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"Control Hypertension: Add Years to Your Life "

Selected Abstracts from the Journal of Hypertension

The official journal of International Society of Hypertension ISH

High-normal blood pressure and abnormal left ventricular geometric patterns

Aim: The association between prehypertension (pre-HTN) and abnormal left ventricular (LV) geometric patterns is unclear. We performed a meta-analysis of echocardiographic studies aimed to provide a new piece of information on LV geometric alterations in untreated pre-HTN individuals.

Design: Studies were identified by crossing the following search terms: 'prehypertension,' 'high normal blood pressure,' 'heart,' 'LV hypertrophy' (LVH), 'LV geometry,' 'LV mass,' and 'echocardiography.'

Results: A total 60 949 participants (38 536 normotensive, 14 453 pre-HTN, and 7960 HTN individuals) were included in six studies. LV concentric remodelling was the most common abnormal pattern in pre-HTN participants followed by eccentric and concentric LVH. Compared with normotensive group, pre-HTN exhibited a higher risk of LV concentric remodelling (OR 1.89, CI 1.70–2.10, $P < 0.001$), eccentric LVH (OR 1.65, CI 1.40–1.90, $P < 0.001$) and concentric LVH (OR 2.09, CI 1.50–3.00, $P < 0.001$). The likelihood of having abnormal LV patterns in HTN was significantly higher ($P < 0.001$ for all) than in pre-HTN participants.

Conclusion: Our meta-analysis shows that alterations in LV geometry in pre-HTN individuals are intermediate between normotensive and HTN individuals. These findings suggest that pre-HTN is associated to early changes in LV structure and geometry, and preventive strategies may have a protective impact on development of subclinical cardiac damage in this setting.

Journal of Hypertension: July 2019 - Volume 37 - Issue 7 - p 1312–1319

The association between morning blood pressure and subclinical target organ damage in the normotensive population

Objective: To investigate whether isolatedly elevated morning blood pressure (BP) is associated with subclinical target organ damage in normotensive individuals.

Methods: In all, 287 normotensive individuals were included in this cross-sectional study. Each participant underwent anthropometric measurements, serum biochemistry evaluation, 24-h ambulatory BP monitoring, echocardiography, and carotid ultrasonography. The morning BP and morning surge were defined as: the average BP within 2 h after waking up, and the difference between the mean systolic BP (SBP) within 2 h after waking up and the mean SBP during the hour that included the lowest BP reading during sleep, respectively.

Results: The prevalence of elevated morning BP was 37.3%. Individuals with elevated morning BP had higher left ventricular mass index and morning surge, and also mean 24-h, daytime, and night-time SBP and diastolic BP, BP variability (all $P < 0.05$). Left ventricular mass index was correlated with 24-h, daytime, night-time, and morning SBP, and morning surge (Pearson's correlation coefficients: 0.271, 0.262, 0.215, 0.368, and 0.415, respectively; all $P < 0.05$); and standard deviations of 24-h, daytime, and night-time SBP (Pearson's correlation coefficient: 0.303, 0.234, and 0.309, respectively), and coefficient of variations of 24-h and night-time SBP (Pearson's correlation coefficients: 0.253 and 0.271, respectively). Morning surge had the strongest correlation with left ventricular mass index in multiple regression analysis. Only daytime and morning SBP could discriminate elevated morning surge (≥35 mmHg), with an area under the curve of 0.744 and 0.864, respectively (both $P < 0.01$), and an optimal threshold of 121.5 mmHg for morning SBP.

Conclusion: Our findings suggest that isolated elevation of morning BP in normotensive individuals is associated with left ventricular hypertrophy. Home monitoring of morning BP may be suitable for detecting abnormal morning surge.

Journal of Hypertension: July 2019 - Volume 37 - Issue 7 - p 1427–1436

Selected Abstracts from the Journal of Hypertension

The official journal of International Society of Hypertension ISH

Creatine kinase is associated with blood pressure during pregnancy

Objective: Hypertensive disorders in pregnancy pose a major burden during pregnancy and are also associated with an increased risk for hypertension later in life. Plasma creatine kinase activity is identified in the general population as an independent risk factor for hypertension. We hypothesize that plasma creatine kinase activity is similarly associated with blood pressure during pregnancy.

Methods: Women who participated in the 'Amsterdam Born Children and their Development-study' were eligible for the current study. The associations between plasma creatine kinase activity and blood pressure measurements during pregnancy, and between plasma creatine kinase activity and hypertensive disorders in pregnancy (gestational hypertension, HELLP, preeclampsia and eclampsia) were evaluated using multiple linear regression and logistic regression models.

Results: In 3619 pregnant women, plasma creatine kinase activity was significatly associated with all blood pressure outcomes. This was most pronounced for the mean SBP throughout pregnancy, with a regression coefficient of 3.48 mmHg (CI 1.67–5.28, P < 0.001) per 1-unit logCK. With respect to the hypertensive disorders in pregnancy, we found a significant association between severe gestational hypertension diagnosed before 34 weeks of gestation (OR 9.16, CI 1.32–63.86, P = 0.025) per 1-unit logCK activity. HELLP and preeclampsia were not significantly associated.

Conclusion: Our data show that plasma creatine kinase activity measured in early pregnancy is associated with blood pressure during pregnancy and associated with severe gestational hypertension diagnosed before 34 weeks of gestation, whereas no significant association was found between creatine kinase and other hypertensive disorders in pregnancy.

Journal of Hypertension: July 2019 - Volume 37 - Issue 7 - p 1467–1474

Recurrent vertigo is a predictor of stroke in a large cohort of hypertensive patients

Objective: Dizziness is associated with hypertension but there are numerous other causes. The aims of the present study were to describe the characteristics and the clinical correlates of dizziness in a large cohort of hypertensive patients, and to test its prognostic value for all-cause, cardiovascular, and stroke mortality.

Methods: A total of 1716 individuals from the OLD-HTA Lyon's cohort of hypertensive patients recruited in the 1970s were categorized according to the absence or the presence of dizziness. The dizziness group was subdivided into vertigo and other dizziness excluding vertigo.

Results: Multiple regression analysis demonstrated that presence of dizziness was predicted by age, female sex, coronary artery disease, and the absence of microalbuminuria. During 30 years of follow-up, we observed 956 deaths, 508 of which with a cardiovascular cause, and 114 fatal acute strokes. In the multivariate Cox regression model, the presence of dizziness had no impact on the risk for all-cause mortality [hazard ratio 0.91; 95% CI (0.78–1.06)], cardiovascular mortality [hazard ratio 0.86; 95% CI (0.70–1.05)], or stroke mortality [hazard ratio 1.27; 95% CI (0.85–1.90)]. In an analysis of the different subgroups of dizziness, only vertigo had a prognostic impact. The increased risk was particularly marked on stroke death with a hazard ratio of 2.43 (95% CI 1.33–4.46) vs. patients without dizziness and 2.22 (95% CI 1.21–4.06) vs. patients with dizziness excluding vertigo.

Conclusion: Hypertensive patients with dizziness did not have a high-risk profile at baseline, but those with vertigo must be carefully followed over years because of the higher stroke mortality.

Journal of Hypertension: May 2019 - Volume 37 - Issue 5 - p 942–948

Conclusions and Recommendations from the third International Conference of the Iraqi Society of Hypertension IqSH

The Iraqi Society of Hypertension, an affiliated International Society, held its successful third Annual International Conference on the beautiful city Sulaimaniya from the 1st to the 3rd of August this year and during the two conference days it tackled a lot of subjects that deal with the hypertension and its related cardiovascular diseases and discussed the recent advances and updated guidelines in the hypertension diagnosis, management and case presentation scenarios from the daily practices through the distinguished local and international speakers and discussion panelist . This year conference also witnessed the attendance and participations of doctors from the north of Iraq .

We can summarize some of its final conclusions and recommendations from its intense scientific program in the following short statements..

1.Hypertension should be considered as a community disease and all its management should be shared with non health and health givers especially the primary health centers ,media and civil society organizations.

2. Emphasize the importance of research and patients data registry as they are the first step in controlling and understanding the burden of any community based disease.

3. Hypertension should be looked at and managed as a whole and the focus should be extended on the secondary and aggravating factors especially obesity.

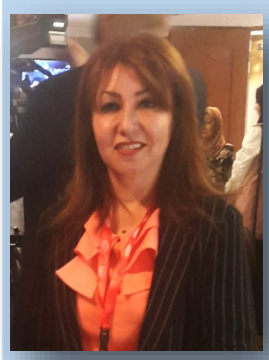
4.To increase the awareness of the possibility of secondary causes like obstructive sleep apnea (OSA) and endocrine diseases as a cause of secondary hypertension especially when dealing with cases of resistant hypertension.

5. Implementing the wide spread use of Ambulatory Blood Pressure Monitoring (ABPM) devices in the HT clinics as it is proved to be useful in the diagnosis of White coat and Masked hypertensions and also in the patient response to drug therapy.

Dr.
Raghdaa D. Sadeq

Hypertension in Pregnancy

Part 1



M.B.Ch.B. M.S community medicine

Manager of training & research department..Public health directorate

Hypertensive disorders are the most common medical complication of pregnancy and the major causes of maternal and perinatal disease and death worldwide. Hypertension affects 12 to 22% of pregnant. Hypertensive disease is directly responsible for approximately 20% of maternal mortality. In Iraq hypertension considered as the 3rd cause of death among female 6.2 in 2017 (annual report, MOH 2017).

Definition:

Hypertension in pregnancy is defined as:

1. Systolic blood pressure greater than or equal to 140 mmHg and/or
2. Diastolic blood pressure greater than or equal to 90 mmHg .

These measurements should be confirmed by repeated readings over several hours at least 6 hours apart, Proper cuff size, measurement taken while seated, Arm at the level of the heart & Use 5th Korotkoff sound.

Etiology:

The etiology of the condition is unknown, but there are some risk factors that increase the possibility of the disease .

Risk Factors:

Chronic renal disease	20:1
Chronic hypertension	10:1
Antiphospholipid antibody syndrome	10:1
Nulliparity	3:1
Age >40 years or <18 years	3:1

- Preeclampsia in a previous pregnancy
- Family history of pregnancy-induced hypertension
- Vascular or connective tissue disease
- Diabetes mellitus (pregestational and gestational).
- Multifetal gestation.
- High body mass index.
- Common in low income countries.

Prevention:

- dietary & life style modification
- Exercise
- Lower salt diet
- Calcium supplements
- Fish oil supplements
- Antioxidants
- use of low-dose aspirin, 50- 150 mg/day, usually 75 mg per day, from 12 weeks of pregnancy .
- Low molecular weight heparin.
- Eclampsia prevention = MgSO4.

Classification:

The hypertensive disorders can be classified into four types:

Gestational hypertension.

2) Preeclampsia and Eclamptic syndrome.

3) Preeclampsia superimposed on chronic hypertension.

4) Chronic hypertension.

Management:

- Assess gestational age
- Assess cervix
- Fetal well-being
- Laboratory assessment
- Rule out severe disease!!
- The ultimate cure is delivery.

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Renal Denervation Therapy (RDN): current perspective in resistant blood pressure control

Dr.
Samir M. Yousif

Internist and Echocardiography specialist

Systemic hypertension remains a major perpetuating cardiovascular disorder globally accounting for substantial mortality and morbidity. Elevated blood pressure from any cause is a precursor for the onset and progression of coronary artery disease, heart failure, cerebrovascular disease (including dementia), peripheral vascular disease, chronic kidney disease, and end-stage kidney disease. Despite significant advances in our understanding and management of hypertension, a large number of patients continue to have blood pressure levels above the recommended goals. Reasons for uncontrolled hypertension, despite effective therapy, are multifactorial, complex, and not fully understood. Several factors contribute to so-called resistant hypertension, one of which is poor compliance with medical therapy, and complementary therapeutic pathways for effective blood pressure control are needed. The seeming complexity of controlling hypertension despite optimal therapy has triggered much research and consideration of device-based interventional therapies, such as catheter-based renal denervation (RDN) and baro-receptor activation. To date, research interest and progress in the potential utility of RDN to treat hypertension has been phenomenal. A pathophysiological role of the sympathetic nervous system in the genesis of hypertension is well established. Before the current era of pharmacological inhibition of sympathetic nervous system, surgical sympathectomy was used to treat severe hypertension. However, surgical sympathectomy was fraught with intolerable adverse consequences such as postural hypotension and was therefore abandoned.

The sympathetic nervous system and the rationale for renal denervation (RDN)

The kidneys are innervated by efferent sympathetic nerves from the central nervous system and by afferent sympathetic sensory nerves from the kidneys to the hypothalamus. Efferent sympathetic activity has three major effects on the kidney: (I) increased renin secretion; (II) increased sodium reabsorption in the tubules; and (III) decreased blood flow to the kidneys. Together, all these mechanisms contribute to elevation of BP. The afferent nerves respond to renal injury and ultimately

cause an increase in central sympathetic outflow as well. In untreated hypertensive patients, a high level of sympathetic activation had been demonstrated. Thus, uncontrolled activation of the renal sympathetic nerves seems to play a role in the pathophysiology of essential HTN and therefore, decreasing the sympathetic outflow to the kidneys may potentially be associated with a decrease in systemic BP

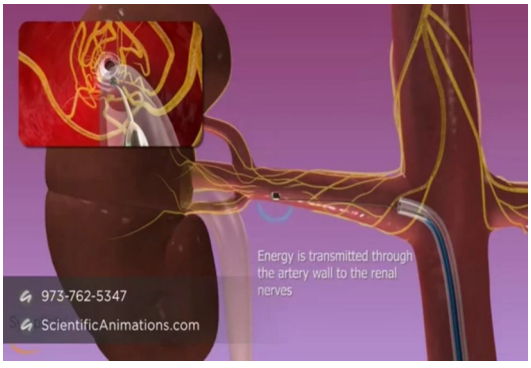
RDN Trials

The Symplicity HTN-1 was the first-in-man trial published in 2009. In this proof-of-concept trial, RDN therapy had been used amongst resistant HTN patients who were selected due to the potential clinical benefit . Later on, the Symplicity HTN-2 was a randomized-controlled, multicenter trial that enrolled 106 patients with resistant HTN and randomized them to RDN therapy plus medical management vs. medical management alone. This trial has shown a significant reduction in BP of approximately 32/12 mmHg in the study group patients as opposed to an increase of 1/0 mmHg in the control group measured 6 months after the procedure. The 3-year follow-up results of these two trials had shown long-term efficacy achieving a reduction in SBP of over 30 mmHg and in DBP of 13 mmHg as compared to baseline, and an overall good safety profile with only one recorded dissection and two cases of kidney injury which had resolved, however , Symplicity HTN-3 trial failed to

show a significant reduction in BP in patients with resistant HTN 6 months after RDN as compared to the sham-controlled arm .

The 12-month report reconfirmed these findings . The results of this trial had a detrimental effect on the prospect of RDN, yet other trials published at the time did manage to show the potential benefit of RDN therapy when attention was paid to the medication regimen and to ABPM measurements .

A number of reasons have been proposed to explain the lack of benefit from RDN in Simplicity HTN-3, such as incomplete denervation, the inexperience of the operators, improper selection of patients, and faulty design of the study itself. The failure of RDN therapy subsequently led to the improvement and redesign of the ablation catheters to provide (comprehensive) 4-quadrant bilateral renal nerve ablation of the main plus accessory renal arteries.These second-generation RDN catheters have yielded blood pressure effects with expectations reset to more realistic modest goals. The Radiosound-HTN comparative trial (Randomized Comparison of Ultrasound Versus Radiofrequency Denervation in Patients With Therapy Resistant Hypertension) showed that, in patients with resistant hypertension, RDN using endovascular ultrasound afforded superior reduction in ambulatory systolic blood pressure (SBP) in comparison with radiofrequency ablation of the renal arteries.



In conclusion The results of these studies, since they address the major misconceptions regarding RDN are empowered to provide the useful clinical information needed to resolve uncertainties for this still promising neuromodulation therapy. Making a long story short, in the near future the scientific community will have more data on answering the question on whether RDN is not only a safe but also an efficacious treatment modality in HT.

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New guidelines for diagnosis and management of hypertension

Dr. Abdulwahab S.A. Abutabiekh

Consultant physician at Alimamain Alkadymain teaching Hospital

MBChB, DM, MRCP

Hypertension is one of the most common worldwide diseases affecting humans and is a major risk factors for stroke, myocardial infarction, vascular disease and chronic kidney disease. Control blood pressure is still suboptimal in the general population due to the associated morbidity and mortality and cost to the society.

Preventing and treatment of hypertension is an important public health challenge. According to the American Heart Association(AHA) approximately 86 million adult population are hypertensive(34% of the population).

Hypertension is defined as a systolic blood pressure 140mmHg and more and diastolic blood pressure 90mmHg and more. Based on recommendation of the 7th report of Joint National Committee(JNC7) classification of blood pressure in adult aged 18 years or older as follows:

- Normal: systolic blood pressure less than 120 and diastolic less than 80.
- Prehypertension: systolic 120 to 139 and diastolic 80 to 89.
- Stage1 hypertension: systolic 140 to 159 and diastolic 90 to 99.
- Stage2 hypertension: systolic 160 and greater and diastolic 100 and greater.

On 2017 ACC/AHA guideline divide classification of prehypertension in two levels:

- Elevated blood pressure systolic 120 to 129 and diastolic less than 80.
- Stage1 hypertension systolic 130 to139 and diastolic 80 to 89.

Management of hypertension

Most guidelines including JNC, American Diabetic Association(ADA)and American Heart Association(AHA) recommend lifestyle modification as first step in managing hypertension which include:

- Weight loss reduction by 10kg reduce systolic blood pressure by 5 to 20mm.
- Limit alcohol intake to no more than 30cc ethanol per day for men and 15cc for women.
- Reduce sodium intake to no more than 100mmol per day(3g). Maintain adequate intake of potassium 90mmol per day.
- Adequate intake of calcium and magnesium.
- Stop smoking and reduce dietary saturated fat.
- Engage in Aerobic exercise at least 30minutes daily.

European Society of Cardiology(ESC) and European Society of Hypertension(ESH) at 2018 recommend low sodium diet limited to 2g per day and reducing body mass index(BMI) to 20 to 25kg/m2 and waist circumference to less than 94cm in men and less than 80cm in women.

Pharmacological therapy

If a lifestyle modification is insufficient to achieve the goal blood pressure or the patient develop end organ damage the drug therapy should be commenced which includes (Thiazide diuretic, ACE inhibitor, ARB, CCB). Calcium channel blockers and Thiazide diuretics are preferred for black populations.The following are drug class recommendations for compelling indications based on various clinical trials:

- Heart failure: Diuretics, Beta blockers, ACE inhibitors/ARBs, Aldosterone antagonist.
- Following myocardial infarcation1: Beta blocker and ACE inhibitor.
- Diabetes mellitus: ACE inhibitor/ARB.
- Chronic Kidney disease: ACE inhibitor/ARB, Calcium channel blocker.

If blood pressure is not controlled on monotherapy, combination therapy is recommended and usually is needed in stage2 hypertension (systolic blood pressure more than 160and diastolic more than 100).

In chronic kidney disease, it's usually recommended to reduce blood pressure to less than 130/80.

In hypertensive patient older than sixty years, treatment of hypertension is started when blood pressure exceeding 150/90.

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What's New in Cigarette smoking and hypertension?

Dr.
Waleed M. Ali

Consultant of internal medicine, Baba GurGur Diabetic Center, Northern Oil Compay,Kirkuk, Iraq

The use of tobacco as cigarettes has created the epidemic of lung and cardiovascular diseases that dominate the current disease manifestations of cigarette smoking. Elevated blood pressure and tobacco smoking are, respectively, the first and second leading causes of preventable mortality worldwide [1].Throughout the world, the absolute number of tobacco smokers has increased because of population growth, despite a decrease in the age-adjusted prevalence of smoking [1]. In the United States, the estimated number of tobacco smokers has dropped, as a result of tobacco-control efforts, from 45.1 million smokers in 2005 to 36.5 million smokers in 2017(Great American Smoke out. 2017). However, while use of tobacco products has decreased, use of electronic cigarettes (e-cigarettes) has increased substantially in the United States, along with concerns about their potential health risks [2]. Tobacco smoking and e-cigarette use may raise blood pressure and accelerate atherothrombotic processes though a variety of potential mechanisms, including harmful effects on endothelial function, inflammation, lipids, and thrombosis. The acute effects of tobacco smoking are related to sympathetic nervous system overactivity , which leads to an increase in blood pressure, heart rate, myocardial contractility, and myocardial oxygen consumption Regarding chronic effect of tobacco smoking ,there are conflicting data that tobacco smoking raises blood pressure chronically or increases the incidence of hypertension. Despite uncertain chronic effects on blood pressure, tobacco smoking increases the risk of masked hypertension , renovascular hypertension, severe hypertensive retinopathy, and arterial stiffness

E-Cigarettes and blood pressure

Electronic cigarettes (e-cigarettes) are products that deliver a nicotine-containing aerosol (commonly called vapor) to users by heating a solution typically made up of propylene glycol or glycerol (glycerin), nicotine, and flavoring agents invented in their current form by Chinese pharmacist Hon Lik in the early 2000s. There has been a rapid increase in use of electronic cigarettes (e-cigarettes), but data on their long-term health effects are sparse. Acute effects of e-cigarettes on blood pressure are inconsistent. A report from the National Academy of Medicine that summarized findings from a variety of studies concluded that e-cigarettes are associated with a mild increase or no effect on systolic blood pressure [2]. By contrast, some studies have documented short-term increases in diastolic blood pressure following the use of e-cigarettes that deliver nicotine; the magnitude of this effect was similar to the increases observed from

tobacco smoking. Data are limited on the chronic blood pressure effects of e-cigarettes. One retrospective, case-control study of 89 hypertensive tobacco smokers reported that systolic and diastolic blood pressure were significantly lower at 12 months among those who switched to e-cigarettes [3].

Cigarette smoking is a powerful cardiovascular risk factor and smoking cessation is the single most effective lifestyle measure for the prevention of a large number of cardiovascular diseases.

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Exercise: A magic prescription for hypertension

Dr.
Abdulkareem
Abdulwahab Al-Othman

FRCP (Galsg),FICMS,MB.Ch.B

Professor of Medicine

Dept. of Medicine

Hawler Medical University

Erbil-Kurdistan,Iraq

Hypertension is one of the most common causes for ischemic heart diseases, heart failure, stroke and chronic kidney disease. Exercise, weight loss, healthy eating, smoking cessation and reduced sodium intake represents the first line in treatment of primary hypertension.

Recent international studies indicate that different types of exercise in a regular daily pattern leads to reduction in blood pressure among non-hypertensive, hypertensive as well as in those who suffer from resistant hypertension.

You have to take a mandatory different types of exercises in a regular daily pattern like brisk walking for a half hour in the morning each day (Outside or inside on a treadmill), swimming and riding your bicycle. Strengthening exercises such as lifting weights (according to the ability of each person and gender), exercise to the legs, upper arm and abdomen.

Before engagement in a regular exercise you should consult your doctors in the following conditions:

If you have chronic health condition, such as diabetes, cardiovascular disease or lung disease.

If you've had a heart attack.

If you have a family history of heart-related problems before age 55 in men and age 65 in women.

If you feel pain or discomfort in your chest, jaw, neck or arms.

If you feel dizzy.

If you're unsure you're in good health

Stop exercising and seek immediate medical care if you experience any warning signs during exercise, including

Chest, neck, jaw or arm pain or tightness.

Severe shortness of breath

An irregular heartbeat

References:

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A link between hypertension and Insulin

Assistant Professor
Dr. Affan Ezzat

Physiology Department/ College of Medicine/Baghdad University

Insulin is an anabolic hormone that plays an important role in the regulation of glucose, lipid homeostasis and energy storage through its metabolic effects on classic insulin-responsive tissues . Vascular protective effects of insulin, including induction of vasorelaxation, and anti-inflammation, are mediated by stimulating nitric oxide-dependent (NO) mechanisms in the endothelium [1]. In addition, insulin increases sodium reabsorption in the kidney and promotes sympathetic nerve activity [2] in physiological condition insulin stimulates endothelial NO production to exert a vasorelaxation and anti-inflammatory effect. Whereas, in the state of insulin resistance, the insulin stimulated NO pathway is selectively impaired and resulting in enhancement of vasoconstriction, proinflammation, increased sodium and water retention and the elevation of blood pressure [3]. The interaction between insulin resistance and hypertension can be viewed either as a non-causal association (two independent processes) or as reflecting a cause-and-effect relationship (insulin resistance as a cause of hypertension). In the non-causal association, both insulin resistance and hypertension may represent two independent consequences of the same cellular disorder, that is, an increase in intracellular free calcium, which can result in both vasoconstriction and impaired insulin action. Furthermore, it is plausible to consider insulin resistance as a molecular marker of multiple metabolic abnormalities frequently associated with hypertension. Alternatively, hyperinsulinemia can be viewed as a major player in the genesis of hypertension via several mechanisms. These include increased sodium reabsorption in the kidney tubules, activation of the sympathetic nervous system, and alteration in vascular resistance though increased calcium concentration in smooth muscle cells[4].

Wang , Han and Hu D,2017 in their study suggest that elevated fasting insulin concentrations or insulin resistance as estimated by homeostasis model assessment is independently associated with an exacerbated risk of hypertension in the general population. Early intervention of hyperinsulinemia or insulin resistance may help clinicians to identify the high risk of hypertensive population [5]. Also, Stamatelopoulos *et al.2019,*concluded that A significant proportion of women entering the menopause present incident hypertension and this is mostly associated with obesity and insulin resistance[6]. The best approach to treating patients with insulin resistance and hypertension requires long-term studies aimed at addressing the risk factors (insulin resistance, hypertension, and so on) and optimizing clinical outcomes[4].

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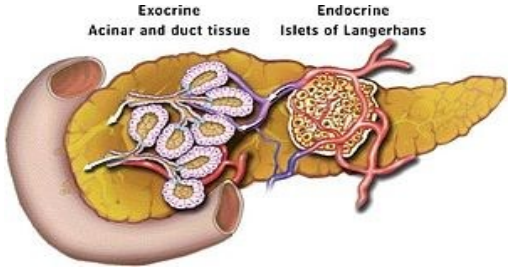

ارتباط ارتفاع ضغط الدم الانتصابي مع الوفيات في دراسة ارتفاع ضغط الدم الانقباضي في

د. سمير مجيد - اختصاصي الباطنية وايكو القلب

عند الوقوف ، تتجمع نسبة كبيرة من حجم الدم في الساقين والدورة الحشوية أسفل البطن. يتم موازنة هذه التغييرات عن طريق تنشيط الجهاز العصبي اللاإرادي بما في ذلك زيادة في التدفق الودي المركزي. الأفراد الأكبر سنا قد يكون حجم الدم لديهم أقل داخل الأوعية الدموية ، قصور وريدي ، وضعف منعكسات الضغط baroreflexes . ونتيجة لذلك ، فليس من غير المألوف ان يحدث انخفاض في الضغط الدم الانتصابي (oHypo)، ويرتبط مع نتائج سريرية أسوأ بما في ذلك احتشاء عضلة القلب ، والسكتة الدماغية ، وفشل القلب ، وضعف الإدراك ، والوفيات ارتفاع ضغط الدم الانتصابي (oHyper)،وهو زيادة في ضغط الدم الانقباضي (SBP)عند الوقوف ، يحدث أيضًا عند الأفراد الأكبر سناً. أفاد Townsend ورفاقه أن oHyper وoHypo قد حدثا بمعدلات مماثلة (حوالي 5 ٪) بين 8662 مشاركًا في دراسة SPRINT يمثل ارتفاع ضغط الدم الانتصابي حالة مهمة سريريًا لوحظت أيضًا في مرضى السكري ، ومرض باركنسون ، وتشوهات الأوعية الدموية الكلوية ، واعتلال الأعصاب اللاإرادية. تتضمن ارتباطات oHyper مع تلف الأعضاء النهائية مثل أمراض القلب التاجية ، وأمراض الأوعية الدماغية ، ومع درجات أقل في اختبارات الوظيفة العصبية السلوكية ، وظواهر المادة البيضاء white matter hyperintensities الأكثر تقدماً في التصوير المقطعي أو التصوير بالرنين المغناطيسي ، وعدم التحمل الانتصابي orthostatic intolerance، وأمراض الكلى المزمنة.

لا يتم عادة تقدير ارتفاع ضغط الدم الانتصابي من قبل الأطباء ، وهناك بيانات ضئيلة عن النتائج السريرية طويلة الأجل من الدراسات الوهمية المسيطر عليها . تم تعريف التغيير الانتصابي لـ SBP على أنه الفرق بين متوسط قياسين في وضع الجلوس و الضغط النقباضي SBP بعد الوقوف لمدة دقيقة واحدة في الزيارة الأساسية. وباستخدام قياس الثلاث دقائق أسفر عن نتائج مماثلة. تم تصنيف التغير الانتصابي من موضع الجلوس إلى الوقوف على أنه (1) ارتفاع ضغط الدم oHyper)، زيادة SBP بمقدار ≥15 مم زئبق) ،(2) انخفاض ضغط الدم oHypo)، انخفاض SBP بمقدار> 20 مم زئبق) ، أو(3) طبيعي oNorm)، تغييرات في SBP بين الفئتين الأخريين)

تأثير التغيير الانتصابي على وفيات جميع الأسباب

بعد تعديل العمر والجنس وخط الأساس SBP لضغط الدم الانقباضي وخط الأساس لضغط النبضة PP، ارتبط oHyper بارتفاع معدل الوفيات لجميع الأسباب عند 4.5 سنوات (HR 1.87)، 95٪ CI 1.30-2.69، (P = 0.0007) وفي 17 عامًا (HR 1.40)، 95) ٪ CI 1.17–1.68، p = 0.0003).بقيت الرابطة ذات دلالة إحصائية بعد التعديل لمعدل الكرياتينين في الدم والسكري ومؤشر كتلة الجسم وحالة التدخين وفشل البطين الأيسر والكوليسترول الحميد ، وكذلك العمر والجنس و وضغط الدم الانقباضي الاساسي ، وخط الأساس لضغط النبضة PP في 4.5 سنوات (HR 1.43)، 95٪ CI 0.99–2.08، (P = 0.0566) وفي 17 عامًا (HR 1.27)، 95٪ CI 1.06-1.53، P = 0.0096).

النقاش

معدل ارتفاع ضغط الدم الانتصابي في (4.3% SHEP) يشبه معدلات ذكرت سابقا. كان المشاركون الذين يعانون من ارتفاع ضغط الدم الانتصابي كبار السن ، مع ارتفاع SBP ومؤشر كتلة الجسم ، وكانوا أكثر احتمالا بان يكون عندهم تاريخ لفشل البطين الأيسر. ارتبط ارتفاع ضغط الدم الانتصابي مع الوفيات الناجمة عن جميع الأسباب بعد التعديل بالنسبة للعمر والجنس و SBP الأساسي و وخط الأساس لضغط النبضة . PP ظلت العلاقة ذات دلالة إحصائية بعد تعديل إضافي لعوامل الخطر والأمراض المصاحبة. على الرغم من أن جميع المشاركين في SHEP لا يقل عمرهم عن 60 عامًا (معدل 72 عامًا) ، إلا أن زيادة العمر ارتبطت بزيادة المخاطر.

آليات ارتفاع ضغط الدم الانتصابي والأسباب المرتبطة به مع زيادة معدل الوفيات ليست مفهومة تماما. ارتفاع ضغط الدم الانتصابي قد يكون بسبب شذوذات منعكسات المستقبلات وزيادة نشاط الأدرينرجين الوعائي وزيادة حساسية ردود الفعل المستقبلة للقلب والشرايين وردود الفعل وزيادة النشاط الودي ، وكذلك تصلب الشرايين المركزية.

ما هو معروف عن هذا الموضوع

1 - ارتفاع ضغط الدم الانتصابي ، زيادة في SBP عند الوقوف ، يحدث في حوالي 5 ٪ من المرضى الذين يعانون من ارتفاع ضغط الدم.

2- ارتفاع ضغط الدم الانتصابي هو أحد عوامل الخطر لأمراض القلب والأوعية الدموية ويرتبط مع تلف الاعضاء النهائية وحوادث القلب والاوعية الدموية.

3- لا يتم عادة تقدير ارتفاع ضغط الدم الانتصابي من قبل الأطباء ، وهناك بيانات ضئيلة عن الوفيات طويلة الأجل من البحوث المسيطر عليها وهميا.

4- يتم اكتشاف ارتفاع ضغط الدم الانتصابي بسهولة ويمكن استخدامه في تقييم مخاطر القلب والأوعية الدموية بشكل دقيق.

ما الجديد في تدخين السجائر وارتفاع ضغط الدم؟

د . وليد علي -استشاري الامراض الباطنية - مركز بابا كركر للسكري - كركوك

أدى استخدام التبغ كسجائر إلى انتشار وباء أمراض الرئة والقلب والأوعية الدموية التي تهيمن على مظاهر المرض الحالية المتمثلة في تدخين السجائر. يعد ارتفاع ضغط الدم وتدخين التبغ ، على التوالي ، السببين الرئيسيين الأول والثاني للوفيات التي يمكن الوقاية منها في جميع أنحاء العالم. في جميع أنحاء العالم ، زاد العدد المطلق لمدخني التبغ بسبب النمو السكاني ، على الرغم من انخفاض معدل الانتشار حسب العمر التدخين . في الولايات المتحدة ، انخفض العدد التقديري لمدخني التبغ ، نتيجة لجهود مكافحة التبغ ، من 45.1 مليون مدخن في عام 2005 إلى 36.5 مليون مدخن في عام 2017 (Great American Smoke out. 2017). ومع ذلك ، في حين انخفض استخدام منتجات التبغ ، زاد استخدام السجائر الإلكترونية (السجائر الإلكترونية) زيادة كبيرة في الولايات المتحدة ، إلى جانب المخاوف بشأن مخاطرها الصحية المحتملة . قد يؤدي تدخين التبغ واستخدام السيجارة الإلكترونية إلى رفع ضغط الدم وتسريع عمليات تصلب الشرايين على الرغم من مجموعة متنوعة من الآليات المحتملة ، بما في ذلك الآثار الضارة على وظيفة بطانة الأوعية الدموية والالتهابات والدهون والتخثر. ترتبط الآثار الحادة لتدخين التبغ بفرط نشاط الجهاز العصبي الودي ، مما يؤدي إلى زيادة في ضغط الدم ومعدل ضربات القلب وانقباض عضلة القلب واستهلاك الأوكسجين في عضلة القلب فيما يتعلق بالتأثير المزمن للتدخين ، هناك بيانات متضاربة تفيد بأن تدخين التبغ يرفع ضغط الدم بشكل مزمن أو يزيد من حدوث ارتفاع ضغط الدم. على الرغم من الآثار المزمنة غير المؤكدة على ضغط الدم ، فإن تدخين التبغ يزيد من خطر ارتفاع ضغط الدم المقنع وارتفاع ضغط الدم الوعائي واعتلال الشبكية وارتفاع ضغط الدم الحاد وتصلب الشرايين.

السجائر الإلكترونية وضغط الدم

السجائر الإلكترونية (السجائر الإلكترونية) هي المنتجات التي توفر الهباء الجوي المحتوي على النيكوتين (المعروف باسم البخار) للمستخدمين عن طريق تسخين محلول يتكون عادة من البروبيلين جليكول أو الجلسرين (الجلسرين) والنيكوتين وعوامل النكهة التي تم اختراعها في شكلها الحالي بواسطة الصيدلي الصيني هون ليك في أوائل 2000 .s كانت هناك زيادة سريعة في استخدام السجائر الإلكترونية (السجائر الإلكترونية) ، ولكن البيانات حول آثارها الصحية طويلة الأجل قليلة. الآثار الحادة للسجائر الإلكترونية على ضغط الدم غير متناسقة. خلص تقرير صادر عن الأكاديمية الوطنية للطب يلخص النتائج المستخلصة من مجموعة متنوعة من الدراسات إلى أن السجائر الإلكترونية مرتبطة بزيادة خفيفة أو أي تأثير على ضغط الدم الانقباضي . على النقيض من ذلك ، فقد وثقت بعض الدراسات زيادات قصيرة الأجل في ضغط الدم الانبساطي بعد استخدام السجائر الإلكترونية التي توفر النيكوتين ؛ كان حجم هذا التأثير مشابهًا للزيادات التي لوحظت من تدخين التبغ. البيانات محدودة حول تأثيرات ضغط الدم المزمن للسجائر الإلكترونية. ذكرت إحدى الدراسات الاستعادية ومراقبة الحالات لـ 89 من مدخني التبغ المصابين بارتفاع ضغط الدم أن ضغط الدم الانقباضي والانبساطي كان أقل بشكل ملحوظ في 12 شهرًا بين أولئك الذين تحولوا إلى السجائر الإلكترونية .

يعد تدخين السجائر أحد عوامل الخطر القلبية الوعائية ، والإقلاع عن التدخين هو المقياس الأكثر فعالية لنمط الحياة للوقاية من عدد كبير من أمراض القلب والأوعية الدموية.

ممارسة الرياضة : وصفة سحريه لارتفاع ضغط الدم

البروفيسور عبد الكريم عبد الوهاب العثمان / كلية طب اربيل

يعتبر ارتفاع ضغط الدم من اهم اسباب الإصابة بقصور الشرايين التاجية ، عجز القلب، الجلطة الدماغية وامراض العجز الكلوي .ان ممارسة الرياضة ،تقليل الوزن ، اتباع نمط غذائي صحي، الاقلاع عن التدخين وتقليل ملح الطعام تعتبر الخط العلاجي الاول في علاج ارتفاع ضغط الدم الاولي .

اشارت الدراسات العالمية الحديثة ان ممارسة تمارين رياضيه مختلفة وبشكل يومي منتظم يسهم في خفض ضغط الدم لدى غير المصابين والمصابين بأرتفاع ضغط الدم بالإضافة لدى المصابين بارتفاع ضغط الدم المقاوم للعلاج.

يجب عليك ممارسة تمارين مختلفة يوميا بشكل منتظم كالمشي السريع خارج المنزل او داخل المنزل باستخدام جهاز المشي نصف ساعه كأقل تقدير في صباح كل يوم.، السباحه، قياده الدراجة الهوائية واجراء تمارين شد العضلات كرفع الاثقال (كل حسب امكانيته وجنسه) وتمارين العضلات للساقين ،الاطراف العليا والبطن.

قبل الانخراط في ممارسة التمارين الرياضية يتوجب عليك استشارة الطبيب في الحالات التالية:

١ـ في حالة اصابتك بمرض مزمن مثل داء السكري، امراض القلب والشرايين وامرض الرئتين.

٢ـفي حالة الإصابة بالجلطة القلبية

٣ـ اصابة احد افراد العائلة بأمراض القلب دون سن ٥٥ لدى الرجال ودون ٦٥ سنه لدى النساء.

٤ـ في حالة الشعور بالم او عدم ارتياح في منطقة الصدر، الفك، الرقبة، الذراع .

٥ـ عند شعورك بالدوار.

٦ـفي حالة عدم تأكدك بان صحتك جيدة.

يتوجب عليك التوقف من التمارين الرياضية وطلب العناية الطبية فوراً في حالة احساسك باي من العلامات الإنذارية التالية خلال التمارين.

١ـ الم او شد في الصدر، الفك او الذراع.

٢ـضيق شديد في التنفس.

٣ـ عدم انتظام نبضات القلب.