases, they usually are not taking their medications correctly. Therefore, this is not a hypertensive crisis, but rather non-controlled chronic hypertension. This type of distinction is important for those working in emergency rooms and intensive care unit. Correct diagnosis must be made to assure the most appropriate treatment.

Keywords: Emergencies; Hypertension/complications; Hypertension/diagnosis; Hypertension/therapy

INTRODUCTION

It is estimated that 3% of all visits to the emergency rooms are due to significant elevation of the arterial pressure (AP). From 1% to 2% of hypertensive patients, at some point of time, present with a condition of increased AP requiring urgent medical care¹. Hypertensive emergency is one of the most severe clinical conditions that merit intensive care and is characterized by a markedly high AP and signs of target organ damage (encephalopathy, acute infarction of

the myocardium, unstable angina, acute pulmonary edema, eclampsia and stroke)^{1,2-7}. Brooks et al., in a retrospective study with 427 patients treated by intravenous antihypertensive therapy for more than 30 min in the emergency ward or intensive care unit (ICU) identified that 57% were considered “excessively managed” and 11% failed in the treatment adopted for the acute stage within 6 hours.⁸ Forty four patients presented with relevant adverse events related to excessive decrease of the mean arterial pressure (MAP).

In prospective studies, Baumann et al., when evaluating prevalence of hypertensive patients in the emergency ward showed that most of them are young, male, smokers, regular alcohol consumers and that 1/3 had no former history of hypertension or is under non-optimal control.⁹

In Brazil, Sobrinho et al., comparing data from public and private hospitals assessed independent predictors for pseudo hypertensive crises.¹⁰ Prevalence was of 48% (95% CI = 39%-58%) and higher in private hospitals (59% *versus* 37%, $p = 0.02$); frequency of incorrect diagnosis was similar between the hospitals (94% *versus* 95%, $p = 0.87$).

DEFINITIONS

Hypertensive crisis is a clinical condition with a sudden elevation of AP ($\geq 180 \times 120$ mmHg), together with symptoms that may be mild (headache, dizziness, tinnitus) or severe (dyspnea, chest pain, coma and even death), with or without acute target organ damage. A hypertensive crisis is defined when symptoms are mild and there is no acute target organ damage. On the other hand, a hypertensive emergency is when symptoms endanger the patient's life, with acute target organ damage.^{2,3,11}

In the pseudo hypertensive crisis, elevation is solely due to physical or psychological stress (i.e. pain). Many patients present with an excessive AP simply because they do not take their medication or because they are unaware bearers of systemic arterial hypertension. That is to say, they have non controlled chronic systemic arterial hypertension (Chart 1 and Figure 1).^{3,7,8,11}

Chart 1 – Findings of the history and physical exam of the different hypertensive emergencies

Hypertensive emergencies	History	Physical Exam	Comments
Acute pulmonary edema	Distressed patient with speech difficulty. Generally already presents some degree of ventricular dysfunction	Apex pulmonary rales. Low oxygen saturation B3 and/or B4 May have stasis of the jugulars (not mandatory)	Sometimes there may be significant wheezing, questioning the differential diagnosis of asthma
Acute coronary syndrome	Pain or feeling of oppressive chest pain. May be with nausea, dyspnea or cold sudoresis	Present B4 In general poor propedeutic findings	Careful characterization of pain is the most important stage of ACS investigation
Acute aortic dissection	Agonizing pain may be precordial or irradiate to the back.	May have asymmetric pulses. May have diastolic murmur in aortic focus	Must be differentiated from ACS
Hypertensive encephalopathy	Lethargy, headache, confusion, vision disorders and seizures, all with an acute or subacute onset	Physical exam may not disclose any finding.	Usually stroke must be excluded by TC Scan
Malignant hypertension	Asthenia, malaise, weight loss, oliguria vague cardiovascular and/or neurological symptoms	Eye papilledema	Potentially fatal, diagnosis as early as possible can only be made by fundoscopy
Ischemic stroke candidate to trombolysis or hemorrhagic	Sudden neurological alteration (usually motor or sensory)	Alteration at neurological exam	Main differential diagnosis is of hypo or hyperglycemia. Attention to sudden headache (subarachnoid hemorrhage)
Eclampsia	Pregnant after the 20 th week of pregnancy or until 6 th week after delivery	Previous diagnosis of preeclampsia and evolves to seizures	

ACS – acute coronary syndrome

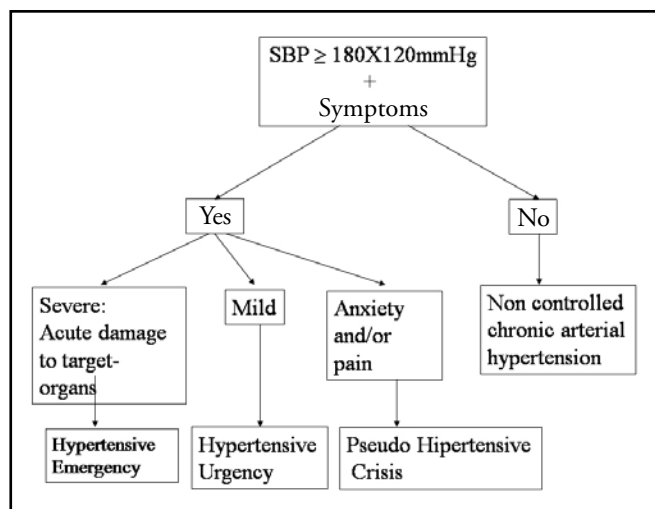


Figure 1 - Differential diagnosis of the arterial pressure elevations.

Semiology of hypertensive emergencies

Propedeutic procedure with hypertensive emergency patients begins with an AP above 180 x 120mmHg, although this pressure level is not absolutely obligatory. Patients with a lesser functional reserve of given organs may present with hypertensive emergency at lower pressure levels. Of fundamental importance is how fast AP increases. Normotensive patients who did not have time to establish self-regulatory mechanisms are more sensitive. AP levels alone do not diagnose an emergency, urgency or pseudocrisis.³⁻⁵

If the patient was previously hypertensive it is important to know about his pressure control, use of antihypertensive drugs, dosage, adherence and when the last tablet was taken. Information about drug consumption (cocaine, amphetamines) or monoxidase (MAO) inhibitors must be queried. Renal impairment may be disclosed by presence of oliguria or hematuria. Measuring of AP must be done with a cuff of appropriate size sphygmomanom-

eter. Physical exam must search for target organ damage by pulse palpation of all limbs, pulmonary auscultation and signs of congestion, heart auscultation for murmurs and gallops and renal artery murmurs. Fundoscopy must always be made in patients with suspicion of a hypertensive crisis.^{2,4,5,12}

“White coat” hypertension is a common and well known occurrence in day care units but forgotten in emergency wards despite the frequency and association to the worst morbidity when present before the hypertensive event.¹³

In intensive care medicine it is fundamental to be aware of pain (even in sedated patients with inadequate analgesia), effect of other agents, rise of catecholamines, postoperative hypertension and hypervolemia. In such patients additional cares are needed to avoid, with rare exceptions (aortic dissection), decrease of MAP over 10% to 20% in the first hours, and afterwards 15% in the two to three subsequent hours, thereby avoiding induction of severe ischemia of noble organs (brain, heart and suprarenals), permitting adjustment of the self-regulatory mechanisms.^{6,8,14,15}

COMPLEMENTARY EXAMS

All patients with hypertensive emergency must undergo the following exams:

complete blood test, serum urea, serum creatinine, electrolytes (sodium, potassium, magnesium), type I urine (search for proteinuria or microscopic hematuria), chest X-ray, electrocardiogram and capillary glycemia.

Comparisons of the last results with former exams may help to determine how acute the specific target organ damage is.⁵⁻⁷

In addition to these general exams, other specific ones will depend upon the type of hypertensive emergency found (Chart 2).

Chart 2 – Specific exams according to each type of hypertensive emergency

Hypertensive emergencies	Specific exam for this emergency
Acute pulmonary edema	Serum BNP (echocardiogram if available)
Acute coronary syndrome	Markers of myocardial necrosis (always), cineangiography (almost always)
Acute aortic dissection	CT scan, transesophageal echocardiogram, angiography
Hypertensive encephalopathy	CT scan to exclude stroke
Malignant hypertension	Nothing in particular
Stroke	Brain CT scan
Eclampsia	Nothing in particular

BNP- B-type natriuretic peptide. CT- computed tomography

TREATMENT

Patients must be admitted and initially cared for in the emergency ward and later transferred to the ICU later.

Patients must be monitored with regard to the electrocardiogram tracing, pulse oxymetry and AP and given some source of oxygen. Venous access must be obtained for administration of vasodilating drugs.

A decrease of about 10% to 20% of MAP is suggested during the first hour⁷. Hypoperfusion may result when AP is abruptly reduced. Invasive monitoring of AP is needed if high doses of intravenous vasodilators are required or when, for any technical reason, noninvasive pressure is not reliable. After six hours of parenteral treatment, oral antihypertensive maintenance therapy must be undertaken. Arterial pressure may be reduced to normal in the next 24 to 48 hours, with gradual decrease of parenteral administration. During the entire treatment patient must remain euvolemic (Chart 3).³⁻⁶

THERAPEUTIC ARMAMENTARIUM

To treat hypertensive emergencies the ideal drug must have a rapid action, quickly reversible and with no side effects, which of course makes it unreal.^{6-9,11} In a system-

atic review Cherney evaluated four studies of hypertensive emergencies and 15 of hypertensive crises to conclude that various drugs were efficient (urapidil, nitroprussiate, nicardipine, lacidipine, nifedipine, enalaprilate and fenoldopam).¹⁶ However, attention was directed to cases of cerebral ischemia related to use of nifedipine. Unfortunately, because of the few randomized studies in this clinical scenario, many questions remain unanswered about the long term follow-up of really beneficial, specific agents in relevant endpoints with a decrease of mortality.

The main parenteral drugs available in Brazil are:

1) Sodium nitroprussiate^{6,9-11,15,17-18} – a vasodilator of immediate action and short duration whose molecule is formed by a ferric core, five cyanate ions and a group of nitric acid. When it interacts with the sulphhydryl groups of blood cells and the vascular wall, cyanate ions and the nitric oxide group are released, the latter acting as a direct vasodilator. It is the most effective parenteral drug for hypertensive emergencies. It has greater action on the arterial system than on the venous system. It acts very quickly (in seconds) and its action lasts for 1 to 2 minutes, with a plasmatic life of 3 to 4 minutes. Therefore, abrupt cessation of the infusion will cause an almost immediate elevation of the pressure.

Despite its ideal pharmacodynamic and pharmacoki-

Chart 3 – Treatment of the pressure levels for each type of hypertensive emergency

Hypertensive emergencies	Treatment of pressure levels (in chronological order of administration)
Acute pulmonary edema	Sublingual nitrate Intravenous furosemide Intravenous morphine Intravenous sodium nitroprussiate, (if SBP > 180 mmHg) or nitroglycerin (if SBP < 180 mmHg)
Acute coronary syndrome (ACS)	Sublingual Nitrate Intravenous morphine Intravenous beta-blocker Intravenous sodium nitroprussiate, (if SBP > 180 mmHg) or nitroglycerin (if SBP < 180 mmHg)
Acute aortic dissection	Intravenous beta-blocker Intravenous morphine Intravenous sodium nitroprussiate
Hypertensive encephalopathy	Intravenous sodium nitroprussiate
Malignant hypertension	Intravenous sodium nitroprussiate
Ischemic stroke candidate to thrombolysis or hemorrhagic	Intravenous sodium nitroprussiate Add oral nimodipine if subarachnoid hemorrhage
Eclampsia	Intravenous hydralazine
Excess of catecholamines	Intravenous alpha-blocker (fentolamine) Benzodiazepine Intravenous beta-blocker

SBP- systolic blood pressure

netic profile, nitroprussiate has restrictions, mainly when used for an extended period (> 24-48 hours) and/or in high doses (> 2 µg/kg/min). Particularly, in patients with renal or hepatic dysfunction it may lead to cyanate intoxication that can manifest itself as metabolic acidosis, mental disturbance, such as encephalopathy, headache and unexplained cardiac arrest. If doses between 4 and 10 µg/kg/min are being used, an infusion of thiosulphate may be administered to prevent cyanate deposit, as well as continued infusion of hydroxycobalamine (safe and efficient in the prevention and treatment of cyanate toxicity associated with nitroprussiate). Thiocyanate intoxication (originated by cyanate when metabolized in the liver) is also possible in such conditions although less severe. In patients with coronary disease, significant reduction of the afterload may reduce coronary arterial flow. In a randomized placebo-controlled clinical trial, nitroprussiate increases mortality in 13 weeks (24.2% versus 12.7%) when administered in the first hours after acute infarction of the myocardium.²⁻³

When given, it must be protected from light, because it is photosensitive. Initially 0.25 µg/kg/min may be given and then increased every 2 minutes until the desired AP is reached. In practice, an ampoule of sodium nitroprussiate (50 mg) is diluted in 250mL of saline solution, bottle and line must be protected from light and, by means of an infusion pump, it is administered at 3 mL/h. AP must then be measured every two minutes and each time, if still above the desired level, 1 or 2 mL/h should be increased. When the desired AP is reached, an oral antihypertensive may be given, preferably with a short mean-life to begin the strategy of reducing infusion speed, ideally with an invasive AP or, in its absence, using a noninvasive monitor of AP, with measurement every 10 minutes.

2) Nitroglycerin¹⁷⁻²⁰ - interacts with the nitrate receptors of the vessels' smooth muscles. It must be administered in glass or polyethylene flasks and conveyed by polyethylene equipment. It has a much more powerful action in venous dilation than in arterial dilation. Sometimes its arterial action is insufficient to satisfactorily reduce AP. Furthermore it has the capacity to dilate epicardial coronary vessels, one of the reasons for its use as anti-angina agent. Possible side effects are: headache, vomit, tachycardia and hypotension. Rarely, it may cause bradycardia or methemoglobinemia. It is contraindicated in cases of right ventricular infarction and in patients who have used sildenafil in the last 24 hours. Patients may develop tolerance if it is used for long periods. Peak action occurs in 2 to 5 minutes and effect lasts for 5 to 10 minutes. Initial dose is of 5 µg/min and may be increased every 5 to 10 minutes if the target-AP

has not been attained. In practice, 50 mg may be diluted in 250mL of saline solution 0.9% taking care of the bag and line. Administration is intravenous with 3mL/h in an infusion pump and increased 2mL/h every 5 minutes, until the desired AP is reached or a side effect occurs (headache is the most frequent).

3) Beta-blockers^{7-10,15} - intravenous metoprolol and propranolol are the most often available in Brazil. They are used when a decrease of the heart rate is the major concern even more so than a decrease of AP. They are contraindicated in patients with decompensated ventricular failure, bearers of decompensated chronic obstructive pulmonary disease (COPD) or asthma, severe peripheral vasculopathy and atrioventricular blocks. Metoprolol is more beta-1 selective than propranolol. Both must be administered intravenously by a slow bolus, without any dilution and can be repeated up to 3 times or until the target heart rate is met. If beta-blockers cannot be used because of some contraindication, a venous calcium antagonist that has a negative chronotropic effect such as verapamil or diltiazem (the latter has the advantage of being less inotropic-negative) may be used. These calcium antagonists are contraindicated in presence of atrioventricular blocks and relatively contraindicated in presence of ventricular failure.

4) Hydralazine^{4,13,14} - is a direct arteriolar vasodilator with quick onset of action (5 to 15 minutes) and prolonged duration (2 to 6 hours). It is metabolized by the liver and eliminated in the urine; therefore doses must be reduced in cases of renal or hepatic diseases. It may cause reflex tachycardia contraindicating its use in acute coronary syndromes and in acute aortic dissection. Usually, it is used in hypertensive emergencies for pregnant women because of its proven safety for the fetus. Side effects are that it may induce rheumatological or immunological diseases and its interruption is usually curative.

THERAPY ACCORDING TO EACH TYPE OF HYPERTENSIVE EMERGENCY

Acute pulmonary edema

Acute hypertensive pulmonary edema must be immediately managed with sublingual nitroglycerin, furosemide and intravenous morphine. The patient must be sitting, preferentially with the legs dangling out of the gurney. Use of noninvasive mechanical ventilation has proven to be one of the most important measures to resolve the symptoms. The antihypertensive to be used is, in general, nitroprussiate.^{5,6,15-19}

Intravenous nitroglycerin is preferred in the case of heart disease or when AP is not too high (arbitrarily a sys-

tolic pressure of less than 180mmHg may serve as criterion). For bearers of mitral stenosis with a good ventricular function, an intravenous beta-blocker may be used.

Acute aortic dissection

A potent analgesia must be achieved with morphine and the heart rate must be reduced to about 60 bpm with intravenous metoprolol. In dissection it is more important to reduce heart rate than to reduce AP. Once both actions have been taken, AP may eventually reach sufficiently low levels to avoid additional administration of intravenous antihypertensive. Acute aortic dissection is one of the exceptions where AP must be reduced to lower levels, with in general, a systolic AP of about 100 to 110 mmHg in the first 20 minutes of treatment. Therefore it is often necessary to use another vasodilator which is usually sodium nitroprussiate. If there is coronary impairment in aortic dissection, intravenous nitroglycerin will be preferred.^{5,6,15-19}

Acute coronary syndrome (ACS)

Initial pressure control is done with sublingual nitrate, followed by intravenous morphine if pain does not abate. After achieving venous access a beta-blocker must be administered until the heart rate is reduced. Then nitroglycerin is given continuously at a titrated speed to reduce AP by 30%. A very abrupt reduction must be avoided as it may reduce coronary perfusion. Furthermore, this chapter does not intend to approach details on how to manage ACS, but only to show the armamentarium available for pressure control in these cases. Angiotensin converter enzyme (ACE) inhibitors may be a significant drug for these patients, even if they become normotensive as the action is favorable on the processes of ventricular remodeling.^{5,6,15-19}

However, among the antihypertensives discussed here, this is the last to be administered, because its benefits are over the long term and the drugs already described are more important for acute control of the painful condition. That is why it must be administered only after the clinical condition is stable and at least 6 hours after onset of the acute event.

Hypertensive encephalopathy

With sudden elevation of AP, the upper threshold of the self-regulatory capacity of the cerebral blood flow may be surpassed with consequent hyper cerebral perfusion leading to an endothelial dysfunction, rupture of the hematoencephalic barrier, cerebral edema and micro hemorrhages. The clinical outcomes are hypertensive encephal-

opathy characterized by acute or subacute appearance of some symptoms such as lethargy, headache, confusion, vision disorders and seizures. Usually, these patients are not hypertensive before the event. A computed tomography scan of the brain will always be required to exclude other neurological affections, mainly stroke. AP must be reduced by some 20% and the drug chosen is sodium nitroprussiate.^{5-6,15-19}

Malignant hypertension

Manifests itself by neuroretinopathy and acute or subacute renal failure. The patient in general exhibits asthenia, malaise, weight loss and cardiovascular or neurological symptoms. If not properly treated, mortality is of approximately 90% in one year. Renal failure may range from previously inexistent proteinuria to overt acute renal failure. Traditionally, retinopathy manifests itself by papilledema at the eye, corresponding to a stage IV retinopathy in the Keith Wagener classification. Treatment is with sodium nitroprussiate until a 20% decrease of AP in 2 hours, followed by gradual pressure control in 2 or 3 days with oral medication. There are authors who believe that patients with mild symptoms, papilledema and slight loss of renal function may be managed as non-emergency hypertensive crises, that is to say, with no intravenous administration of antihypertensives.^{2,7,14-18}

Stroke

Often an elevated AP is found during a stroke therefore raising the frequent doubt if AP is the cause or the consequence of the cerebral event. Most patients do not require any treatment for pressure control because once pain, anxiety and distress are controlled the AP tends to remain near normal values. Hypertension at this acute stage may be a beneficial effect to protect cerebral perfusion in the penumbra area. However, under certain conditions treatment is necessary. According to recommendations standardized by the American Heart Association, with slight adaptations, use of nitroprussiate is suggested when AP >180 x 105 mmHg in patients with hemorrhagic or ischemic stroke, candidates to thrombolysis. Patients with ischemic stroke, excluding those that meet the criteria for thrombolysis, in general must be treated as hypertensive crisis, initially providing relief from pain and anxiety. If both factors are controlled and the patient continues to present AP > 22 x 120 mmHg, nitroprussiate may be used to reduce AP by 10% to 20% in 24 hours. Arterial pressure, usually, drops spontaneously to the levels prior to ischemic stroke in 4 days, without any antihypertensive treatment. In subarachnoid

hemorrhage, the same procedures suggested for hemorrhagic stroke are pertinent, however nimodipine must be the first agent of the therapeutic armamentarium for this condition, as it reduces risk of cerebral infarction associated to vasospasm. For any stroke condition, the neurological worsening associated to a decrease of AP must be treated by reducing or even interrupting nitroprussiate administration.¹⁵⁻²⁰

Eclampsia

It is defined as appearance of proteinuria and arterial hypertension after the 20th week of pregnancy and until the 6th week after delivery. Generally it is accompanied by edemas. Five percent of pregnant women bearers of preeclampsia evolve to seizures, that characterize eclampsia. Although there are various control measures for this disease and to prevent the evolution, only delivery with removal of the placenta will reverse the entire physiopathology involved in this morbid process. That is why, whenever preeclampsia appears after the 36th week of pregnancy, the conduct is to anticipate delivery. If it is a case of severe preeclampsia or eclampsia, fetal maturity must be verified by amniocentesis. Experienced physicians must then decide upon delivery or follow a strictly drug approach while fetal maturity is provoked with corticoids. In eclampsia, the agent chosen for pressure control is hydralazine as it does not damage the fetus. The use of sodium nitroprussiate is only authorized when delivery is imminent and pressure control is not achieved with intravenous hydralazine. The goal should be to keep systolic AP between 140 and 160 mmHg and diastolic AP between 90 and 105 mmHg.¹⁵⁻¹⁸

Hypertensive emergencies caused by excess of catecholamines

True hypertensive emergencies caused by excess of catecholamines are rare. The main causes are pheochromocytoma, users of MAO inhibitors that ingest food containing tyramine, cocaine or amphetamine users or sudden interruption of antihypertensives such as clonidine and beta-blockers (easily manipulated by restarting treatment). Occasionally pheochromocytomas present with the typical triad of headache, sudoresis and severe hypertension. Treatment of hypertensive emergencies caused by pheochromocytoma or cocaine begins with an intravenous alfa-blocker (fentolamine). Benzodiazepine and a beta-blocker may next be added, however must be avoided initially to prevent unopposed alpha-adrenergic activity, with potential elevation of the arterial pressure (Table 1).^{3,4,9,13-15}

CONCLUSION

Hypertensive emergency consists of a syndrome where significant elevation of the systemic arterial pressure leads to acute target-organ damage, threatening life. Energetic measures must be taken for immediate treatment, initially with intravenous administration of drugs to reduce arterial pressure. Sodium nitroprussiate is the most often used drug, but in some cases it may not be the best. To recognize the disease in question and know how to properly treat it may be the difference between life and death in a few hours or minutes.

RESUMO

As urgências e as emergências hipertensivas são ocorrências clínicas que podem representar mais de 25% dos atendimentos a urgências médicas. O médico deverá estar habilitado a diferenciá-las, pois o prognóstico e o tratamento são distintos. Estima-se que 3% de todas as visitas às salas de emergência decorrem de elevações significativas da pressão arterial. Nos quadros relacionados a estes atendimentos, a emergência hipertensiva é a entidade clínica mais grave que merece cuidados intensivos. É caracterizada por pressão arterial marcadamente elevada e sinais de lesões de órgãos-alvo (encefalopatia, infarto agudo do miocárdio, angina instável, edema agudo de pulmão, eclâmpsia, acidente vascular encefálico). O objetivo deste estudo foi apresentar os principais pontos sobre o seu apropriado diagnóstico e tratamento. Foi realizada busca por artigos originais com os unitermos “crise hipertensiva” e “emergência hipertensiva” nas bases de dados Pubmed e MedLine nos últimos dez anos. As referências disponíveis destes artigos foram verificadas. Os artigos foram identificados e revisados e o presente estudo condensa os principais resultados descritos. Para esta revisão foram considerados ensaios clínicos em língua inglesa, estudos retrospectivos e artigos de revisão. A crise hipertensiva é a entidade clínica com aumento súbito da PA ($\geq 180 \times 120$ mmHg), acompanhada por sintomas, que podem ser leves (cefaléia, tontura, zumbido) ou graves (dispnéia, dor precordial, coma e até morte), com ou sem lesão aguda de órgãos-alvo. Se os sintomas forem leves e sem lesão aguda de órgãos-alvos, define-se a urgência hipertensiva. Se o quadro clínico apresentar risco de vida e refletir lesão aguda de órgãos-alvo tem-se, então, a emergência hipertensiva. Muitos pacientes também apresentam uma PA elevada demais, por não usarem suas medicações, tratando-se apenas de hipertensão arterial sistêmica crônica não controlada. Este conhecimento deve ser rotineiro ao emergencista e intensivista no momento de decidir sobre a conduta.

Descritores: Emergências; Hipertensão/complicações; Hipertensão/diagnóstico; Hipertensão/terapia

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