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Review Article

The diagnostic approach and management of hypertension in the emergency department

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ABSTRACT

Hypertension urgency and emergency represents a challenging condition in which clinicians should determine the assessment and/or treatment of these patients. Whether the elevation of blood pressure (BP) levels is temporary, in need of treatment, or reflects a chronic hypertensive state is not always easy to unravel. Unfortunately, current guidelines provide few recommendations concerning the diagnostic approach and treatment of emergency department patients presenting with severe hypertension. Target organ damage determines: the timeframe in which BP should be lowered, target BP levels as well as the drug of choice to use. It's important to distinguish hypertensive emergency from hypertensive urgency, usually a benign condition that requires more likely an outpatient visit and treatment.

1. Introduction

Cardiovascular disease is the leading cause of death worldwide, and hypertension is the main cardiovascular risk factor. Despite the increased use of antihypertensive medications, the prevalence of hypertension has remained constant or has decreased only slightly over the past four decades, with an increase in low- and middle-income countries [1]. In 2010, 31.1 % of adults (1.39 billion) worldwide had hypertension, and among adults, this condition affected 31.5 % of the population (1.04 billion people) in low income countries, and 28.5 %, (349 million people) in the high income ones [2]. Hypertension becomes progressively more common with advancing age, with a prevalence of up to >60 % in people aged >60 years [1].

One of the problems in the management of hypertension is the proper diagnostic approach and treatment of the huge number of patients that come to the emergency department (ED) because of high BP levels. It is estimated that 145 million visits take place in the ED in the USA. The prevalence of elevated BP is close to 45 % [3], and close to 4,5 % of the ED visits are given intravenous treatment for an elevated BP [4]. Whether this elevation is temporary and in need of treatment or reflects a chronic hypertensive state is not always easy to unravel. Existing guidelines provide few recommendations concerning the diagnostic approach and treatment of ED patients presenting with high BP levels. In addition, questions exist regarding how much stress or pain may contribute to BP elevation in the ED, when to start drug treatment, how aggressive treatment should be, and finally, for those not hospitalized, whether they need outpatient follow-up. To answer these questions, it is required to have an in-depth, individualized clinical understanding of any individual patient. However it seems that this number is very low, accounting for less than 2 % of patients with high BP [5]. Is this a BP elevation in a patient with long standing hypertension? Is there organ damage, and if so, is it due to high BP? Is this an emergency situation, critical for his/her life, and do we need aggressive treatment? It is important to remember that many patients presented in the ED with acute pain or stress, may have an acute BP increase due to pain, stress, or the "white coat" effect, and that very often it is impossible to follow the guidelines for blood pressure measurement in an ED. However, some studies have shown that there is no close relation between pain or stress and BP in the ED [6]. Severe asymptomatic hypertension may occur in previously hypertensive patients, and is mainly due to noncompliance in

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a percentage that goes up to 65 % or, in rare cases, to unrecognized secondary hypertension [7]. BP measurements in the ED are often taken incorrectly without following the ESC/ESH hypertension guidelines [8] leading to abnormally high BP readings and unnecessary drug treatment. If the BP is severely elevated (Grade III), it is important to allow the patient to rest because data have shown that in 30 % of patients the BP goes down to Grade II or lower in 30 min [9]. Data have shown that BP remains high after ED discharge and even after decreasing, does not reach normal levels [10]. So they need close follow-up with clinic, home, and even ABPM monitoring. The aim of this review is to help answer some important questions that doctors face when confronted with ED patients because of BP elevation.

2. Causes and diagnostic approach

Hypertension emergencies are conditions in which a BP elevation is associated with acute left ventricular failure, acute aortic dissection, acute coronary syndromes, hypertensive encephalopathy, acute ischemic stroke or intracerebral hemorrhage, pheochromocytoma and other endocrine conditions. Hypertension that originates from intense pain and/or anxiety due to medical or surgical conditions can also be an emergency, and this is also the case for hypertension due to medications, medication withdrawal, or eclampsia. Sometimes essential hypertension, renovascular disease, renoparechymal disease, or more rare entities (brain tumors, autonomic hyperreactivity) are the only identifiable underlying conditions [11–14].

Pathophysiologic approach in the following categories [4,12]:

a) Malignant hypertension with or without thrombotic microangiopathy and/or acute kidney failure. This condition is characterized by small artery fibrinoid necrosis in the kidney, retina, and brain. There might be associated fundoscopic changes (haemorrhages – papilloedema), microangiopathy, disseminated intravascular coagulation, and even encephalopathy and/or acute heart failure.
b) Severe hypertension associated with conditions that will need aggressive BP management: Aortic aneurysm or dissection, acute heart failure, acute myocardial infarction, acute stroke (hemorrhagic or thromboembolic). In this case, the emergency is compatible with a relatively modest BP increase, which is sufficient to precipitate organ failure.
c) Hypertension caused by phaeochromocytoma or exogenous sympathomimetics substances (e.g.: Substance abuse)
d) Eclampsia/ severe preeclampsia with HELLP (hemolysis elevated liver enzymes, low platelets) syndrome

The assessment of hypertensive emergencies is illustrated in Fig. 1.

3. Aortic dissection

Aortic dissection (AD) represents a severe, life threatening condition characterized by the disruption of the media that leads to the separation of the aortic layers. The most common risk factor in AD patients is arterial hypertension (65–75 % of AD patients), and usually BP levels are poorly controlled [15]. Usually, these patients present to the ED with

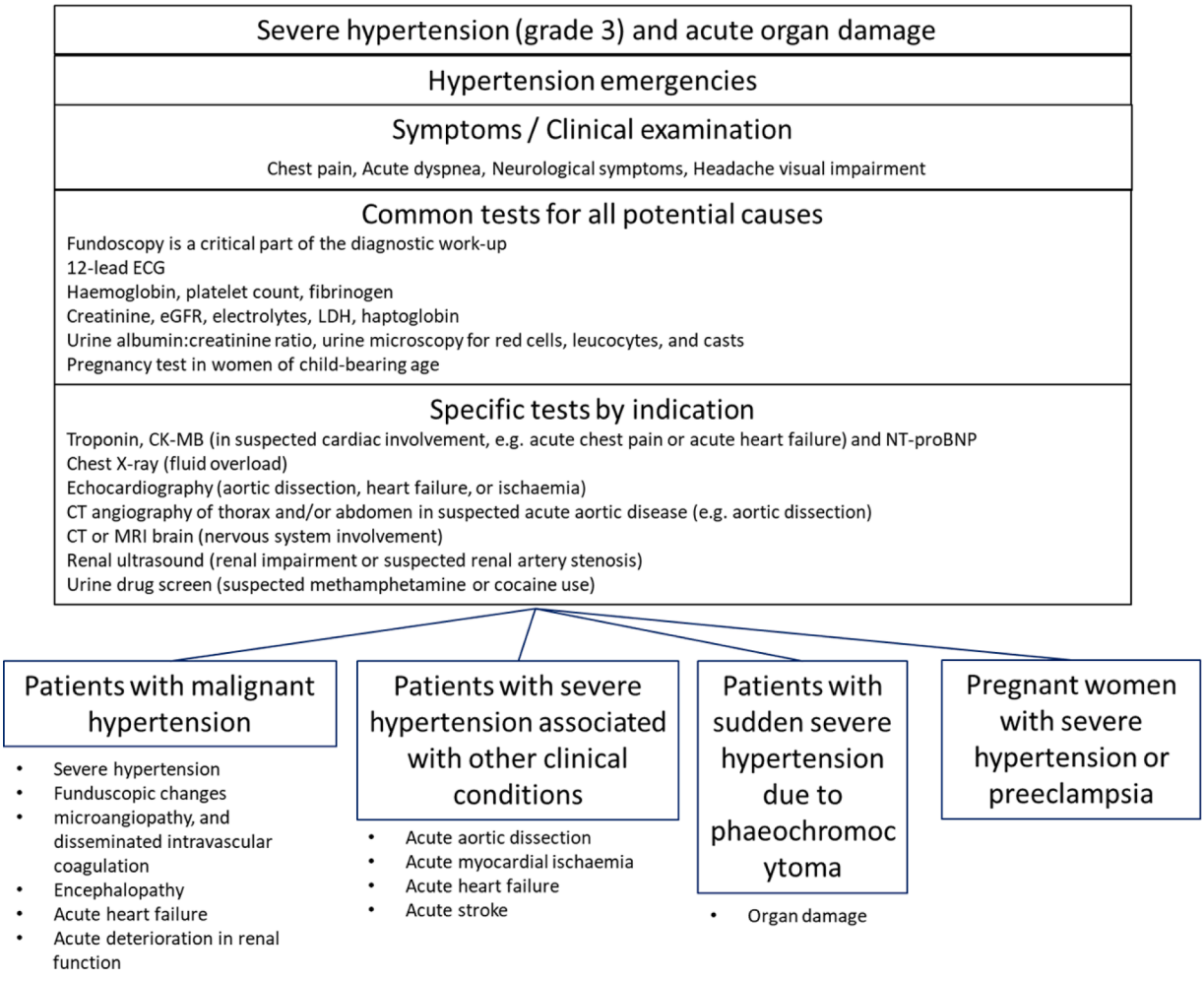

Fig. 1. Algorithm for the assessment of hypertensive emergencies.

pain in the chest (80 %), in the buck (40 %) or abdomen (25 %), while myocardial infarction, acute heart failure, or neurological symptoms frequently coexist [15]. Whether or not the patient undergoes any intervention, medical treatment is essential to maintain a proper hemodynamic status and to control pain. The preferred analgesic that also reduces the sympathetic drive is morphine administered iv. At the same time, physicians should target on decreasing the aortic wall tension in order to limit the extent of the dissection. Beta blockers (the use of short half-life agents is preferred) administered iv represent the treatment of choice in these patients although there are no randomized controlled trials in patients with acute AD. Beta blockers can reduce both BP and heart rate (HR) levels, targeting on 100–120 mmHg for systolic BP levels and approximately 60 bpm for HR [15]. In case of severe aortic regurgitation however HR levels may be higher in order to maintain a certain grade of reflex tachycardia that decreases intracavitary ventricular pressures. Thus, iv esmolol, propranolol, metoprolol, or labetalol are preferred, and in case of beta blocker contraindication, non-dihydropyridine calcium channel blockers iv can be used. If BP levels remain uncontrolled, in addition to beta blockade, vasodilators may be added, and the use of iv nitroprusside, nicardipine, nitroglycerin and fenoldopam may represent a reasonable option [16–18].

4. Acute heart failure

A significant proportion of patients with acute heart failure (HF) present elevated BP levels (> 140/90 mmHg), since only 5–8 % of these patients present SBP < 90 mmHg [19]. The excessive increase in BP levels may lead to acute pulmonary oedema and prompt BP reduction is considered the primary therapeutic target. Thus, the use of iv loop diuretics (in order to reduce congestion) and iv vasodilators (nitroglycerine, isosorbide dinitrate, nitroprusside, nesiritide) in order to reduce preload and afterload is recommended [19]. Moreover, non-invasive positive pressure ventilation (CPAP, BiPAP) should be considered in patients with respiratory distress (respiratory rate >25 breaths/min, SpO2 <90 %) since it not only improves arterial oxygen saturation, but decrease also preload and acts as pulmonary decongestion because of the positive pressure ventilation [19]. In addition, the decrease in preload and the improvement of in respiratory distress will also decrease BP levels. The clinical presentation of a patient with an excessive rise of BP levels is usually warm and wet or warm and dry, suggesting that the patient may present increased peripheral vascular resistance and BP levels and/ or fluid overload. In patients with acute pulmonary oedema BP levels should be decreased initially by approximately 25 % (first hours) and cautiously thereafter.

5. Acute myocardial infarction

The decrease of BP levels in patients with acute myocardial infarction decreases myocardial work and myocardial wall stress, decreasing myocardial oxygen consumption. The use of iv beta blockers in the acute setting (preferably with a short half-life and not in patients with signs of acute HF) decreases HR and BP levels and is recommended by current guidelines [20]. Likewise, the use of iv nitrates decreases pre and after load while improves coronary perfusion [20]. Moreover, the decrease of myocardial oxygen consumption also improves angina symptoms. BP should be decreased with caution however since BP levels < 120/70 mmHg could trigger the j curve phenomenon [21–25].

6. Substance abuse acute coronary symptoms

The use of sympathomimetic substances such as cocaine or amphetamine may lead to rupture of a vulnerable atherosclerotic plaque and/ or coronary spasm, with subsequent development of an acute coronary syndrome (ACS). In addition, these agents increase HR and BP levels, increasing myocardial oxygen demand. Unfortunately, there are no randomized controlled trials regarding therapies that improve outcomes in patients with sympathomimetic substances associated with ACS. Thus, recommendations are mainly based on animal, observational, or cardiac catheterization studies or case reports. In these patients, the use of benzodiazepines relieves chest pain while improving hemodynamics and neuropsychiatric symptoms [26]. In cases of persistent hypertension, iv vasodilators such as nitrates and nitroprusside are preferred. The use of beta blockers should be avoided since they may intensify coronary vasoconstriction.

7. Central nervous system

Blood pressure is frequently found elevated in patients with intracerebral haemorrhage (ICH) or acute ischemic stroke, while hypertensive encephalopathy represents a serious hypertensive emergency. The management of BP in such cases remains poorly clarified, since available evidence is limited, and thus current recommendations are largely based on expert opinion and clinical wisdom. From the clinical point of view three main issues have to be answered: when to initiate therapy, which is the blood pressure goal, and how it will be achieved.

8. Intracerebral haemorrhage

Several studies evaluated the management of elevated BP levels in Intracerebral haemorrhage (ICH) patients. SBP levels >180 mmHg were associated with hematoma growth in a small Spanish study of 117 patients with ICH [27]. In the SAMURAI trial, 211 patients with ICH and SBP >180 mmHg were treated with nicardipine towards a SBP goal of 120–160 mmHg; it was found that high achieved BP levels were associated with poor clinical outcomes [28]. The INTERACT-2 trial compared aggressive (<140 mmHg) versus conservative (<180 mmHg) antihypertensive therapy in 2794 ICH patients and did not found any statistically significant differences in death or severe disability between the two strategies, except for a small functional benefit of aggressive therapy [29]. Similarly, the ATACH-2 trial randomized 1000 ICH patients to aggressive (110–139 mmHg) or conservative (140–179 mmHg) SBP goals, by using IV nicardipine. Intensive BP lowering did not provide any significant mortality or morbidity benefit; in contrast it was associated with increased rates of renal adverse effects [30]. Based mainly on the above-mentioned limited data, the 2018 ESC/ESH guidelines for the management of arterial hypertension do not recommend immediate BP lowering in ICH patients, except for patients with pronounced BP elevation (>220 mmHg), for whom careful acute BP reduction to conservative goals (<180 mmHg) under close monitoring should be considered [8].

9. Acute ischemic stroke

The above described uncertainty regarding BP management in ICH patients is even greater in acute ischemic stroke. A large meta-analysis of 13 randomized controlled trials with almost 13,000 participants failed to uncover any significant functional, morbidity, or mortality benefit of early BP reduction compared with control in patients with acute ischemic stroke [29]. Likewise, no significant benefit was observed by the continuation of prior antihypertensive therapy in the first few days after an acute ischemic stroke [31–33]. Therefore, the 2018 ESH/ESC guidelines for the management of arterial hypertension do not support early BP lowering in patients with acute ischemic stroke, except in two cases: patients with pronounced BP elevation or patients who are eligible for thrombolysis [8]. In patients with acute ischemic stroke and marked BP elevation (defined as SBP >220 mmHg and/or DBP >120 mmHg), careful BP lowering may be considered – based on clinical judgment – with a 15 % reduction seeming a rational goal for the first post-stroke day. In patients who are eligible for thrombolysis, a more aggressive approach seems reasonable, due to the risk of haemorrhage following thrombolysis. Indeed, the SITS-ISTR and the TIMS-China trials showed that high BP levels were associated with increased risk for

intracerebral haemorrhage in patients receiving thrombolytic therapy [34,35]. Therefore, the ESC/ESH guidelines recommend that BP should be lowered to <180/105 mmHg before thrombolysis and maintained at these levels for the first post-thrombolysis day [8]. The target BP after thrombolytic therapy is still a matter of discussion [36].

10. Hypertensive encephalopathy

Hypertensive encephalopathy is not a very common form of hypertensive emergencies, although its prevalence may be as high as 15 % in patients with malignant hypertension [37]. Hypertensive encephalopathy is a rather vague term describing a cluster of clinical symptoms from the brain, ranging from the gradual onset of mild neurological symptoms, such as headache, nausea, vomiting, and somnolence, to more severe symptoms, such as visual disturbances, restlessness, irritation, seizures, confusion, lethargy, and even coma. Neuroimaging (CT or preferably MRI) is essential to uncover local brain oedema, as well as to exclude ischemic stroke, haemorrhage, or tumors, which are included in the differential diagnosis. The mediating pathophysiological mechanisms include increased perfusion pressure, disruption and increased permeability of the blood-brain barrier, endothelial dysfunction, oedema, and microvascular alterations. Hypertensive encephalopathy represents a form of hypertensive emergency, since it may progress to intracranial haemorrhage and death if left untreated. Therefore, an immediate reduction of BP is recommended by the ESC/ESH guidelines; the goal should be to reduce mean arterial pressure by 20–25 %, since larger reductions may be associated with neurological complications [38,39]. Labetalol and nicardipine represent first-choice agents, while sodium nitroprusside, fenoldopam, and clevidipine may be used as alternatives. Labetalol seems to be preferred over sodium nitroprusside, since it affects less cerebral blood flow [40]. Posterior reversible encephalopathy syndrome (PRES) is a clinicoradiologic disorder that resembles hypertensive encephalopathy, and shows significant overlap. Renal hypertension, autoimmune diseases, transplantation, immunosuppression, and pre-eclampsia/eclampsia are the main causes of the syndrome. Of note, PRES has been recently reported in COVID-19 patients, reaching a prevalence rate of 3.9 % [41–43].

11. Others

Several other conditions may present as hypertensive urgencies or emergencies. For the purposes of this review paper, three such conditions have been selected (pheochromocytoma, pregnancy, and drug-induced) for a brief description, due to their major clinical importance and specific management recommendations.

12. Pheochromocytoma

Pheocromocytomas and paragangliomas are very rare neuroendocrine tumors that arise from chromaffin cells (adrenal and extra-adrenal, respectively), causing paroxysmal hypertension, along with headache, palpitations, hyperhidrosis, and a plethora of other clinical symptoms. Surgical excision is the gold-standard therapy for pheochromocytomas and paragangliomas; therefore, hypertensive crises in such patients should be considered in the context of pre-operative management or during the surgery, while hypertensive crises with relevant symptoms should raise suspicion in undiagnosed cases. Acute blood pressure elevation due to excessive release of catecholamines should be considered a hypertensive emergency, due to the potential risk of myocardial ischemia, fatal arrhythmias, and even sudden death. In addition, tumors mainly releasing norephinephrine (1/3 of the cases) are often characterized by chronic hypertension and the absence of rarity of episodes of paroxysmal hypertension while those mainly releasing epinephrine are often characterized by episodes of paroxysmal hypertension. Most adrenal pheochromocytomas however secrete both norepinephrine and epinephrine [46]. Oral selective, competitive alpha-1 blockers, such as prazosin, doxazosin, and terazosin can be used in pre-operative management (for 2–4 weeks), while phenoxybenzamine (a non-competitive alpha blocker) might prevent displacement by catecholamine surge [44]. The mandatory pre-operative use of alpha blockers has been recently questioned [45], but it still remains standard of care in the vast majority of expert centers worldwide. Intravenous agents, including phentolamine (a non-selective alpha blocker) and sodium nitroprusside are appropriate for rapid BP reduction under very close monitoring, either in the ICU for hypertensive emergencies or in the operation room during surgery. In the case of tachycardia or other tachyarrhythmias, beta blockers should not be administered until alpha blockade is achieved due to the risk of hypertension and tachycardia aggravation via unopposed alpha adrenergic receptors [46]. In such cases, either labetalol (an alpha- and beta- blocker) or bradycardic calcium antagonists may be used [47].

13. Pregnancy associated hypertensive syndromes

Hypertension in pregnancy is a heterogeneous disorder, including pre-existing hypertension, gestational hypertension, or a combination of both conditions. Pre-eclampsia is a specific form of hypertension in pregnancy, in which hypertension may be accompanied by neurological symptoms (headache, nausea, vomiting, and visual disturbances) and vague abdominal pain, with laboratory abnormalities (proteinuria: >300 mg/24 h, increased uric acid and aminotrasferase levels, low platelets, and hemolysis); in eclampsia, seizures occur, mandating immediate management and delivery. Neuroimaging studies reveal that brain involvement occurs in the vast majority of cases [48]. Epidemiological evidence indicates that hypertension in pregnancy accounts for 15 % of direct maternal deaths, with eclampsia accounting for half of them [49]. In addition, severe pre-eclampsia/eclampsia accounts for one third of acute ischemic strokes in pregnancy [50]. Trials regarding BP management in pregnant women are very limited, and thus recent recommendations on both sides of the Atlantic are mainly based on expert opinion. All societies support the administration of antihypertensive therapy in women with BP levels >160/100 mmHg, while BP levels between 140 and 160 mmHg for SBP and 90–100 mmHg for DBP represent a gray zone. According to the ESC/ESH guidelines [8,51], blood pressure levels >170 mmHg for SBP and/or >110 mmHg for DBP are considered a hypertensive emergency in pregnancy and require hospital admission for immediate therapy, in order to reduce blood pressure <160/100 mmHg. All guidelines agree that available options include methyldopa per os, calcium antagonists per os or IV, and labetalol IV; hydralazine is not recommended by the ESC/ESH guidelines due to the increased risk of perinatal complications [52], while obstetric guidelines include IV hydralazine in therapeutic options for emergency treatment [53]. In severe pre-eclampsia, magnesium sulfate should be given intravenously for the prevention of seizures, along with intravenous labetalol or nicardipine which represent first-line options [54]. In the few cases of pulmonary oedema, nitroglycerin should be preferred over sodium nitroprusside due to the risk of foetal cyanide poisoning with the latter agent. Of major importance, delivery represents the ‘final cure’ in severe pre-eclampsia/eclampsia, and thus should be considered as soon as the clinical condition is stabilized and gestational age permits [55]. Major drugs used in hypertensive emergency are shown in Table 3.

14. Hypertension as a result of medications

Several drugs and other substances are known to cause BP elevation; NSAIDs represent the most common class of these drugs, followed by gluco- and mineralo-corticoids. Other drugs in this group include oral contraceptives and diet pills, cyclosporine, and erythropoietin, while some herbal remedies and liquorice may also increase BP levels. All these drugs usually result in chronic hypertension or loss of prior BP control, thus representing hypertensive urgencies. In contrast, illicit substances, such as cocaine, ecstasy, LSD, and amphetamines may cause

acute hypertension along with other complications, thus representing hypertensive emergencies. Special consideration is needed for the newest member of drugs raising BP. The advent of anticancer therapy generated biological therapies that revolutionized the field of chemotherapy [56]. Monoclonal antibodies targeting the vascular endothelial growth factor (VEGF) pathway (bevacizumab, ramucizumab, aflibercept), as well as tyrosine kinase inhibitors (sunitimib, pazopanib, sorafenib, axitinib, lapatinib, cediramib, lenvatinib, vandetanib, regorafenib, lucitanib) are currently used for the treatment of metastatic renal, breast, and liver cancer, hematologic malignancies, and other indications. BP elevation is the most common cardiovascular event caused by these drugs, representing collateral damage from reduced nitric oxide bioavailability and microvascular damage, caused by such molecules [57,58]. Although BP elevation has been reported in rates as high as 75 % of treated patients [59], it seems to occur in about one third of patients receiving these agents [60], and seems to be even higher with newer agents [61] that have much higher affinity against VEGF-receptors [62]. The US National Cancer Institute graded the severity of anticancer drug-induced hypertension, with grade III hypertension defined as BP levels >160/100 mmHg, and grade IV hypertension defined as hypertension associated with life-threatening consequences, suggesting that grade IV hypertension is mostly an emergency, while grade III hypertension is mostly an urgency. Grade IV hypertension seems to be three times higher with these agents than conventional chemotherapy [59], and the incidence rate ranges from 0.5 to 2 % [63–65]. The management of hypertension includes first the withdrawal of the implicated drug [66], which however has two caveats: a) the substitution of the drug with another does not exclude the possibility of reappearance, and b) BP elevation may last long due to the long half-life and the high receptor affinity of these drugs [62]. Although nitric oxide donors, such as nitroglycerin and sodium nitroprusside seem ideal candidates from the pathophysiological point of view, current hypertension treatment follows a classical approach, with calcium antagonists and RAS-inhibitors being used for hypertensive urgencies, while for hypertensive emergencies treatment is offered according to the specific type of emergency [8]. Non-dihydropiridinic calcium channel blockers should not be used due to the risk of drug-drug interactions.

15. Hypertension urgency

Hypertension urgency is a term used to describe severe hypertension in patients presenting to the ED with no clinical evidence of an acute subclinical or clinical condition. The burden of hypertensive urgency is not well defined, mainly because of the different criteria used for the definition of the so called “hypertensive crisis” that have influenced the epidemiological data collected in the literature. It seems that approximately 28 % of the patients admitted to the ED for hypertensive crisis were attributed to hypertensive emergencies while the prevalence of hypertensive urgencies represents 0.9 % of the admissions to the ED [67–69]. The aetiology of this acute increase in BP levels is variable and may be attributed, at least in part, to non-compliance with antihypertensive therapy, the use of sympathomimetics or NSAIDs, thyroid dysfunction or causes that increase BP levels such as anxiety, pain etc. These patients do not need aggressive antihypertensive therapy but a careful consideration and assessment of their BP burden, emphasizing the need for compliance with medications and close primary care follow-up. Indeed, in a randomized study, the use of antianxiety treatment was effective in lowering BP in patients with excessive hypertension [74]. Patients with hypertensive urgency may be completely asymptomatic and more likely to make a visit to an outpatient clinic. Since data on short and long term prognosis of patients with acute BP increase who lack acute HMOD or patients who present with asymptomatic uncontrolled hypertension is lacking, it would be reasonable to maintain the term “hypertensive urgency” and not substitute “hypertensive urgency” with the term uncontrolled hypertension [75].

16. Who should be hospitalized

Epidemiological data regarding the admission percentage due to hypertensive urgencies or emergencies are limited, with a range of 0.46–0.73 % of total admissions [70,71]. Until recently, there was a lack of worldwide accepted guidelines regarding indications for hospitalization for patients presented with uncontrolled BP in the ED. According to the latest published International Society of Hypertension (ISH), ESC)/ESH guidelines, the decision for hospital admission is based on risk stratification tools and well described definitions of the current terms “hypertensive emergencies” and “hypertensive urgencies”. Upon presentation to the ED, after initial assessment, the main indication for hospitalization is the identification of a hypertensive emergency. Hypertensive emergency is often a life-threatening clinical condition and is defined as the presence of abnormal BP values associated with acute hypertension-related organ damage. Clinically evident injuries of the target organs include malignant hypertension (>180/120 mmHg), renal impairment, retinopathy, encephalopathy, thrombotic microangiopathy, cardiovascular events (acute coronary syndrome, aortic dissection, acute pulmonary edema) and cerebrovascular emergencies such as acute ischemic or hemorrhagic stroke [72]. An algorithm for the assessment of hypertensive emergencies is shown in Fig. 1. Those patients classified as hypertensive emergencies cannot be managed as outpatients with oral antihypertensive drugs, so urgent admission is required for in-hospital management (Table 1). Acute management during hospitalization includes close hemodynamic monitoring, controlled and timely BP reduction with intravenous antihypertensive agents in order to limit further organ damage and avoid complications [73]. The treatment of choice, the timeframe and the level of BP reduction are determined by clinical presentation (Table 2). Further management includes diagnostic work up for common causes and specific interventions according to indications [8]. In pregnancy, according to 2018 ESC Task Force, BP > 170/110 mmHg, immediate hospitalization (class I, level C) is required for intravenous treatment and urgent delivery if indicated [8,75]. Criteria for hospital admission are shown in Table 1. Patients classified as hypertensive urgency, who present with severe asymptomatic hypertension (≥180/110 mmHg) without clinical evidence of acute hypertension-mediated target organ dysfunction, usually do not require admission. Gradual blood pressure reduction with oral agents is indicated, and they can be discharged safely from the emergency department unit [73].

17. Most common errors in diagnosis and therapies in clinical practice

First of all, clinicians must distinguish hypertension urgency (a benign condition that does not require hospitalization) from emergency, a condition that requires immediate therapy and hospitalization. The presence or absence of acute organ damage will solve this query. Treatment of hypertensive urgency doesn’t require immediate therapy, and in these patients, the hypertensive burden must be assessed before starting antihypertensive treatment. On the contrary, a hypertensive

Table 1
Most common urgency conditions and criteria for hospital admission.

Hypertensive emergencies	Hypertensive urgencies
Severe hypertension (>200/120 mmHg) and	Non-adherence with antihypertensive therapy, Use of sympathomimetics or NSAIDs
Fundoscopic abnormalities	Thyroid dysfunction
Encephalopathy	Causes that increase BP levels (stressors such as anxiety, pain ext.)
Thrombotic microangiopathy	Out patient (most cases)
Cardiovascular events	Oral treatment
Pregnancy Blood Pressure >170/110 mmHg	

Table 2
Diagnostic Approach and Treatment.

Comorbidity	BP Target	Preferred Drugs
ACS	<140 mm Hg Not <120 mm Hg	NTG, b-blockers
Aortic Dissection	<120 mm Hg, HR: <60b/min	Esmolol, NTP, NTG, Labetalol Nicardipine
Cocaine use	Not <120 mm Hg	Phentolamine
Acute Pulmonary Oedema	<140 mm Hg Not <120 mm Hg	NTG, Loop Diuretics
Ischemic Stroke	<220/110 mm Hg 1H, MAP 15 %	Labetalol, Nicardipine, Clevidipine
Hemorrhagic Stroke	<160 mm Hg	Labetalol, Nicardipine, Clevidipine
Eclampsia	<160/100 mm Hg	Labetalol, Nicardipine, Clevidipine
Pheochromocytoma	Immediate 25 %	Phentolamine, Clevidipine
HTN Encephalopathy	Immediate 25 %	Nicardipine, Clevidipine, Labetalol, NTP

NTP: nitroprusside, NTG: Nitroglycerine, HTN: Hypertension, ACS: Acute coronary sindrome, MAP: mean arterial pressure, HR: heart rate.

Table 3
Drugs, Duration of Action, Doses, and Contraindications.

Drug	Duration	Dose	Contraindication
NTG	3–5 min	5–200 mg/min I.V.	
NTP	1–2 min	0.3–10 mg/kg/min I.V. Increase by 0.5 mg/kg/min	CKD, Liver Failure
Esmolol	10–30 min	0.5–1 mg/kg/ I.V. bolus 50–300 mg/kg/min I.V	2nd or 3rd AV block HFrEF, COPD, Low HR
Labetalol	3–6 h	0.25–0.5 mg/kg I.V. bolus 2–4 mg/min I.V.	2nd or 3rd AV block HFrEF, COPD, Low HR
Clevidipine	5–15 min	2 mg/h I.V. increase every 2 min 2 mg/h	
Nicardipine	30–40 min	5–15 mg/h I.V. invrease 2.5 mg every 15–30 min	Liver failure
Phentolamine	10–30 min	0.5–1 mg/kg I.V. bolus or 50–300 mg/kg/min I.V.	

NTP: nitroprusside, NTG: Nitroglycerine, COPD: chronic obstructive pulmonary disease, HR: heart rate, HfrEF: heart failure redused ejection fraction, AV: atrioventricular, CKD: chronic kidney disease.

emergency requires intravenously administered drugs with a short onset and duration of action in order to rapidly adjust the fluctuations in BP levels. In addition, frequently patients with dyspnea and respiratory failure present also increased BP levels. However, the increase in BP is a consequence and not the cause of dyspnea. In several cases different stressful stimuli may increase BP levels. In this patients treatment of the main respiratory problem will lead to a BP reduction, while specific laboratory (Brain natriuretic peptides) and non-laboratory tests (e.g. echocardiography, radiologic studies etc.) will also help to solve this query. Meticulous BP measurements even in the emergency department is of crucial importance and should be done in both arms and limbs as per guidelines. Repeated BP measurements will allow discriminating physical and mental stressful situations or a white coat effect as cause of temporary urgencies, where TOD symptoms are absent. Lastly, patients with aortic dissection may present with signs and symptoms of ischemic stroke, myocardial infarction and limb ischemia [76]. These complications are mainly related to the malperfusion of specific organs because of the aortic dissection. Moreover, signs and symptoms of aortic dissection are not always typical, and the patient may present with chest pain or neurological symptoms. It's imperative to suspect and confirm the aortic dissection before proceeding to myocardial infarction or ischemic stroke treatment since the latter will have devastating results. The use of echocardiography may reveal the presence of a dissection membrane or a dilatation of the aortic root while a further evaluation with transoesophageal echocardiography and CT angiography will help to exclude or confirm the diagnosis of aortic dissection. In these patients, DDimer levels are always increased.

18. Conclusions

The diagnostic approach and treatment of hypertensive emergencies in the emergency department are often challenging. This life- threatening situation requires an immediate and well controlled intervention to decrease BP levels in most cases with intravenous therapy. It's important to distinguish hypertensive emergency from hypertensive urgency, which is usually considered a more benign condition that requires more likely an outpatient visit and treatment.

Declaration of Competing Interest

None declared

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