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CHARACTERISTICS OF THE FIRST ORGANS DAMAGED IN HYPERTENSION THE BRAIN, HEART AND KIDNEYS

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Systemic arterial hypertension is characterized by persistently high blood pressure (BP) in the systemic arteries. BP is commonly expressed as the ratio of the systolic BP (that is, the pressure that the blood exerts on the hypertension depend on the measurement method. Several aetiologies can underlie arterial walls when the heart contracts) and the diastolic BP. The BP thresholds that define hypertension. The majority (90–95%) of patients have a highly heterogeneous essential or primary hypertension with a multifactorial gene-environment aetiology [1,2,3].

The heart, kidney, brain, and arterial blood vessels are prime targets of hypertensive damage. Uncontrolled hypertension accelerates the damage to these organs and results in eventual organ failure and cardiovascular death and disability. Current guidelines for the appropriate treatment and control of hypertension requires an assessment of the presence of target organ damage. When present, evidence of target organ damage determines the urgency and intensity of drug treatment and may also dictate the choice of initial antihypertensive drug class.

Thus, evaluation of persons with suspected or established hypertension must include a meticulous search for evidence of target organ damage. Fortunately, treatment with all antihypertensive medications that results in significant BP reductions also reduces fatal and nonfatal hypertensive complications and significantly slows down the progression to organ failure. Because of the important role that adverse activation of the renin-angiotensin-aldosterone system plays in target organ damage, drugs that antagonize this system have provided consistent and compelling proof of organ protection in both primary and secondary prevention of adverse outcomes. The challenge now is to use these and all other antihypertensive agents effectively to control BP to target levels in patients with hypertension. Continued emphasis on the adoption of lifestyle changes for prevention of hypertension in the first place or as adjunctive therapy in hypertensive patients is essential [4,5,6,7].

Hypertension is the most common preventable risk factor for cardiovascular disease (CVD; including coronary heart disease, heart failure, stroke, myocardial infarction, atrial fibrillation and peripheral artery disease), chronic kidney disease (CKD) and cognitive impairment, and is the leading single contributor to all-cause death and disability worldwide. The relationship between BP and the increased risk of CVD is graded and continuous, starting as low as 115/75 mmHg, well within what is considered to be the normotensive range. Successful prevention and treatment of hypertension are key in reducing disease burden and promoting longevity in the world's population. In

treating hypertension, it is important to consider a person's predicted atherosclerotic CVD (ASCVD) risk more than the level of BP alone, as persons with high CVD risk derive the greatest benefit from BP lowering treatment. BP is determined by several parameters of the cardiovascular system, including blood volume and cardiac output as well as the balance of arterial tone that is affected by both intravascular volume and neurohumoral systems. The maintenance of physiological BP levels involves a complex interplay of various elements of an integrated neurohumoral system that includes the renin-angiotensin-aldosterone system (RAAS), the role of natriuretic peptides and the endothelium, the sympathetic nervous system (SNS) and the immune system. Malfunction or disruption of factors involved in BP control in any of these systems can directly or indirectly lead to increases in mean BP, BP variability or both, over time resulting in target organ damage (for example, left ventricular hypertrophy and CKD) and CVD outcomes [8,9,10,11].

Hypertensive Heart Disease: Hypertensive heart disease is subclassified by the presence or absence of heart failure as the management of heart failure requires more intensive goal-directed therapy. Hypertensive heart disease can lead to either diastolic heart failure, systolic failure, or a combination of the two. Such patients are at a higher risk for developing acute complications such as decompensated heart failure, acute coronary syndrome, or sudden cardiac death. Hypertension disrupts the endothelial system which increases the risk of coronary artery disease and peripheral

arterial disease and thus represents a significant risk factor for the development of atherosclerotic disease. However hypertensive heart disease ultimately encompasses all of the direct and indirect sequelae of chronic high blood pressure which include systolic or diastolic heart failure, conduction arrhythmia especially atrial fibrillation, and increased risk of coronary artery disease. Hypertensive heart disease is a chronic progressive disease that carries a significantly increased risk of cardiovascular death. Hypertension is one of the predominant risk factors for the development of several cardiovascular diseases such as coronary artery disease, congestive heart failure, atrial fibrillation, cerebrovascular disease, peripheral arterial disease, aortic aneurysm, and chronic kidney disease. The overall prognosis of hypertensive heart disease is variable but depends on a variety of factors such as the specific manifestations of the disease, the presence of concomitant cardiovascular disease or risk factors, and other comorbid conditions. Cardiovascular risk calculators are available, and patients should be stratified into either high or low risk for cardiovascular events. Specific manifestations of HHD such as heart failure or atrial fibrillation carry a substantially increased risk of cardiovascular mortality. Patients with diastolic heart failure confer a similar risk and morbidity to patients with low ejection heart failure with 6-month mortality observed as high as 16%. Hypertensive heart disease is a syndrome of complication relating to the cardiovascular complications associated with chronic hypertension. Hypertension ranks as the most prevalent modifiable risk factor for

premature cardiovascular disease and cardiovascular mortality and requires constant surveillance to identifying complications and slow their development. Prolonged hypertension promotes left ventricular hypertrophy which will eventually lead to heart failure (both systolic and diastolic). Eccentric hypertrophy leads to increased oxygen demand by the myocardium which can result in angina or ischemic symptoms. Hypertrophy of the muscle can disrupt conduction pathways predisposing to atrial fibrillation which leads to ischemic stroke. Acute alterations in blood pressure can predispose patients to intracerebral hemorrhage or retinopathy. Prolonged hypertension is the predominant risk factor for the development of cardiac disease including atherosclerotic disease, heart failure, valvular disease, atrial fibrillation as well as cerebrovascular disease, chronic kidney disease, retinal disease, and metabolic disease. Nearly half of strokes and ischemic heart disease are attributable to sustained hypertension [12,13,14,15].

Sympathetic Nervous System: Baroreceptors, mechanoreceptors that sense pressure changes of the circulatory system, are housed in various locations in the arterial tree, a key place being the carotid sinus, a dilated area at the base of the internal carotid artery just superior to bifurcation of the common carotid artery. When this artery is stretched by elevated BP, nerve bundles projecting from the baroreceptors in the carotid sinus send messages to the brain to reduce sympathetic outflow of nerve impulses or nerve traffic and, thereby, BP. The SNS is generally more activated in persons with

hypertension than in normotensive individuals. SNS activity is also greater in individual with obesity, in men than in women, in younger than in older persons, and in those with advanced kidney disease. Many patients with hypertension are in a state of autonomic imbalance with increased sympathetic and decreased parasympathetic activity. SNS hyperactivity is relevant to both the generation and maintenance of hypertension. Studies in humans have also identified markers of sympathetic overactivity in normotensive individuals with a family history of hypertension. Among patients with hypertension, increasing severity of hypertension is associated with increasing levels of sympathetic activity measured by microneurography. Plasma catecholamine levels, microneurographic recordings and systemic catecholamine spillover studies have given evidence of increased sympathetic activity in patients with hypertension who are obese, in those with the metabolic syndrome, and in those whose hypertension is complicated by heart failure or kidney disease [16,17].

Endocrine Hypertension: The importance of the SNS in the pathogenesis of hypertension has been defined in a variety of experimental models. Models of obesity-related hypertension demonstrate that increased renal sympathetic nerve activity and its attendant increase in renal sodium reabsorption are key factors in the maintenance of sustained hypertension. In another animal model, rats that received daily infusions of phenylephrine for 8 weeks developed hypertension during the infusions; their BP normalized under a low salt diet after discontinuation of

phenylephrine, but once re-challenged with a high salt diet, the animals became hypertensive again. The degree of BP elevation on the high salt diet was directly related to the degree of renal tubulointerstitial fibrosis and decrease in glomerular filtration rate, suggesting that catecholamine-induced hypertension causes renal interstitial injury and a salt-sensitive phenotype that persists even after sympathetic overactivity is no longer present. In addition, enhanced SNS activity results in α -1 adrenergic receptor mediated endothelial dysfunction, vasoconstriction, vascular smooth muscle proliferation and increased arterial stiffness, which contribute to the development and maintenance of hypertension. Finally, there is evidence that sympathetic overactivity enhances salt-sensitivity owing to a reduction in activity of the WNK lysine deficient protein kinase 4 (WNK4) gene, which encodes a serine/threonine kinase that inhibits the thiazide-sensitive-Na-Cl co-transporter, resulting in increased distal tubular Na^+ retention [17,18].

A Obesidade é um problema global de saúde publica que na maioria das vezes pode ser melhorado com a mudança nos hábitos alimentares, diminuindo o consumo de alimentos processados e aumentando a actividade física como forma de combater os diferentes desequilíbrios hormonais que podem surgir em muitos indivíduos. Entre os factores causadores ou concomitantemente observados na hipertensão endócrina inclui-se um aumento na actividade hormonal, como por exemplo, da aldosterona, dos mineralocorticóides, do cortisol, das catecolaminas e as hormonas do

crescimento, da tireóide e da paratireóide. Por outro lado, a insulino-resistência, a deficiência na hormona do crescimento, a testosterona, a Vitamina D e as hormonas da tireóide podem também contribuir para a hipertensão endócrina. Neste trabalho apresenta-se uma revisão dos mais recentes avanços obtidos na já tradicional hipertensão endócrina de causa suprarrenal e são ainda indicados outros aspectos menos referidos desta hipertensão, como por exemplo a nossa hipótese de que a dependência da comida, de doces e de sal é um dos aspetos mais importantes em muitas sociedades e como tal devem ser abordados de forma holística e conforme o antigo pensamento filosófico de que uma mente sã levará a um corpo sã. As we now know, the BMI definition of obesity is not perfect when assessing the risk for developing type 2 diabetes, as it does not tell the body composition, i.e. lean and fat mass. It also does not assist in separating obese individuals with insulin resistance and an inflammatory state from those with insulin sensitivity and/or no inflammatory state. This explains why obesity-associated hypertension is a complex entity that belongs to a form of endocrine hypertension, as in some patients, testosterone deficiency and growth hormone deficiency, as well as vitamin D deficiency, may coexist with obesity. In women with polycystic ovarian syndrome, it remains controversial whether associated hypertension is independent of obesity. On the other hand, elevated blood pressure related to the intake of oral contraceptives and postmenopausal hormone therapy can be reversed by stopping hormonal therapy [4,7,9].

Diagnosis: The evaluation of a patient with hypertension requires more than the diagnosis of elevated BP. It should also include assessment of the CVD risk, target organ damage, and concomitant clinical conditions that may affect the BP or related target organ damage as well as recognition of features suggestive of secondary hypertension. Some of these investigations are routine tests necessary in all patients, but others only in specific patient groups identified by history, clinical examination, and routine tests. In rare inherited forms of hypertension, a single gene mutation explains the pathogenesis of hypertension. A small proportion of patients have a potentially reversible cause of hypertension, and a correct diagnosis might lead to a cure or a substantial improvement in BP control with a reduction of CVD risk. It is therefore appropriate to implement a simple screening for secondary hypertension in all patients. The screening is based on clinical history, physical examination and routine laboratory investigations. Secondary hypertension should also be considered in cases of a sudden worsening of hypertension, poor BP response to drug treatment or severe target organ damage, which is out of proportion to the duration and severity of hypertension. In these cases, specific diagnostic tests are indicated [14].

The medical history has to address the time of the first diagnosis of hypertension, current and past BP measurements and antihypertensive medications. A history of pregnancy-related hypertension is an important factor in the assessment of women with hypertension. Hypertension results in an

increased risk of CVD complications, and chronic kidney disease (CKD). Thus, a careful medical history should be taken in all patients to allow for assessment of global CVD risk, with special emphasis on current and past smoking habits and evidence of dyslipidaemia and diabetes mellitus. CVD risk should be estimated using an established calculator. Adults at high risk for CVD have a high probability to benefit from antihypertensive drug therapy in addition to lifestyle change [10]. The physical examination aims to establish the diagnosis of hypertension and screen for target organ damage and secondary causes. The patient should sit quietly for 5 minutes before a BP reading is taken and BP cuff should be at heart level. An average of 2 to 3 BP measurements obtained at 2 to 3 separate occasions provides an accurate basis for estimation of BP. At least once, BP should be measured on both arms, and differences in SBP > 20 mmHg and/or in DBP > 10 mmHg should initiate investigations of vascular abnormalities.

Pharmacological Interventions:

Low-dose pharmacological therapy has also been shown to be effective in lowering BP and preventing hypertension in three randomized controlled trials conducted in adults with high normal BP. The Brazilian multi-center PREVER-Prevention Trial compared treatment with the low-dose long-acting thiazide-like diuretic chlorthalidone in combination with the potassium sparing agent amiloride with treatment with placebo. Treatment with the low-dose chlorthalidone and amiloride combination resulted in both a decrement in BP and prevention of hypertension and a reduction in left

ventricular mass. A drug intervention is easier to implement and maintain than a lifestyle change intervention but there is a natural reluctance to recommend a lifetime of pharmaceutical therapy for prevention of hypertension. Consideration of low-dose pharmacotherapy should be restricted to those who are at high risk of developing hypertension despite energetic efforts to lower BP by means of one or more nonpharmacological interventions.

Intensive BP treatment in SPRINT resulted in a significantly greater (25%) reduction in the primary endpoint, compared with standard treatment. Office BP measurement in SPRINT was performed with an automated device timed to start measurement after 5 minutes of rest in an effort to standardize measurements in the various clinics and minimize the white coat effect. Because large differences had been observed between automated office BP measurement and conventional auscultatory measurements, some groups have questioned the applicability of the SPRINT intensive systolic BP target of < 120 mmHg to ordinary office practice. Both the appropriate methods of measuring office BP and the appropriate BP targets for antihypertensive treatment are currently topics of vigorous debate. In summary, newer guidelines published after the SPRINT trial generally have more aggressive goals, at least for individuals < 65 years of age .

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XÜLASƏ

HİPERTONİYADA ZƏDƏLƏNMİŞ ORQANLARIN - BEYİN, ÜRƏK VƏ BÖYRƏKLƏRİN XÜSUSİYYƏTLƏRİ

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Arterial hipertenziya arteriyalarda davamlı yüksək qan təzyiqi (BP) ilə xarakterizə olunur. Qan təzyiqi adətən sistolik qan təzyiqi (yəni, qanın hipertoniyaya göstərdiyi təzyiq ölçmə metodundan asılıdır. Ürək yığılan zaman arterial divarların əsasında bir neçə etiologiya dayana bilər) və diastolik təzyiqin nisbəti kimi ifadə edilir. Hipertoniyanı təyin edən BP hədləri xəstələrin əksəriyyətində (90-95%) multifaktorial gen-mühit etiologiyası ilə yüksək heterojen və ya birincili hipertenziyaya əsaslanır.

Ürək, böyrəklər, beyin və arterial qan damarları hipertansif zədələnmənin əsas hədəfləridir. Nəzarətsiz hipertoniya bu orqanların zədələnməsini sürətləndirir və nəticədə orqan çatışmazlığına, ürək-damar xəstəliklərinə ölüm və əlilliyə səbəb olur. Hipertoniyanın müalicəsi və nəzarəti üçün lazım olan təlimatlar, hədəf orqan zədələnməsinin mövcudluğunun qiymətləndirilməsini tələb edir. Hədəf orqan mövcud olduqda, orqan zədələnməsinin sübutu dərman müalicəsinin aktuallığını və intensivliyini müəyyən edir və həmçinin ilkin antihipertenziv dərman sinfinin seçimini təyin edə bilər.

Beləliklə, şübhəli və ya müəyyən edilmiş hipertansiyonu olan şəxslərin qiymətləndirilməsi hədəf orqanın zədələnməsinə dair sübutların dəqiq axtarışını əhatə etməlidir. Xoşbəxtlikdən, qan

təzyiqinin əhəmiyyətli dərəcədə azalması ilə nəticələnən bütün antihipertenziv dərmanlarla müalicə ölümçül və ölümçül olmayan hipertansif ağırlaşmaları azaldır və orqan çatışmazlığının gedişatını əhəmiyyətli dərəcədə ləngidir. Renin-angiotenzin-aldosteron sisteminin mənfi aktivləşdirilməsinin hədəf orqan zədələnməsində oynadığı mühüm rola görə, bu sistemi antaqonlaşdıran dərmanlar mənfi nəticələrin həm birincili, həm də ikincil qarşısının alınmasında orqan qorunmasının ardıcıl və inandırıcı sübutunu təmin etmişdir.

Açar sözlər: *hipertoniya, beyin, ürək, böyrək*

РЕЗЮМЕ

ХАРАКТЕРИСТИКА ОРГАНОВ, ПОРАЖАЕМЫХ ПРИ ГИПЕРТОНИИ - МОЗГ, СЕРДЦА И ПОЧЕК

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Артериальная гипертензия характеризуется устойчиво высоким артериальным давлением (АД) в системных артериях. АД обычно выражается как отношение систолического АД (то есть давления, которое кровь оказывает на артериальную гипертензию, зависит от метода измерения. Несколько этиологий могут лежать в основе артериальных стенок при сокращении сердца) и диастолического АД. Пороговые значения АД, определяющие гипертензию. Большинство (90–95%) пациентов имеют крайне гетерогенную эссенциальную или первичную гипертензию с многофакторной генно-средовой этиологией. Сердце, почки, мозг и артериальные кровеносные сосуды являются основными целями гипертонического повреждения. Неконтролируемая гипертония ускоряет повреждение этих органов и приводит к возможной органной недостаточности и сердечно-сосудистой смерти и инвалидности. Текущие рекомендации по надлежащему лечению и контролю гипертонии требуют оценки наличия повреждения целевого органа. При наличии доказательства повреждения целевого органа определяют срочность и интенсивность медикаментозного лечения, а также могут диктовать выбор первоначального класса антигипертензивных препаратов.

Таким образом, оценка лиц с подозреваемой или установленной гипертонией должна включать тщательный поиск доказательств повреждения целевого органа. К счастью, лечение всеми антигипертензивными препаратами, которое приводит к значительному снижению АД, также снижает фатальные и нефатальные гипертонические осложнения и значительно замедляет прогрессирование органной недостаточности. Из-за важной роли, которую играет неблагоприятная активация ренин-ангиотензин-альдостероновой системы в повреждении целевого органа, препараты, которые антагонизируют эту систему, предоставили последовательные и убедительные доказательства защиты органов как при первичной, так и при вторичной профилактике неблагоприятных исходов.

Ключевые слова: *гипертония, мозг, сердце, почки*