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ARTERIAL HYPERTENSION IN THE ELDERLY AND THE VERY OLD: PATHOPHYSIOLOGY, CLINICAL PHENOTYPES, AND PROGNOSTIC IMPLICATIONS

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Abstract. Arterial hypertension represents one of the most significant medical and social challenges in the context of global population aging. Advancing age is associated with a sharp increase in hypertension prevalence, substantial changes in its clinical profile, and an amplified contribution to cardiovascular, cerebrovascular, and cognitive morbidity. This review analyzes age-related blood pressure dynamics, the pathophysiological mechanisms underlying hypertension in elderly and very old individuals, and its atypical clinical phenotypes, including isolated systolic hypertension, pseudohypertension, orthostatic, office, and masked hypertension. Particular emphasis is placed on the role of arterial stiffness, impaired baroreflex function, and circadian blood pressure dysregulation. The prognostic implications of hypertension and hypotensive states with regard to stroke, heart failure, dementia, and mortality are discussed. The review highlights the necessity of an individualized diagnostic and risk stratification strategy in older patient populations.

Key words: arterial hypertension, elderly, isolated systolic hypertension, arterial stiffness, orthostatic hypotension, dementia.

АРТЕРИАЛЬНАЯ ГИПЕРТЕНЗИЯ В ПОЖИЛОМ И СТАРЧЕСКОМ ВОЗРАСТЕ: ПАТОФИЗИОЛОГИЯ, КЛИНИЧЕСКИЕ ФЕНОТИПЫ И ПРОГНОСТИЧЕСКОЕ ЗНАЧЕНИЕ

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Аннотация. Артериальная гипертензия является одной из ведущих медико-социальных проблем в условиях глобального старения населения. С возрастом отмечается резкое увеличение распространенности гипертензии, изменение ее клинической структуры и усиление вклада в сердечно-сосудистую, цереброваскулярную и когнитивную заболеваемость. Настоящий обзор посвящен анализу возрастзависимых изменений артериального давления, патофизиологических механизмов гипертензии в пожилом и старческом возрасте, а также ее атипичных клинических форм, включая изолированную систолическую, псевдогипертензию, ортостатическую, офисную и маскированную гипертензию. Особое внимание уделено роли сосудистой жесткости, нарушений барорефлекторной регуляции и циркадных ритмов артериального давления. Обсуждается прогностическое значение гипертензии и гипотензивных состояний в отношении инсульта, сердечной недостаточности, деменции и смертности. Подчеркивается необходимость индивидуализированного подхода к диагностике и стратификации риска у пациентов старших возрастных групп.

Ключевые слова: артериальная гипертензия, пожилой возраст, изолированная систолическая гипертензия, сосудистая жесткость, ортостатическая гипотензия, деменция.

KEKSALAR VA JUDA KEKSA YOSHDAGI BEMORLARDA ARTERIAL GIPERTENZIYA: PATOFIZIOLOGIYA, KLINIK FENOTIPLAR VA PROGNOSTIK AHAMIYATI

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Annotatsiya. Arterial gipertenziya aholining global qarishi sharoitida eng muhim tibbiy va ijtimoiy muammolardan biri hisoblanadi. Yosh o‘tishi bilan gipertenziyaning tarqalishi keskin oshadi, uning klinik tuzilmasi o‘zgaradi va yurak-qon tomir, serebrovaskulyar hamda kognitiv kasallanishga qo‘shadigan hissasi kuchayadi. Ushbu sharh maqolada keksa va juda keksa yoshdagi bemorlarda arterial bosimning yoshga bog‘liq dinamikasi, gipertenziyaning patofiziologik mexanizmlari hamda uning atipik klinik shakllari, jumladan izolyatsiyalangan sistolik gipertenziya, psevdogipertenziya, ortostatik, ofis va maskirlangan gipertenziya tahlil qilinadi. Arterial qattiqlik, baroreflektor regulyatsiya buzilishlari va arterial bosimning sutkalik ritmlarining ahamiyatiga alohida e’tibor qaratilgan. Gipertenziya va gipotenziyali holatlarning insult, yurak yetishmovchiligi, demensiya va o‘lim xavfi bilan bog‘liqligi muhokama qilinadi. Keksa yoshdagi bemorlarda individual yondashuvga asoslangan diagnostika va xavfni stratifikatsiya qilish zarurligi ta’kidlanadi.

Kalit so’zlar: arterial gipertenziya, keksa yosh, izolyatsiyalangan sistolik gipertenziya, arterial qattiqlik, ortostatik gipotenziya, demensiya.

Introduction. Arterial hypertension represents one of the most prevalent chronic non-communicable diseases worldwide and constitutes a central determinant of cardiovascular morbidity and mortality, particularly in aging populations. The global demographic shift toward population aging has inevitably resulted in a disproportionate increase in the burden of hypertension among older and senescent individuals. Epidemiological data consistently demonstrate that the prevalence of hypertension rises sharply with advancing age, transcending racial, ethnic, and sex-related boundaries. In individuals over 60 years of age, arterial hypertension is diagnosed approximately twice as frequently as in middle-aged populations, while after 65 years of age more than two-thirds of individuals in developed countries exhibit persistently elevated blood pressure levels [9]. Longitudinal observations from the Framingham Heart Study further indicate that normotensive men and women at the age of 55 years have a lifetime risk exceeding 90% for developing hypertension by the age of 80, underscoring the near-inevitability of this condition in the context of biological aging [1].

Age-related elevation of blood pressure is widely regarded as an obligatory manifestation of vascular aging rather than a stochastic phenomenon. Systolic blood pressure demonstrates a continuous and monotonic increase across the adult lifespan, whereas diastolic blood pressure follows a nonlinear trajectory characterized by elevation until approximately the fifth decade of life and subsequent decline after the sixth decade. The divergence of these trajectories results in a progressive widening of pulse pressure, which serves as an indirect marker of large-artery stiffness and vascular remodeling [1,20]. This hemodynamic pattern reflects profound structural and functional alterations within the arterial tree, particularly within elastic conduit arteries such as the aorta.

The pathogenesis of age-related hypertension is multifactorial and encompasses complex interactions between intrinsic biological aging processes and modifiable environmental influences. Progressive fragmentation of elastin fibers, increased collagen deposition, medial calcification, and endothelial dysfunction collectively reduce arterial compliance and augment pulse wave velocity. The accelerated return of reflected pressure waves from peripheral arterial sites during systole, rather than diastole, leads to augmentation of systolic pressure and concomitant reduction of diastolic pressure, thereby increasing left ventricular afterload and compromising coronary perfusion [10]. These vascular changes are further exacerbated by age-associated dysregulation of neurohumoral systems, including impaired baroreflex sensitivity, heightened sympathetic nervous system activity, altered renin–angiotensin–aldosterone system signaling, and dysregulation of the hypothalamic–pituitary–adrenal axis [8].

Importantly, the contribution of arterial hypertension to adverse cardiovascular outcomes intensifies with advancing age. Elevated systolic and pulse pressures emerge as dominant predictors of myocardial infarction, ischemic and hemorrhagic stroke, chronic heart failure, atrial

fibrillation, chronic kidney disease, and both vascular and Alzheimer-type dementia [1,4,10]. In elderly populations, the prognostic significance of diastolic blood pressure undergoes inversion, such that excessively low diastolic values may reflect advanced arterial stiffness and are independently associated with increased mortality risk, particularly in the presence of elevated systolic pressure [6]. Large-scale meta-analyses involving more than one million individuals have demonstrated a continuous, log-linear relationship between blood pressure and cardiovascular mortality extending into advanced age, with stroke risk rising from systolic levels as low as 115 mmHg [7].

Isolated systolic hypertension constitutes the predominant hypertension phenotype in older adults and represents the extreme expression of age-related arterial stiffening. Defined by systolic blood pressure ≥140 mmHg in the presence of diastolic pressure <90 mmHg, this condition accounts for approximately two-thirds of hypertension cases in individuals over 60 years of age and nearly three-quarters of cases among those older than 75 years [53]. Isolated systolic hypertension is associated with a marked increase in cardiovascular mortality, with risk amplification particularly pronounced in elderly women [13]. Notably, arterial stiffness itself has emerged as an independent prognostic marker, underscoring the need to conceptualize vascular aging as a primary therapeutic target rather than merely a passive correlate of blood pressure elevation [4,9].

The clinical expression of hypertension in old age is further complicated by a spectrum of atypical and situational blood pressure phenotypes that are particularly prevalent in geriatric populations. Pseudohypertension, characterized by falsely elevated cuff-measured blood pressure due to non-compressible, calcified arteries, poses a significant diagnostic challenge and may lead to inappropriate intensification of antihypertensive therapy. Although the Osler maneuver has historically been proposed as a screening tool, its limited sensitivity and specificity preclude routine clinical application, leaving intra-arterial blood pressure measurement as the definitive diagnostic standard [5,7].

Orthostatic hypotension represents another frequent and clinically significant phenomenon in elderly individuals, defined by a sustained reduction in systolic blood pressure of at least 20 mmHg or diastolic pressure of at least 10 mmHg upon standing. Its prevalence increases markedly with age and comorbidity burden, reaching rates as high as 25–30% in very old populations [6]. The condition is often attributable to impaired autonomic regulation and is strongly associated with falls, syncope, cerebrovascular hypoperfusion, cognitive decline, and increased mortality [1,9]. Pharmacologically induced orthostatic hypotension is particularly common in geriatric patients exposed to polypharmacy, emphasizing the need for meticulous blood pressure monitoring during therapeutic titration [7].

Conversely, orthostatic hypertension, defined by a paradoxical rise in blood pressure upon standing, has recently garnered attention as a potential independent cardiovascular risk factor. This phenomenon is thought to reflect exaggerated sympathetic activation and has been associated with subclinical cerebrovascular disease and increased stroke risk in elderly hypertensive patients [3]. Similarly, postprandial hypotension, characterized by a significant decline in blood pressure following meals, is highly prevalent among older adults with isolated systolic hypertension and contributes to syncope and falls, particularly in the morning hours. Its pathophysiology involves splanchnic vasodilation mediated by gastrointestinal peptides and insulin-related mechanisms, compounded by impaired baroreflex compensation [2,3].

Age-related alterations in circadian blood pressure regulation further complicate risk stratification and management in elderly populations. Abnormal nocturnal blood pressure patterns, including non-dipping and extreme dipping phenotypes, are increasingly common with advancing age and are independently associated with target organ damage and cerebrovascular events. Elevated nocturnal blood pressure variability and exaggerated morning blood pressure surges have been identified as strong predictors of stroke and cardiovascular mortality in elderly cohorts [4,10].

Conclusion. Arterial hypertension in old age represents a heterogeneous, multifaceted clinical entity rooted in the biology of vascular aging and amplified by comorbidity, neurohumoral

dysregulation, and environmental factors. Its dominant systolic phenotype, high prevalence of atypical blood pressure patterns, and complex interaction with cognitive, renal, and cardiovascular outcomes necessitate a nuanced, individualized diagnostic and therapeutic approach. Recognition of the distinctive pathophysiological mechanisms and clinical manifestations of hypertension in elderly populations is essential for optimizing risk reduction strategies and improving long-term outcomes in this rapidly expanding demographic group.

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