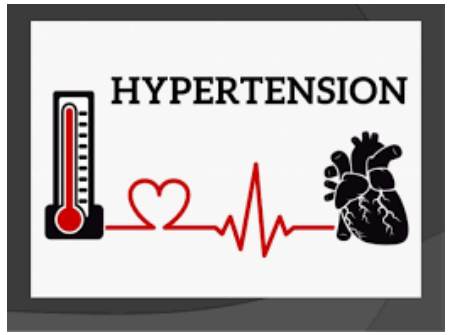

In The
Name Of
GOD



Risk Factors & Organ damages in hypertension

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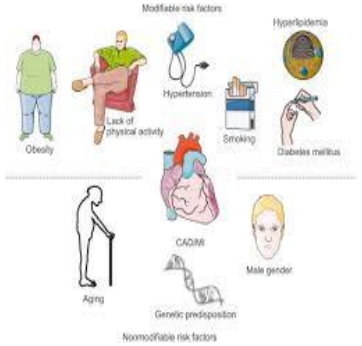
Risk factors for hypertension

- It is any attribute, characteristic or exposure of an individual that increases the likelihood of developing a disease or injury.

- Two types: modifiable & non-modifiable



Modifiable Risk Factors

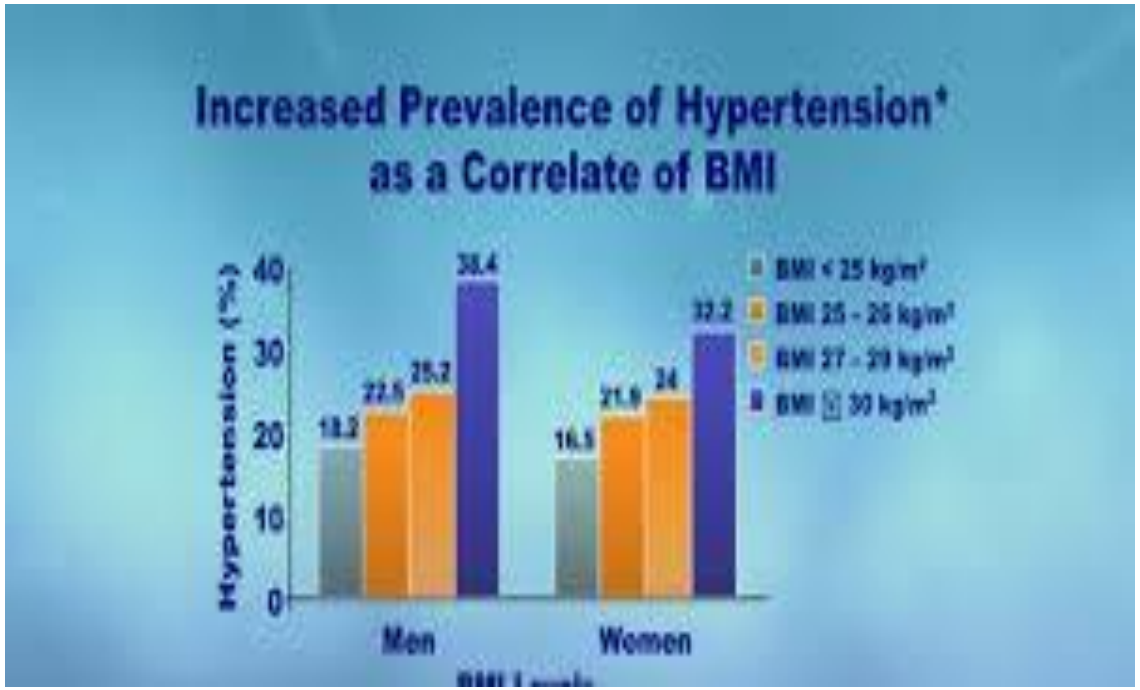
- Obesity
- Salt intake(>3 g/day Nacl)
- Potassium intake
- Alcohol
- Dyslipidemia
- Dietary fiber & Saturated fat
- Smoking
- Stress
- Physical activity
- Socio-economic status
- Personal history of CVD (MI, HF, stroke, TIA, DM, CKD)

Non-modifiable Risk Factors

- Age(>65 years)
- Gender(male)
- Genetic factors
- Ethnicity (non-white)
- Menopause (early-onset)
- Family history of HTN, premature CVD, (familial) hypercholesterolemia, DM

- Obesity – Obesity and weight gain are major risk factors for hypertension and are also determinants of the rise in blood pressure that is commonly observed with aging.
- Central obesity
- Linear correlation between BMI & BP

BMI less than 18.50	Underweight
BMI 18.50 -24.99	Healthy weight
BMI 25.00 -29.99	Overweight
BMI 30.00 or more	Obese


Increased Prevalence of Hypertension*
as a Correlate of BMI
Hypertension (%)
40
30
20
10
0
18.2
22.5
25.2
35.4
16.5
21.9
24
32.2
Men
Women
BMI < 25 kg/m²
BMI 25 - 26 kg/m²
BMI 27 - 29 kg/m²
BMI ≥ 30 kg/m²

PREVALENCE AND ASSOCIATED RISK FACTORS OF HYPERTENSION: A CROSS-SECTIONAL STUDY IN NORTH JAKARTA PUBLIC HEALTH CENTER

- This research was a cross-sectional study of 789 patients as primary data by measuring BMI and waist circumference with questionnaire encompassed identity and risk factor of hypertension. This research was held in the Public Health Center of Penjaringan District, North Jakarta.
- **Result:**

Research outcome showed that out of 789 respondents, 260 (33%) had pre-hypertension, 265 (33,6%) had hypertension stage 1, and 142 respondents (18%) had hypertension stage 2—only 122 (15,5%) respondents who had normal tension. Based on statistical analysis, there were some factors associated with hypertension, which were smoking, body mass index, and waist circumference. Chi-square test showed p value 0.008 between hypertension and smoking. Kruskal Wallis test showed p value of 0.020 between hypertension and body mass index, and p-value 0.002 between hypertension and waist circumference. The conclusion of this research was most of the respondents had hypertension. It also revealed that smoking, an increase in body mass index, and waist circumference became the risk factors of hypertension. This could be happened due to an unhealthy lifestyle. Therefore, recommendations should be made to enhance the monitoring of blood pressure regularly and improve health promotion of hypertension risk factors.

Journal of Hypertension: July 2020 - Volume 38 -

- Potassium supplements lower the BP of mild to moderate HTN.
- Alcohol increases systolic BP more than diastolic BP.

It contains calories and may contribute to weight gain.

- Stress can lead to temporary rise in BP because of increased level of catecholamine by sympathetic over activity.
- Chronic stress can lead to unhealthy behavior such as overeating, alcoholism that contributes to HTN.
- Physical activity : BP can be lowered by 30 min / day regular moderately intense activity such as brisk walking.

Other Additional Risk Factors

- Uric Acid is a Strong Risk Marker for Developing HTN from Prehypertension. It should be treated with diet, urate influencing drugs (losartan, fibrates, atorvastatin) or urate lowering drugs in symptomatic patients (gout with s-UA >6mg/dl [0.357 mmol/L]).
- An increase in cardiovascular risk must be considered in patients with HTN and chronic inflammatory diseases, COPD, psychiatric disorders, psychosocial stressors where an effective BP control is warranted.

Uric Acid and HTN: An Update With Recommendations

- The association between increased serum urate and HTN has been a subject of intense controversy. Extracellular uric acid drives uric acid deposition in gout, kidney stones, and possibly vascular calcification. Mendelian randomization studies, however, indicate that serum urate is likely not the causal factor in hypertension although it does increase the risk for sudden cardiac death and diabetic vascular disease. Nevertheless, experimental evidence strongly suggests that an increase in intracellular urate is a key factor in the pathogenesis of primary hypertension. Pilot clinical trials show beneficial effect of lowering serum urate in hyperuricemic individuals who are young, hypertensive, and have preserved kidney function. Some evidence suggest that activation of the renin-angiotensin system (RAS) occurs in hyperuricemia and blocking the RAS may mimic the effects of xanthine oxidase inhibitors. A reduction in intracellular urate may be achieved by lowering serum urate concentration or by suppressing intracellular urate production with dietary measures that include reducing sugar, fructose, and salt intake. We suggest that these elements in the western diet may play a major role in the pathogenesis of primary hypertension. Studies are necessary to better define the interrelation between uric acid concentrations inside and outside the cell. In addition, large-scale clinical trials are needed to determine if extracellular and intracellular urate reduction can provide benefit hypertension and cardiometabolic disease.

American Journal of Hypertension, 2020,18;33(7)

- Genetic factors & Family history : it is polygenic in inheritance.

Monozygotic > Dizygotic twins

HTN is about twice as common in subjects who have one or two hypertensive parents, and multiple epidemiologic studies suggest that genetic factors account for approximately 30 % of the variation in BP in various populations.

- Race : HTN tends to be more common, be more severe, occur earlier in life, and be associated with greater target-organ

damage in blacks.

- Reduced nephron number: Reduced adult nephron mass may predispose to hypertension, which may be related to genetic factors, intrauterine developmental disturbance (eg, hypoxia, drugs, nutritional deficiency), premature birth, and postnatal environment (eg, malnutrition, infections).

SECONDARY OR CONTRIBUTING CAUSES OF HYPERTENSION

- Prescription or over-the-counter medications : NSAIDs, OCP(estrogen) , Antidepressants (TCA, selective SRI, and MOI), Corticosteroids (GC & mineralocorticoids), Decongestants, Some weight-loss medications, Sodium-containing antacids, EPO, CSA & TAC, methamphetamines and cocaine ,…..
- Primary renal disease : Both acute and chronic kidney disease
- Renovascular hypertension
- Obstructive sleep apnea
- Primary aldosteronism , Cushing's syndrome, Pheochromocytoma
- Other endocrine disorders : Hypothyroidism, hyperthyroidism, and hyperparathyroidism
- Coarctation of the aorta

Role of the Immune System in Hypertension

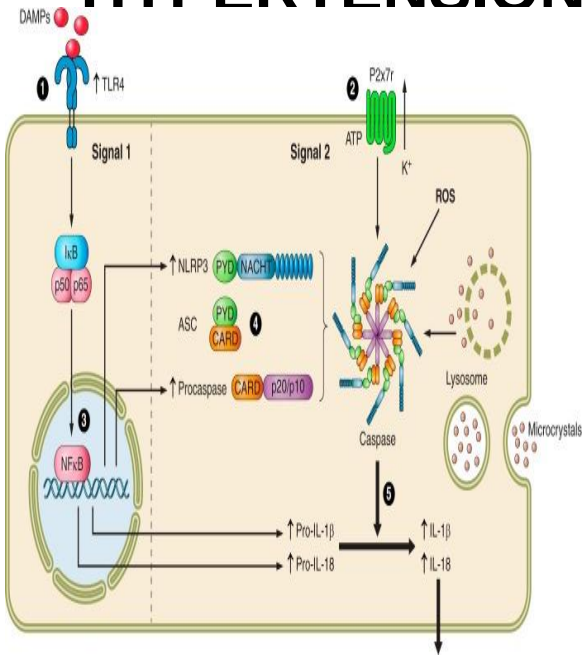
- Immunosuppressive drugs and inhibition of individual cytokines prevent or ameliorate experimental hypertension, and studies in genetically-modified mouse strains have demonstrated that lymphocytes are necessary participants in the development of hypertension and in hypertensive organ injury. Furthermore, immune reactivity may be the driving force of hypertension in autoimmune diseases. Infiltration of immune cells, oxidative stress, and stimulation of the intrarenal angiotensin system are induced by activation of the innate and adaptive immunity. High blood pressure results from the combined effects of inflammation-induced impairment in the pressure natriuresis relationship, dysfunctional vascular relaxation, and overactivity of the sympathetic nervous system. Imbalances between proinflammatory effector responses and anti-inflammatory responses of regulatory T cells to a large extent determine the severity of inflammation. Experimental and human studies have uncovered autoantigens (isoketal-modified proteins and heat shock protein 70) of potential clinical relevance. Further investigations on the immune reactivity in hypertension may result in the identification of new strategies for the treatment of the disease.

Physiol Rev. 2017 Jul 1; 97(3): 1127–1164.

Summary

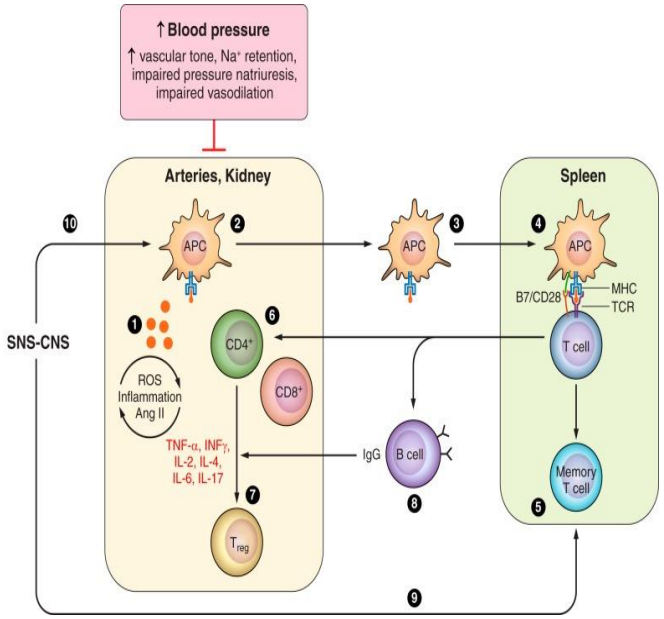
Immunosuppressive interventions associated with reduction of inflammation, improvement of oxidative stress, and reduction in renal angiotensin II activity have been shown to prevent, improve, or correct hypertension in genetic and experimentally induced models of hypertension. T and B lymphocytes, monocytes/ In macrophages, natural killer cells, and dendritic cells are the central cellular elements in immune-driven reactivity. Their participation in the pathogenesis of hypertension results from the activation of the innate and adaptive pathways of immune reactivity.

INNATE IMMUNITY IN HYPERTENSION

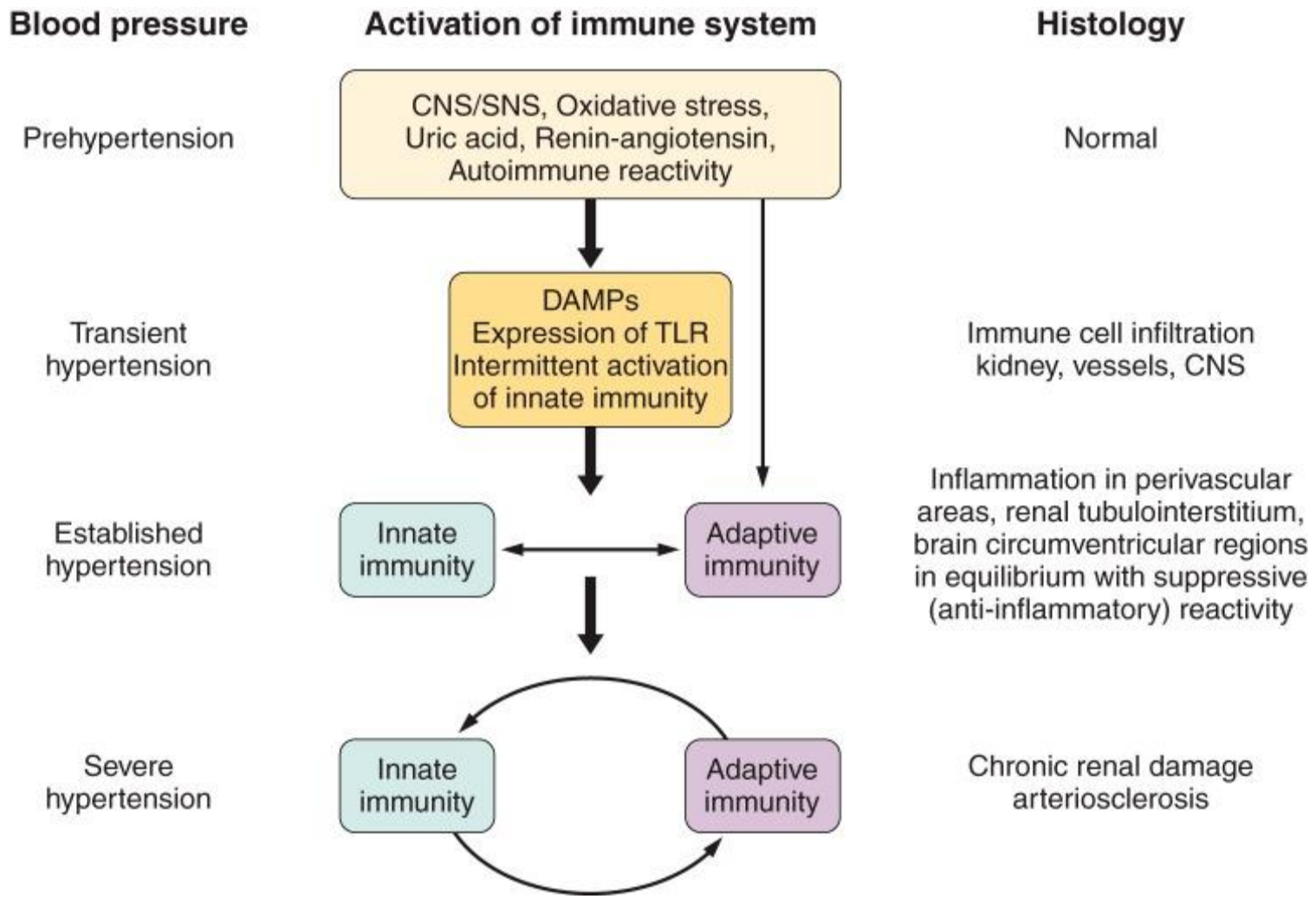


Participation of the NLRP3 inflammasome in the pathogenesis of experimental hypertension. *1*) Suppression or deficiency of TLR4 ameliorates or prevents hypertension (20, 46, 247). *2*) Inactivation or deficiency of the P2x7 receptor ameliorates hypertension in the Dahl SS rat (120). *3*) Suppression of NFκB activation ameliorates hypertension in SHR (218), Fawn Hooded rat (137), and the dTGF rat (185). *4*) Deficiency of ASC ameliorates DOCA-salt hypertension (140). *5*) NLRP3 inflammasome components overexpressed in the SHR (see FIGURE 2) and inhibition of inflammasome activation ameliorate DOCA-salt hypertension

Adaptive immunity in experimental HTN



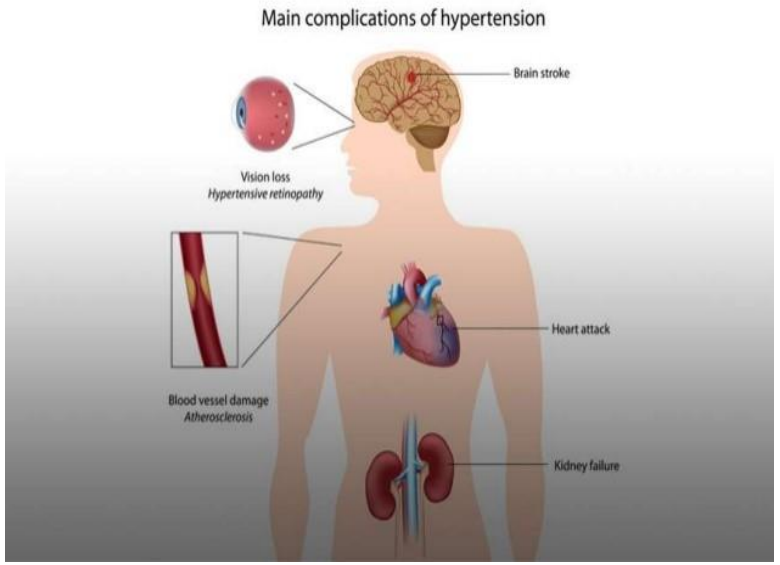
Investigations demonstrating involvement of adaptive immunity in experimental models of HTN include evidence that isoketal–modified proteins and overexpression of HSP70 are potential antigens in hypertension-associated immune reactivity (*1*). Dendritic cells process the antigen (*2*), travel to lymphoid organs (*3*), and present it to the TCR in T cells in the context of the MHC in association with costimulatory signals (*4*). Memory T cells (*5*) are developed and stored for inducing accelerated responses to subsequent antigenic challenge and activation and expansion of effector T cells (*6*) that result in proinflammatory cytokine responses and regulatory T cells (*7*). B-cell activation (*8*) is necessary for the development of hypertension when the immune system is intact .The CNS-SNS axis is recruited by oxidative stress involving angiotensin II receptors and results in SNS-induced stimulation of the release of activated T cells from the spleen (*9*) and stimulation of target organ immune infiltration and reactivity (*10*). APC, antigen presenting cells; MHC, major histocompatibility complex; TCR, T cell receptor; CNS, central nervous system; SNS, sympathetic nervous system.



Activation of the immune system and the natural history of essential hypertension. Prehypertension is associated with irregular generation of stimulatory signals associated with rise in blood pressure. Transient episodes of hypertension are associated with episodic generation of danger-associated molecular patterns (DAMPs) and expression of Toll-like receptors (TLRs) that activate intermittently the innate immune system with episodic inflammatory infiltration in target organs. Established hypertension results from the activation of both the innate and the adaptive immunity that support one another and drive a permanent renal and vascular inflammation that is, nevertheless, in a state of unsteady equilibrium with the suppressive (anti-inflammatory) responses. This balance is capable of maintaining a well-preserved renal function. The development of chronic renal damage and arteriosclerosis, resulting from persistent and increasing inflammation fueled by the unchecked generation of neoantigens, is manifested by hypertension of increased severity and resistance to treatment.

End organ damage in hypertension

- Hypertension-mediated organ damage (HMOD) is defined as the structural or functional alteration of the arterial vasculature and/or the organs it supplies that is caused by elevated BP. End organs include the brain, the heart, the kidneys, central and peripheral arteries, and the eyes.



The concentration of TNF in the blood serum and in the urine and selected early organ damages in patients with primary HTN

- Arterial hypertension is considered to be an inflammatory condition with low intensity. Therefore, an elevated concentration of inflammatory cytokines can be expected in patients with systemic arterial hypertension, including tumor necrosis factor (TNF).
- The study included a group of 96 persons aged 18 to 65 years: 76 patients with primary arterial hypertension and 20 healthy individuals (control group). Blood pressure was measured in all individuals using the office and ambulatory blood pressure monitoring (ABPM) measurement, blood was collected for laboratory tests [tumor necrosis factor (TNF), tumor necrosis factor receptor 1 (TNFR1)], and 24-hour urine collection was performed in which albuminuria and TNF concentration were assessed. Moreover, assessment of the intima-media thickness (IMT) in ultrasonography and left ventricular mass index (LVMI) in echocardiography were carried out.
- Statistically elevated TNF concentration in the blood serum ($P = .0001$) and in the 24-hour urine collection ($P = .0087$) was determined in patients with hypertension in comparison with the control group. The TNF and TNFR1 concentration in the serum and TNF in the 24-hour urine in the group of patients with arterial hypertension and organ damages and without such complications did not differ statistically significantly.
- We observed a positive and statistically significant correlation between TNFR1 concentration in the serum and TNF urine excretion in patients with hypertension ($r = 0.369$, $P < .05$)
- Patients with arterial hypertension are characterized by higher TNF concentrations in blood serum and higher TNF excretion in 24-hour urine than healthy persons.
- TNF and TNFR1 concentration in blood serum and TNF excretion in 24-hour urine in patients with early organ damages due to arterial hypertension do not differ significantly from those parameters in patients with arterial hypertension without organ complications.
- There is a positive correlation between TNFR1 concentration in the serum and TNF urine excretion in patients with hypertension.

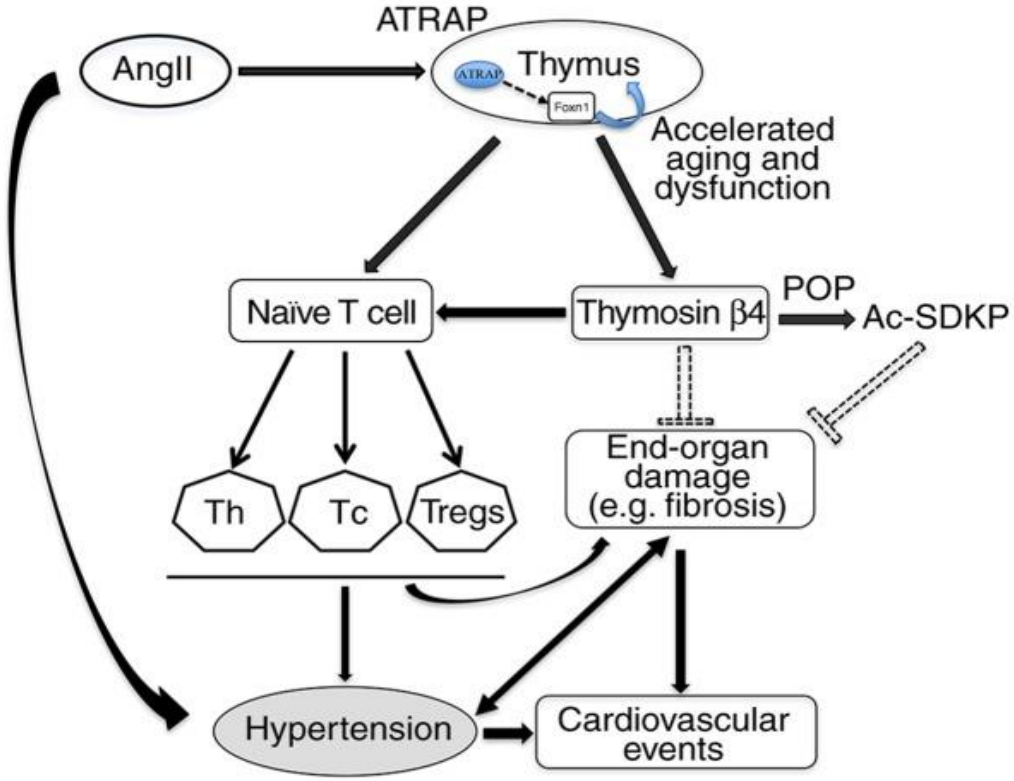
Medicine (Baltimore).
2019 May; 98(22)

Mechanisms in hypertension and target organ damage: Is the role of the thymus key?

- A variety of cells and cytokines have been shown to be involved in the whole process of hypertension. Data from experimental and clinical studies on hypertension have confirmed the key roles of immune cells and inflammation in the process. Dysfunction of the thymus, which modulates the development and maturation of lymphocytes, has been shown to be associated with the severity of hypertension. Furthermore, gradual atrophy, functional decline or loss of the thymus has been revealed to be associated with aging. The restoration or enhancement of thymus function via upregulation in the expression of thymus transcription factors forkhead box N1 or thymus transplantation may provide an option to halt or reverse the pathological process of hypertension. Therefore, the thymus may be key in HTN and HMOD, and may provide a novel treatment strategy for the clinical management of patients with hypertension in addition to different commercial drugs. The purpose of this review is to summarize and discuss the advances in our understanding of the impact of thymus function on hypertension from data from animal and human studies, and the potential mechanisms.

Int J Mol Med. 2018 Jul; 42(1): 3–12

Role of the thymus in hypertension and target organ damage.



Thymus dysfunction leads to the imbalance of T cell subsets and a change in the secretion of Tβ4, thereby aggravating the progression of hypertension and target organ damage, in addition to other cardiovascular events. Tβ4, thymosin β4; Ang II, angiotensin II; ATRAP, Ang II type 1 receptor-associated protein; Foxn1, forkhead box N1; POP, prolyl oligopeptidase; Ac-SDKP, N-acetyl-seryl-aspartyl-lysyl-proline; Th, T helper; Tc, cytotoxic T cell; Tregs, regulatory T cells.

Inflammatory and immune system mechanisms are crucial in the pathophysiology of hypertension and cardiovascular disease.

EOD

- Hypertensive cerebrovascular diseases

Hypertensive encephalopathy

CVA (ischemic , hemorrhagic)

- Hypertensive heart diseases

LVH

CAD

CHF

- Hypertensive artery diseases
- Hypertensive eye diseases

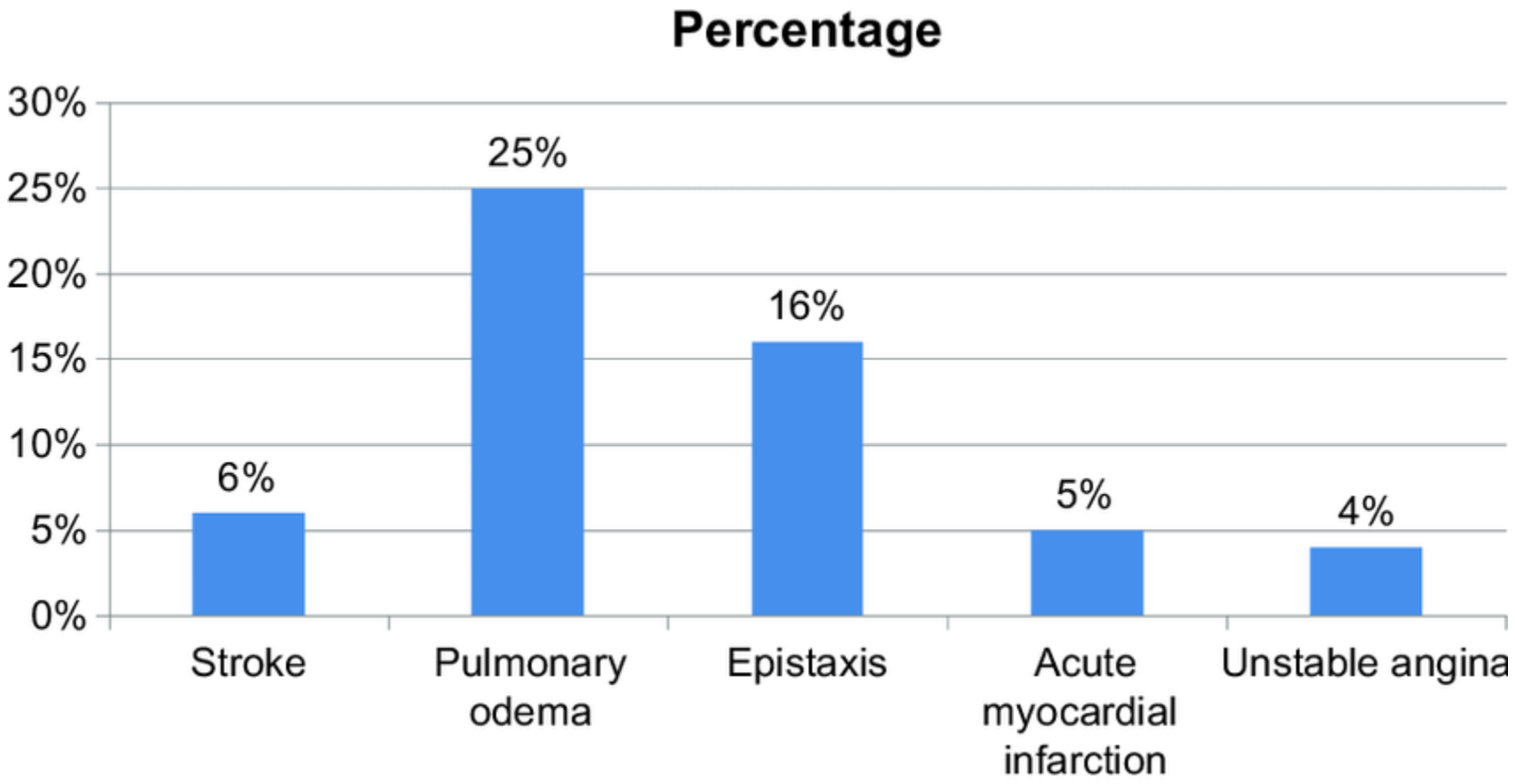
Retinopathy

- Hypertensive kidney diseases

Malignant nephrosclerosis

Benign nephrosclerosis

Percentage of end organ damage in patients admitted with hypertensive emergency



Tissue sodium content in hypertension and related organ damage

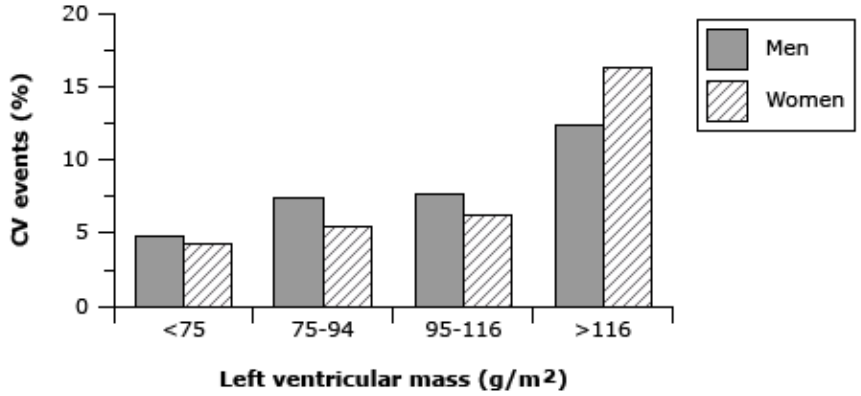
- Most textbooks state that sodium (Na) accumulation goes hand in hand with fluid retention to maintain the environmental isotonicity. In the last century, several studies found, however, that Na is stored in the extravascular space leading to an activation of the monocyte phagocytic system cells that work as a regulator of the interstitial electrolyte homeostasis. ^{23}Na-MRI was developed to quantify noninvasively, accurately and reliably tissue Na content. In this review, we give an up-to-date overview of clinical studies utilizing this ^{23}Na-MRI technique to elucidate the importance of tissue Na content in patients with cardiovascular risk factors leading to microvascular and macrovascular complications. Na storage leads ultimately to organ damage such as LVH or hypertrophic vascular remodeling of resistance vessels. Elevated Na content in muscle and skin has been detected in patients with treatment resistant hypertension, type 2 DM, acute and chronic heart failure, CKD and ESRD.
- Pharmacological interventions have shown that a mobilization of extracellular accumulated Na is possible and may emerge as a new therapeutic approach in some diseases.

Journal of Hypertension: July 27, 2020

Hypertensive Heart Diseases (HHD)

➢HHD are caused by the direct or indirect effects of elevated BP such as:

- LVH
- CAD
- CHF
- Cardiac arrhythmias



Simplified Classification of HTN Risk according to additional Risk Factors, (HMOD), and Previous Diseases

Other Risk Factors, HMOD, or Disease	High-Normal SBP 130–139 DBP 85–89	Grade 1 SBP 140–159 DBP 90–99	Grade 2 SBP ≥160 DBP ≥100
No other risk factors	LOW	LOW	Moderate High
1 or 2 risk factors	LOW	Moderate	High
≥3risk factors	Low Moderate	Moderate	High
HMOD, CKD grade 3, diabetes mellitus, CVD	High	High	High

*Example based on a 60 year old male patient. Categories of risk will vary according to age and sex.

• The therapeutic strategy must include lifestyle changes, BP control to target and the effective treatment of the other risk factors to reduce the residual cardiovascular risk. • The combined treatment of hypertension and additional cardiovascular risk factors reduces the rate of CVD beyond BP control

LVH

- LVH is more common in people who have uncontrolled high BP.
- LVH usually develops gradually.

➢Symptoms:

- Shortness of breath
- Fatigue
- Chest pain, often after exercising
- Palpitations
- Dizziness or fainting

Simple criteria for LVH

- Sokolow-Lyon index: SV1+RV5 ≥35 mm
- Cornell index: SV3+RaVL >28 mm for men or >20 mm for women
- A two-dimensional transthoracic echocardiogram (TTE) is the method of choice to accurately assess LVH (left ventricular mass index [LVMI]: men >115 g/m2 ; women >95 g/m2) and relevant parameters including LV geometry, left atrial volume, LV systolic and diastolic function and others.

Clinical features of CAD

- Chest pain (angina)
- Shortness of breath
- Heat attack (completely blocked coronary artery)

Cardiac arrhythmia

- Ventricular or supra-ventricular arrhythmia
- Bradycardia or tachycardia

➢Symptoms:

- Asymptomatic
- Dizziness
- Fainting
- Palpitation
- Lightheadedness
- Loss of consciousness

Symptoms of CHF

- Easy fatiguability
- Diminished exercise capacity
- Shortness of breath
- Edema

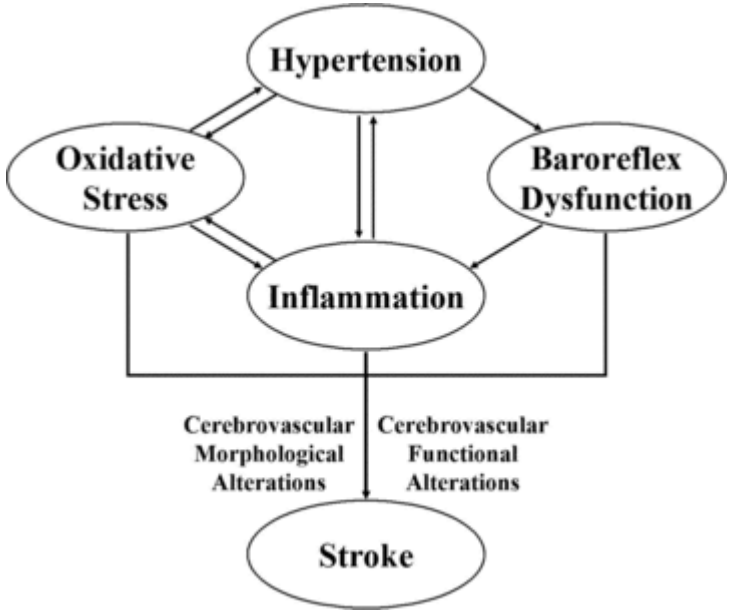
Hypertensive cerebrovascular diseases

- Hypertensive encephalopathy
- Cerebrovascular accidents

Hemorrhagic CVA(SAH, intracerebral)

Ischemic CVA(thrombotic & embolic)

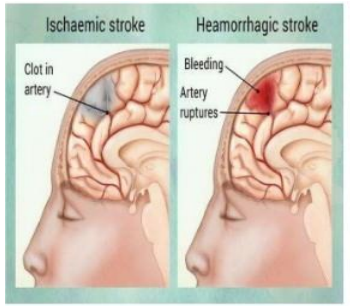
➢STROKES & TIA



Hypertensive encephalopathy

- It is a syndrome consisting of a sudden elevation of arterial pressure usually preceded by severe headache and followed by convulsions, coma or a variety of transitory cerebral phenomena.
- It may include: headache, vomiting, trouble with balance, confusion
- Onset is generally sudden
- Complication : seizures

CVA symptoms



- Muscular: paralysis with weak muscles, stiff muscles, overactivity reflexes, …
- Whole body: balance disorder, fatigue, light-headedness, vertigo
- Visual: double vision, blurred vision, sudden visual loss
- Speech: difficulty or speech loss, slurred speech
- Sensory: pins and needles or reduced sensation of touch
- Facial: numbness or muscle weakness
- Limbs: numbness or weakness
- Others: headache, difficulty swallowing, mental confusion,…

investigations

- Radiological tests
- Blood tests
- EEG
- MRI : white matter lesions, silent microinfarcts, microbleeds, and brain atrophy.

Blood pressure variability and hypertensive target organ damage

- **Objectives:**
- to investigate an impact of blood pressure variability (BPV) on heart and brain hypertensive damage in the middle-aged untreated patients with grade 1–2 essential arterial hypertension (EAH) without concomitant cardiovascular diseases.
- **Methods:**
- we examined 60 naive hypertensive patients (HP) with EAH and 44 sex- and age-matched healthy individuals. Comprehensive transthoracic echocardiography (Vivid 7 Dimension, GE) with 2D speckle tracking analysis and ambulatory blood pressure monitoring were performed in all participants. BPV was estimated as standard deviation. 41 HP underwent brain magnetic resonance imaging (MAGNETOM Skyra 3.0T, Siemens AG). Fazekas scale was used to quantify the amount of white matter hyperintensities (WMH).
- **Results:**
- nighttime systolic BPV (sBPV) and nighttime diastolic BPV (dBPV) were significantly ($p < 0,001$ for all) higher in HP (12,1 ± 0,1 vs. 9,3 ± 0,4 mmHg in controls and 9,8 ± 0,4 vs. 6,9 ± 0,3 mmHg in controls, respectively). Correlation analyses in HP revealed significant association of nighttime sBPV with left ventricular (LV) end-systolic elastance ($r = −0,375$; $p < 0,01$) and LV end-diastolic stiffness ($r = −0,429$; $p < 0,01$). Daytime dBPV significantly correlated with LV diastolic elastance ($r = 0,265$; $p < 0,05$), nighttime dBPV - with left atrial expansion index ($r = −0,282$; $p < 0,05$). WMH (Fazekas 1 and 2) were found in 53,7% HP patients. In HP with WMH 24 hours sBPV (19,7 ± 3,9 mmHg) and daytime sBPV (18,0 ± 4,5 mmHg) were significantly ($p < 0,05$ for all) higher compared to same parameters in HP without WMH (17,2 ± 3,3 and 15,7 ± 3,1 mmHg, respectively).
- **Conclusion:**
- middle-aged untreated patients with grade 1–2 uncomplicated EAH are different from the sex- and age-matched healthy individuals by higher nighttime BPV. BPV associated with heart and brain hypertensive organ damage.

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Hypertensive Retinopathy

- It is retinal vascular damage caused by HTN.
- Symptoms and signs usually do not develop until late in the disease.
- Symptoms: blurred vision, visual field defects
- Funduscopic EXM:

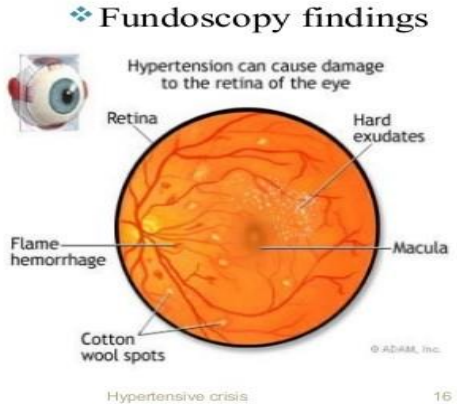
Fundoscopy should be performed in patients with grade 2 hypertension

AV nicking, vascular wall changes,

arteriolar constriction, flam shape hemorrhages,

cotton-wool spots, optic disk edema,

yellow hard exudates



Hypertensive Nephropathy (HN)

- It is a medical condition referring to damage to the kidney due to chronic high BP. The incidence of this condition follows closely the incidence and duration of high blood pressure throughout the population.
- Benign HN: It is common in individuals over the age of 60
- Malignant HN is uncommon and affects 1-5% of individuals with high blood pressure, that have D BP passing 130 mmHg.
- CKD & ESRD

Risk factors of HN

- 1. HTN
  2. DM(Type 1, Type 2)
- **Malignant Nephrosclerosis:**

1. Malignant hypertension (Diastolic BP > 130mm Hg)
2. Male gender
3. Pre-existing HTN
4. Pre-existing renal disease

- **Malignant nephrosclerosis:** Patients will usually have a pre-existing renal disease. At the onset of escalating blood pressure, protein and blood may appear in the urine. Hours to days later, renal function begins to diminish and the patient will quickly develop renal failure. This represents a medical emergency and every effort must be made to reduce blood pressure to preserve renal function.

Pathogenesis

Chronic high BP causes damages to kidney tissue including:

- The small vessels , glomeruli, kidney tubules & interstitial tissues.
- The tissue hardness & thickness which is known as nephrosclerosis.
- The narrowing of the blood vessels means less blood is going to the tissue and so less oxygen is reaching the tissue resulting in tissue death (ischemia).

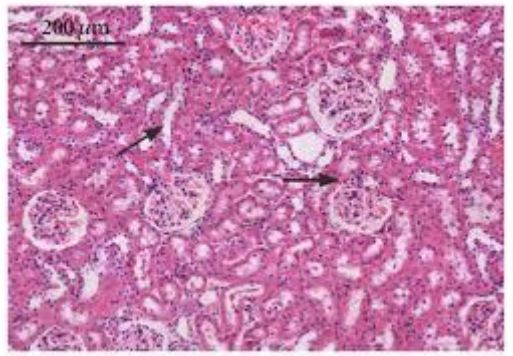
➢Glomerular ischemia

High BP damages the endothelium which leads to a build-up of plaques and eventual renal arteries stenosis with consequent ischemic kidney disease leading to a decrease in the size of the kidneys.

➢Glomerular HTN & glomerular hyperfiltration

Diagnosis

- Hematuria
- Proteinuria & Albuminuria
- Cr, eGFR
- Definitive diagnosis requires morphological EXM GS (focal or global)



Arteries

- Three vascular beds are commonly assessed to detect arterial HMOD:

(1) the carotid arteries through carotid ultrasound to detect atherosclerotic plaque burden/stenosis and intima media thickness (IMT)

(2) the aorta by carotid-femoral pulse wave velocity (PWV) assessment to detect large artery stiffening

(3) the lower extremity arteries by assessment of the ankle-brachial index (ABI).

- Although there is evidence to indicate that all three provide added value beyond traditional risk factors, their routine use is currently not recommended unless clinically indicated, that is, in patients with neurologic symptoms, isolated systolic hypertension, or suspected peripheral artery disease, respectively.

Association between common carotid artery diameter and target organ damage in essential hypertension

Abstract

Objectives:
To investigate the relationship between common carotid artery diameter (CCA-D) and target organ damage (TOD) in essential hypertension.
Methods and results:
A total of 200 essential hypertensive patients were enrolled (mean age 62.5 ± 9.5, men 59.0%) and were classified into two groups by the height-adjusted mean median of CCA-D: patients with CCA-D/height less than 3.905 mm/m (n = 100, 50%) and patients with CCA-D/height more than 3.905 mm/m (n = 100, 50%). Patients with CCA-D/height more than 3.905 mm/m have higher left ventricular mass index (LVMI) (P < 0.001) and higher prevalence of left ventricular hypertrophy (LVH) (P < 0.001), higher mean common carotid intima–media thickness (P = 0.008) and higher prevalence of carotid artery sclerosis (P = 0.03), higher pulse wave velocity (PWV) (P < 0.001) and higher prevalence of increased arterial stiffness (P = 0.01), higher urinary albumin/creatinine ratio (P = 0.001) and higher prevalence of microalbuminuria (P = 0.02) and greater number of TODs (P < 0.001) compared with the patients with CCA-D/height less than 3.905 mm/m. CCA-D was significantly correlated with LVMI, PWV and logarithmically transformed urinary albumin/creatinine ratio (r = 0.299, P < 0.001; r = 0.212, P = 0.007; r = 0.224, P = 0.005, respectively) after adjusting for cardiovascular risk factors. Multivariable stepwise linear regression analysis showed that number of TODs as well as individual TOD, including LVMI, PWV and logarithmically transformed urinary albumin/creatinine ratio, were independently correlated to height-adjusted mean CCA-D (all P < 0.05).
Conclusion:
Height-adjusted mean CCA-D was an independent risk factor for individual TOD, including LVMI, PWV and urinary albumin/creatinine ratio, as well as overall number of TODs in essential hypertension.

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EOD In Malignant HTN

- **Neurologic emergencies**:
- Ischemic stroke
- Hemorrhagic stroke
- Hypertensive encephalopathy
- **Cardiac emergencies:** The most common cardiac emergencies associated with severely elevated blood pressure are acute left ventricular dysfunction with pulmonary edema and acute coronary syndrome (including acute myocardial infarction).
- Acute heart failure
- Acute coronary syndrome
- **Vascular emergencies (**Acute aortic dissection)
- **Renal emergencies:** malignant nephrosclerosis
- **Retinopathy emergencies**


Thank
you