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Study About High Blood Pressure & Pathophysiology In Human And study the effect of drugs

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ج

وَلَمَّا بَلَغَ أَشُدَّهُ آتَيْنَاهُ حُكْمًا وَعِلْمًا ۚ

وَكَذَٰلِكَ نَجْزِي الْمُحْسِنِينَ (٢٢)

سورة يوسف

صدق الله العظيم

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DEDICATION

Before all great thanks to (God) Allah who gave us strength and patience to complete this research.

I present the summary of my scientific effort To the one who taught me how to stand firmly above the ground

Dear Dad

To the wellspring of love, altruism and generosity.

Dear mother

To everyone from whom I received advice and support

List of Contents

Abstract

The studies presented systemic arterial hypertension as the most common chronic disease in subjects male and females and This has been confirmed by many studies, as well as the examinations that we conducted on patients lying in the hospital, after reviewing their medical history and the ages at which high blood pressure began and the diseases that accompanied it, in addition to blood analyzes as well as the percentage of salts and minerals in the body.and After taking a number of patient cases and conducting a comprehensive statistics on them, we arrived at:The mean age of the sample was 51.00 ± 12.21 years, with 50% of participants being females and a total of 50 % belonging to male groups with mean age 49.50 ± 9.04 And hypertensive individuals were more frequent in the age groups of 61 to 80 years or more than in the age group of 41 to 60 years, when compared to nonwhite and with 20- 40 years of both male and female same occurred for the self-evaluation of bad health, poor quality of life regarding physical condition, obesity, presence of a CVD, presence of all CKDs evaluated (diabetes, cerebrovascular disease, and coronary disease), and metabolic syndrome. At the end of our research, we concluded that high blood pressure is a serious and chronic disease that should not be tolerated, especially when any medical symptom appears in the patient, because it may cause a lifelong health disability that affects the entire lifestyle. The goal of our study was to try to uncover this disease and raise people's awareness. It reduces the risk of infection, and we ask God to grant everyone good health .

1. Introduction

Hypertension, also known as high blood pressure, is a chronic condition marked by elevated blood pressure in the arteries, posing a significant global public health concern due to its link to various cardiovascular diseases, including heart attacks, strokes, and kidney disease. Primary hypertension, which develops gradually over time without an identifiable cause, contrasts with secondary hypertension, which arises from underlying conditions like kidney disease or hormonal disorders. Risk factors include age, genetics, poor lifestyle choices such as a high-sodium diet, lack of physical activity, obesity, excessive alcohol consumption, and smoking, along with certain chronic conditions like diabetes, kidney disease, and sleep apnea. Hypertension is often asymptomatic, earning it the moniker "silent killer," though severe cases or hypertensive crises can manifest symptoms like headaches, shortness of breath, dizziness, or nosebleeds. Diagnosis relies on blood pressure readings, with hypertension typically defined as consistently exceeding 130/80 mmHg. Additional tests may be conducted to identify underlying causes or assess complications. Treatment and management involve lifestyle modifications like dietary changes, regular exercise, weight management, limiting alcohol consumption, and smoking cessation, alongside various classes of antihypertensive medications tailored to individual circumstances. Regular blood pressure monitoring is crucial, particularly for those diagnosed with hypertension. Managing underlying conditions contributing to secondary hypertension is also vital, as is early detection and management to prevent complications like heart disease, stroke, kidney disease, and vision loss. Hypertensive emergencies, characterized by severely elevated blood pressure levels, demand immediate medical attention to avert organ damage. [1]

2. Risk Factors for Hypertension

Risk of developing hypertension is strongly influenced by family history and age. A positive family history of hypertension is a risk factor for the development of hypertension, and it is well known that risk for hypertension increases as we age [٢]. Other factors influencing risk of hypertension include sex and race. Overall, the prevalence of hypertension in the United States is relatively similar for men and women. However, the prevalence of hypertension is higher in men than women before 45 years of age. By age 65, this trend is reversed and women have a higher prevalence of hypertension than men [٣]. As the rates of hypertension are lower among premenopausal women as compared to postmenopausal women, it is hypothesized that estrogen protects women from developing hypertension [٤]. Racial and ethnic differences in hypertension are also evident. Compared to non-Hispanic Whites or Caucasians, African Americans have a higher prevalence of hypertension. In addition, hypertension in African Americans is more severe and has an earlier age of [٥].

3. Causes

3.1 Causes of primary hypertension

A complicated interplay between genetics and environmental variables leads to hypertension. Both common genetic variations with minor effects on blood pressure and rare genetic variants with significant impacts on blood pressure have been found.[6] Genome-wide association studies (GWAS) have identified 35 genetic loci linked to blood pressure, with 12 newly discovered loci impacting blood pressure. Each locus contains a sentinel SNP associated with DNA methylation at nearby CpG sites, primarily in genes related to renal and vascular smooth muscle function. Although the mechanisms remain unclear, DNA methylation may connect common genetic variations to various symptoms. Using these sentinel SNPs, genetic variations, alone or combined, have been shown to increase the risk of clinical characteristics related to high blood pressure. [7]

Coronary artery ectasia (CAE) involves the enlargement of a coronary artery to 1.5 times or more than other non-ectasia segments. The unadjusted odds ratio (OR) of CAE in

individuals with hypertension (HTN) compared to those without HTN was estimated to be 1.44. [8] Blood pressure tends to increase with age, especially when coupled with a western diet and lifestyle, significantly raising the risk of hypertension later in life. Various environmental factors influence blood pressure, including high salt intake, lack of exercise, and central obesity. [9]

Early life events like low birth weight, maternal smoking, and lack of breastfeeding might elevate the risk of adult essential hypertension, though the exact mechanisms are unclear. Individuals with untreated hypertension tend to have higher levels of blood uric acid compared to those with normal blood pressure, though whether this is causative or secondary to poor kidney function is uncertain. [10]

3.2 Causes of secondary hypertension

Secondary hypertension results from an identifiable cause. Kidney disease is the most common secondary cause of hypertension. Hypertension can also be caused by endocrine conditions, such as Cushing's syndrome, hyperthyroidism, hypothyroidism, acromegaly, Conn's syndrome or hyperaldosteronism, renal artery stenosis (from atherosclerosis or fibromuscular dysplasia), hyperparathyroidism, and pheochromocytoma.[11] Other causes of secondary hypertension include obesity, sleep apnea, pregnancy, coarctation of the aorta, excessive eating of liquorice, excessive drinking of alcohol, certain prescription medicines, herbal remedies, and stimulants such as coffee, cocaine and methamphetamine.[12]

A 2018 review found that any alcohol increased blood pressure in males while over one or two drinks increased the risk in females.[13]

4. Pathophysiology

In most people with established essential hypertension, increased resistance to blood flow (total peripheral resistance) accounts for the high pressure while cardiac output remains normal. There is evidence that some younger people with prehypertension or 'borderline hypertension' have high cardiac output, an elevated heart rate and normal peripheral resistance, termed hyperkinetic borderline hypertension.[14] These individuals develop the typical features of established essential hypertension in later life as their cardiac output falls and peripheral resistance rises with age.[14] Whether this pattern is typical of all people who ultimately develop hypertension is disputed. The increased peripheral resistance in established hypertension is mainly attributable to structural narrowing of small arteries and arterioles, although a reduction in the number or density of capillaries may also contribute.[15]

It is not clear whether or not vasoconstriction of arteriolar blood vessels plays a role in hypertension. Hypertension is also associated with decreased peripheral venous compliance which may increase venous return, increase cardiac preload and, ultimately, cause diastolic dysfunction.[16]

Pulse pressure (the difference between systolic and diastolic blood pressure) is frequently increased in older people with hypertension. This can mean that systolic pressure is abnormally high, but diastolic pressure may be normal or low, a condition termed isolated systolic hypertension. The high pulse pressure in elderly people with hypertension or isolated systolic hypertension is explained by increased arterial stiffness, which typically accompanies aging and may be exacerbated by high blood pressure.[17]

Many mechanisms have been proposed to account for the rise in peripheral resistance in hypertension. Most evidence implicates either disturbances in the kidneys' salt and water handling (particularly abnormalities in the intrarenal renin–angiotensin system) or abnormalities of the sympathetic nervous system.[18] These mechanisms are not mutually exclusive and it is likely that both contribute to some extent in most cases of essential hypertension. It has also been suggested that endothelial dysfunction and vascular

inflammation may also contribute to increased peripheral resistance and vascular damage in hypertension. Interleukin 17 has garnered interest for its role in increasing the production of several other immune system chemical signals thought to be involved in hypertension such as tumor necrosis factor alpha, interleukin 1, interleukin 6, and interleukin 8.[19]

Excessive sodium or insufficient potassium in the diet leads to excessive intracellular sodium, which contracts vascular smooth muscle, restricting blood flow and so increases blood pressure.[20]

5. Diagnosis

Diagnosing hypertension extends beyond identifying high blood pressure; it involves evaluating cardiovascular disease risk, assessing target organ damage, and considering associated clinical conditions. Some diagnostic investigations are standard for all patients, while others are tailored to specific groups based on history, examination, and initial tests. In rare cases of inherited hypertension, a single gene mutation may elucidate the condition's underlying cause. [21]. (Figure 1) A subset of hypertension cases may have reversible causes, and accurate diagnosis could lead to significant improvement or cure, reducing cardiovascular disease risk. Hence, it's advisable to conduct routine screening for secondary hypertension in all patients, utilizing clinical history, physical examination, and basic laboratory tests. Secondary hypertension should be considered in scenarios like sudden hypertension worsening, inadequate response to medication, or disproportionate target organ damage. Specific diagnostic tests are warranted in such cases. [21].

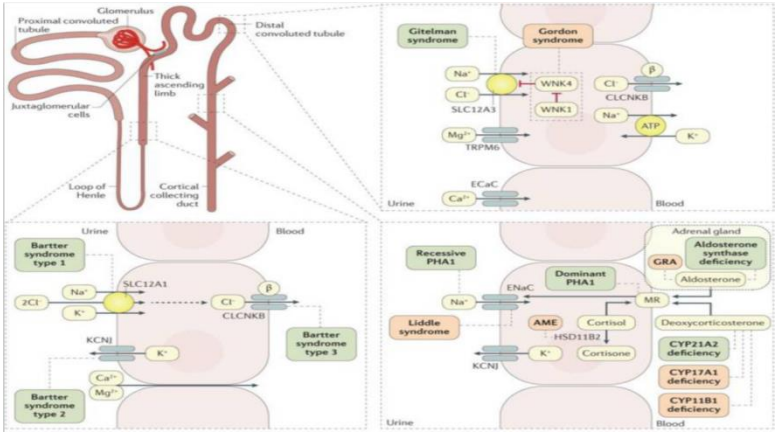

Figure 1 Pathways affected in single gene, Mendelian hypertension and hypotension syndromes

When taking a medical history for hypertension, it's crucial to note the timing of the initial diagnosis, current and past blood pressure readings, and use of antihypertensive medications. In women, a history of pregnancy-related hypertension is significant. Hypertension elevates the risk of cardiovascular disease (CVD) complications and chronic kidney disease (CKD), warranting a thorough medical history to assess overall CVD risk. Special attention should be given to smoking history, dyslipidemia, and diabetes mellitus. Calculators can help estimate CVD risk, and high-risk adults may benefit from both lifestyle changes and antihypertensive drug therapy. [22]

During the physical examination for hypertension, the goal is to confirm the diagnosis, assess for target organ damage, and screen for secondary causes. Patients should be seated quietly for 5 minutes with the blood pressure cuff at heart level before readings are taken. Averaging 2 to 3 measurements obtained at separate occasions provides an accurate estimation of blood pressure. Both arms should be measured at least once, and significant differences in readings may indicate vascular abnormalities. Using appropriately sized cuffs is important, especially for obese patients. Blood pressure should also be measured in both sitting and standing positions to check for orthostatic hypotension, particularly in the elderly. [23]

6. MANAGEMENT

6.1 Non-Pharmacological Management

Lifestyle modifications are recommended for all individuals with hypertension and are similar to those for preventing hypertension. Dietary changes, such as reducing sodium intake to below 2.3 grams per day (or even lower for those most sensitive to sodium), and increasing potassium intake to 3.5-5.0 grams per day [24], can reduce systolic blood pressure by 2-4 mmHg. Current salt intake far exceeds recommendations, with estimates ranging from 9-12 grams per day in most countries[25] . The American Heart Association suggests lowering salt intake to 3.8 grams per day, while European guidelines recommend 5-6 grams per day[23] . Randomized controlled trials consistently show that reducing sodium intake lowers blood pressure, with the DASH-sodium trial providing strong evidence[26] . Increasing potassium intake is associated with reduced blood pressure,

particularly in individuals with low baseline potassium intake, and the effect is greater with lower salt intake[27]. Moderate alcohol consumption, limited to ≤2 standard drinks per day for men and ≤1 standard drink per day for women, can contribute to a 2-4 mmHg reduction in blood pressure[28]. Regular physical activity, particularly endurance training, can significantly reduce blood pressure, with sessions lasting 40-60 minutes at least three times per week having the greatest effect[29]. Weight loss is also important, as excess adiposity raises blood pressure and may require more antihypertensive medications for control.[30]

6.2 Antihypertensive Pharmacotherapy

Antihypertensive pharmacotherapy has evolved over several decades driven by development of various antihypertensive medication classes and large-scale outcomes trials proving their benefits on CVD morbidity and mortality. Clinicians are now faced with a plethora of antihypertensive medications of different drug classes and a variety of fixed dose combinations. Typically, antihypertensive pharmacotherapy begins with first-line antihypertensive medications either in monotherapy or in combination[31]. Combination therapy may be preferable in patients with higher levels of pretreatment BP. First-line antihypertensive medications include ACE inhibitors, angiotensin II receptor blockers (also known as sartans), dihydropyridine calcium channel blockers, and thiazide diuretics. Beta-blockers are also indicated in patients with heart failure and reduced left ventricular ejection fraction or post myocardial infarction, and some guidelines recommend beta-blockers as first line antihypertensive medications [32]. The choice should be based on individual efficacy and tolerability. Ethnicity affects the response to antihypertensive medications, and it has been suggested that calcium channel blockers and diuretics may be the first choice in blacks. Further, in specific clinical situations, for example hypertension in pregnant women, other medications such as alpha-methyldopa (an agonist of alpha adrenoreceptors in the central nervous system that inhibits the sympathetic nervous system) or labetalol (a beta adrenoreceptor blocker) are preferable, whereas some first line antihypertensives, for example ACE inhibitors and angiotensin II receptor blockers, are contraindicated because of increased risk for renal teratogenicity.

Divided dosing of antihypertensive drugs tends to decrease adherence and should be avoided when possible.[33]

6.2.1 Diuretics

Low-dose diuretic therapy is effective and reduces the risk of stroke, coronary heart disease, congestive heart failure, and total mortality. Whilst thiazides are most commonly used, loop diuretics are also used successfully and the association with a potassium sparing diuretic reduces the risk of both hypokalaemia and hypomagnesaemia. Even in small doses diuretics potentiate other antihypertensive drugs. The risk of sudden death is reduced when potassium-sparing diuretics are used. In the long-term, spironolactones reduce morbidity and mortality in patients with heart failure that is a typical complication of long-standing hypertension.[34,35]

6.2.2 Beta-blockers

High sympathetic tone, angina, and previous myocardial infarction are good reasons for using β-blockers. As a low dose minimizes the risk of fatigue (an unpleasant effect of β-blockade) addition of a diuretic or a calcium channel blocker is often beneficial. However, β-blockade therapy is associated with symptoms of depression, fatigue, and sexual dysfunction. These side-effects have to be taken into consideration in the evaluation of the benefits of treatment.[36] Over the past few years β-blockers have been used increasingly frequently in the management of heart failure, a known complication of arterial hypertension. They are effective but their introduction in the presence of heart failure has to be very cautious, starting with very low doses to avoid an initial worsening of heart failure.[36]

6.2.3 Calcium channel blockers

Calcium channel blockers can be divided into dihydropyridines (e.g. nifedipine, nimodipine, amlodipine) and non-dihydropyridines (verapamil, diltiazem). Both groups decrease peripheral vascular resistance but verapamil and diltiazem have negative inotropic and chronotropic effects. Short-acting dihydropyridines such as nifedipine cause

reflex sympathetic activation and tachycardia, while long-acting drugs such as amlodipine and slow-release preparations of nifedipine cause less sympathetic activation. Short-acting dihydropyridines appear to increase the risk of sudden death. However, the systolic hypertension in Europe (SYST-EUR) trial which compared nitrendipine with placebo had to be stopped early because of significant benefits of active therapy.[37]

Calcium channel blockers are effective in the elderly and may be selected as monotherapy for patients with Raynaud's phenomenon, peripheral vascular disease, or asthma, as such patients do not tolerate β-blockers. Diltiazem and verapamil are contraindicated in heart failure. Nifedipine is effective in severe hypertension and can be used sublingually; there is need for caution because of the risk of excessive hypotension. Calcium channel blockers are often associated with β-blockers, diuretics and/or ACE inhibitors.[38]

6.2.4 Angiotensin converting enzyme inhibitors and Angiotensin II receptor blockers

ACE inhibitors are increasingly favored as first-line therapy for hypertension due to their few side effects and contraindications, except for bilateral renal artery stenosis. While effective for unilateral renovascular hypertension, there's a risk of ischemic atrophy, making angioplasty or surgical renal artery reconstruction preferable to long-term medical therapy. In diabetic hypertensive patients, ACE inhibitors are preferred as they slow the progression of renal dysfunction. They are also the first-choice agents in hypertension with heart failure. The HOPE trial demonstrated that ramipril reduced the risk of cardiovascular events, even without hypertension, suggesting a protective effect beyond blood pressure reduction. [38] The LIFE study compared the angiotensin receptor antagonist losartan with a β-blocker (atenolol) in over 9000 hypertensive patients. Patients receiving losartan experienced better reductions in mortality and morbidity, primarily due to a greater decrease in strokes. Losartan was also more effective in reducing left ventricular hypertrophy, an independent risk factor for adverse outcomes. This superiority of losartan was particularly pronounced in patients with isolated systolic hypertension. These positive findings led to an editorial titled "Angiotensin blockade in hypertension: a promise

fulfilled." It's worth noting that the comparator in the study was a β-blocker, which historically showed no significant benefits over placebo in the elderly. [38]

7. EPIDEMIOLOGY

In pre-industrial societies, BP levels had narrow distributions with mean values that changed little with age and averaged around 115/75 mmHgb[39], a value that probably represents the normal (or ideal) BP for humans. However, in most contemporary societies, systolic BP levels rise steadily and continuously with age in both men and women. This ubiquitous finding could be explained because age is a proxy for the probability and duration of exposure to the numerous environmental factors that increase BP gradually over time, such as excessive sodium consumption, insufficient intake of dietary potassium, overweight and obesity, alcohol intake and physical inactivity. Other factors, such as genetic predisposition or adverse intrauterine environment (such as gestational hypertension or pre-eclampsia), have small but definite associations with high BP levels in adulthood. Even modest rises in mean population BP lead to large increases in the absolute number of people with hypertension. [40]

As economic development progresses, hypertension initially affects those with a high socioeconomic status, but at later stages of economic development, the prevalence of hypertension and its consequences are greatest in those with lower socioeconomic status; this phenomenon is seen both within and between countries. Further, the speed of change prevalence of hypertension since 2000 to 2010 has been much more rapid than in previous epidemiological transitions. [41]

٨-Material and methods

Study design :

This study was conducted to find out the causes of high blood pressure and the factors that cause it, with a focus on age groups and the reasons for its prevalence in males and females.

Study population:

We took information on approximately 100 patients from Babylon Governorate of both sexes and of various age groups, social classes, and educational and job levels.

Data collection:

The samples were collected after we visited the Imam Al-Sadiq Teaching Hospital located in Babylon and under the supervision of our esteemed doctor, by reviewing the archive records of the patients in the hospital, as well as reviewing their blood tests, to conduct our research and evaluation statistics for them. Although it was not easy, thanks to God, we were able to do so. With the help of our doctor, everything went well after we completed 3 months of visiting the hospital to collect and study these samples.

Note that we started collecting samples on 12/15/2023 until 3/1/2024.

٩-Results

The mean age of the sample was 51.00 ± 12.21 years, with 50% of participants being females and a total of 50 % belonging to male groups with mean age 49.50 ± 9.04 , The description of the remaining variables in the study can be seen in Table 1.

Table (4-1): Main features of the study population with Systemic arterial hypertension

Variations	NO%
Gender	
Male	50(100%)
Female	50(100%
Age groups	
20-40	8(8%)
41-60	34(34%)
61-80	62(62%)
Addres	
Babylon	100(100%)
Occupation	
working	44(44%)
No working	41(41%)
Retirement	15(15%)
Medical history	
HT /DM /CVA	33(33%)
HT	26(26%)
HT/Hiperlipidema	8(8%)
ht/Jionts pain	6(6%)
HT/CKD	8(8%)
HT/Agina	4(4%)
HT/CANCER	4(4%)
HT/CVA/prostate hyber	5(5%)
HT/ASTHMA	3(3%)
HT/HF	3(3%)
HOM for HT	
Bispralol/crestor/diostar	4(4%)
carvidilol/lasix/atrovastatin	6(6%)
LAXIX/Avas	5(5%)
Amalodipine	4(4%)
HIPRIL A	6(6%)
atacand crestor	5(5%)
diovan	6(6%)
betaloc	3(3%)
extra plus / amlodpin	4(4%)
tensart	6(6%)
distro plus	4(4%)
metoprilol diovan	6(6%)
betaloc metopolol	5(5%)
ralsartan carvidolol	3(3%)
tenormin	4(4%)
temlodipin	6(6%)

Carvidiol	5(5%)
Colpidogril	3(3%)
HIPRIL A	
Lisinopril	4(4%)
Aimalodpin	6(6%)
isosartan	5(5%)

As shown in Table 2, hypertensive individuals were more frequent in the age groups of 61 to 80 years or more than in the age group of 41 to 60 years, when compared to nonwhite and with 20-40 years of both male and female same occurred for the self-evaluation of bad health, poor quality of life regarding physical condition, obesity, presence of a CVD, presence of all CKDs evaluated (diabetes, cerebrovascular disease, and coronary disease), and metabolic syndrome.

TABLE 2 Prevalence of high blood pressure among male and females in the study

Parameters	Male Mean±S.err	Female Mean±S.err	T test	P= value
Age	49.50 ± 9.04	51.00 ± 12.21	3.65	0.04*
WBCs	9.50 ± 3.04	9.59 ± 2.64	0.13	0.07
Neu	7.17 ± 2.27	10.43 ± 1.86	1.38	0.28
Lym	1.95 ± 0.42	1.77 ± 0.22	0.40	0.08
mon	0.56 ± 0.15	0.53 ± 0.06	0.19	0.16
Eos	0.32 ± 0.05	0.26 ± 0.05	0.73	0.29
Bas	0.01 ± 0.001	0.13 ± 0.04	2.33	<0.0001*
Lym%	68.02 ± 4.81	71.28 ± 2.50	0.56	0.58
Mon%	19.88 ± 4.04	12.06 ± 1.26	2.13	0.02*
Eos%	9.48 ± 1.74	3.68 ± 0.50	3.72	0.001*
Bas%	4.57 ± .84	6.39 ± 1.41	2.66	0.11
RBC	0.13 ± 0.02	1.10 ± 0.34	2.33	0.000*
HGB	3.32 ± 0.24	4.41 ± 0.33	2.42	0.025*
HCT	103.22 ± 6.45	115.33 ± 5.75	1.38	0.18
MCB	49.94 ± 2.56	33.36 ± 1.31	0.89	0.021*
MCH	84.40 ± 3.03	56.03 ± 6.50	3.43	0.005*
PLT	188.88 ± 27.94	175.39 ± 30.99	0.29	0.77
MPV	10.36 ± 0.61	8.51 ± 0.31	2.92	0.23
BDW	15.11 ± 0.44	53.15 ± 3.93	3.16	0.005*
PCT	0.27 ± 0.08	011 ± 0.002	7.01	0.000*
PLCC	64.88 ± 5.44	51.04 ± 3.97	0.75	0.048*

P<0.05 significant

١٠-Discussion

The studies presented systemic arterial hypertension as the most common chronic disease in subjects male and females [47]. Most studies showed that hypertensive subjects diagnosed had a mean age of more than 60 years , Added to this condition, age also emerges as one of the risk factors for the development of hypertension, since during the

physiological process of aging occur morphological changes, including progressive stiffening and loss of compliance of the major arteries that influence blood pressure levels[48].

In this scenario, the hypothesis raised would be that the oxidative damage accumulated due to the aging process, added with a weakened antioxidant defense system could cause a disturbance in the balancing redox, which would cause an increase in reactive oxygen species. Thus, oxidative stress may enhance cellular responses of early mediators of inflammation. Besides affecting the innate and adaptive immune system, aging is also associated with a pro-inflammatory state in the host[49].

In this scoping review, the studies showed that male subjects with systemic arterial hypertension as well as more likely to die than female subjects28,30. One hypothesis that could explain these findings is attributed to a potential protection of the X chromosome and sex hormones, which play an important role in the innate and adaptive development of immunity. Since the ACE2 gene is located at the Xp22 locus on the human X chromosome[50]

Haematological indices, particularly red cell distribution width (RDW), neutrophil lymphocyte ratio (NLR) and mean platelet volume (MPV), were established as markers of systemic inflammation and vascular pathology[51-53]. Their prognostic value was clearly demonstrated in coronary artery disease, stroke and several other vascular diseases. Correlation of such haematological indices and HT was also investigated and it was proposed that haematological indices may predict the severity of HT and end-organ damage[54-56]. With this review, we aimed to show the place of haematological indices in the essential HT and demonstrate its clinical implication. The aetiology of essential HT is not clear, however, it has been accepted as a multifactorial disease arising from the combined action of many genetic, environmental and behavioural factors. Renal sodium retention, vascular hypertrophy, endothelial cell dysfunction, sympathetic nervous system hyperactivity, upregulation of the renin-angiotensin-aldosterone system, altered T-cell function, insulin resistance and dietary and habitual factors were postulated as common

mechanisms of HT[52]. However, oxidative stress and inflammation seem to play a major role in the pathophysiology of HT and also concomitant end-organ damage[55].

White blood cell (WBC) count constitutes an inflammatory marker and it tends to increase in HT. The WBC count was higher in non-dipping HT and WBC counts in the highest quartile may reflect enhanced inflammatory response and end-organ damage [57].

Mean platelet volume has known to be an indicator of platelet activation and, its correlation with cardiovascular disease is well established [58]. Platelets play a pivotal role in the development of atherosclerotic lesions, plaque destabilization, and atherothrombosis. It has been clearly demonstrated that MPV is an unfavourable prognostic factor in ischaemic coronary heart disease[59]. A few studies have also proposed that MPV may predict microvascular injury in coronary vessels and diabetic microvascular complications, including nephropathy and hypertensive microvascular end-organ damage[60] There is a stepwise increase between MPV and the severity of hypertensive disease. Mean platelet volume was also found higher in ophthalmologic complications, Moreover, its level was increased in masked HT[61-62].

As far as drug treatment is concerned, there was no association between the use of any class of antihypertensive and a higher risk of mortality [63-64]. Subjects with systemic arterial hypertension and no antihypertensive treatment had higher mortality rate when compared to hypertensive subjects with antihypertensive treatment[65]. However, other studies showed that subjects with and without antihypertensive treatment had similar laboratory profile results, and showed no differences in the occurrence of adverse effects or clinical outcomes[66].It is noteworthy that because hypertension is a chronic disease, its control requires treatment with pharmacological and non-pharmacological measures throughout life. Therefore, it is essential to investigate adherence to drug therapy, and lifestyle habits of hypertensive individuals.

Conclusion :

Haematological indices, predominantly RDW, NLR and MPV, reflect oxidative stress and inflammatory state, which also postulate as major mechanisms of HT and its vascular

complication. There is a stepwise relation between the severity of HT, hypertensive end-organ damage and haematological indices. However, it is still not clear whether these parameters are responsible in the pathogenesis of HT or they increase as a result of the progression of hypertensive disease. There is a need of further investigations to clarify definitive pathophysiologic mechanism of HT regarding the role of hematological indices. Nevertheless, there is a clear consensus that these haematological parameters have a prognostic value in the essential HT and their abnormality may strongly suggest hypertensive endorgan damage.

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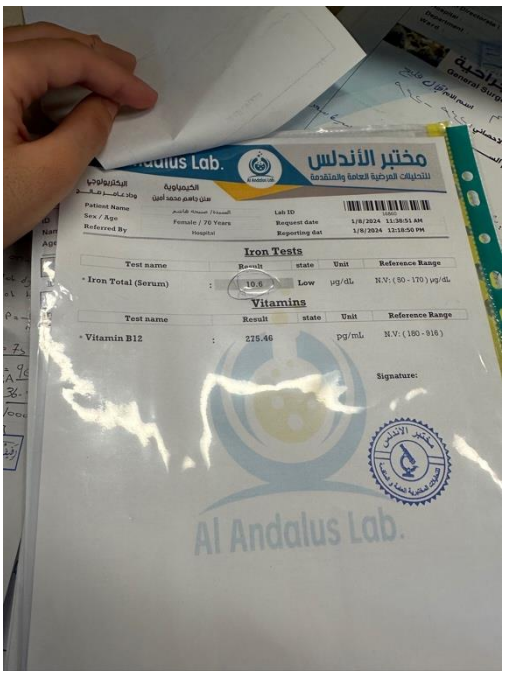
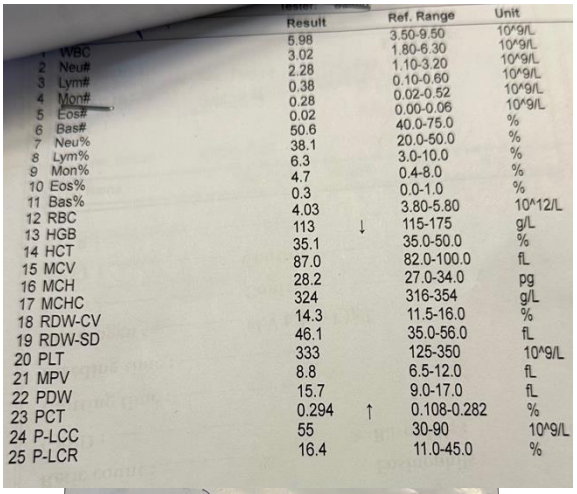
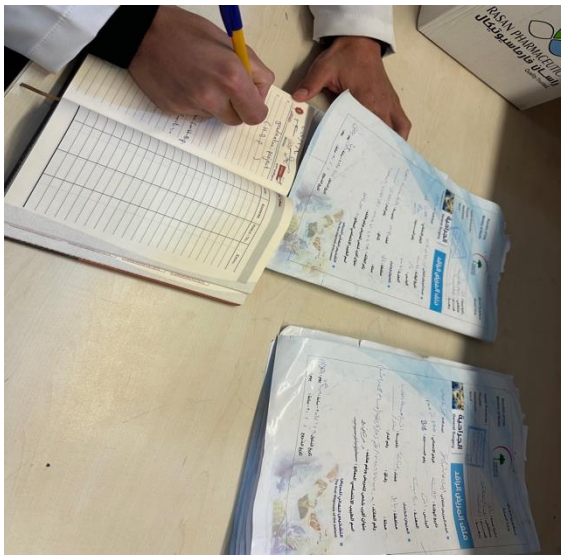
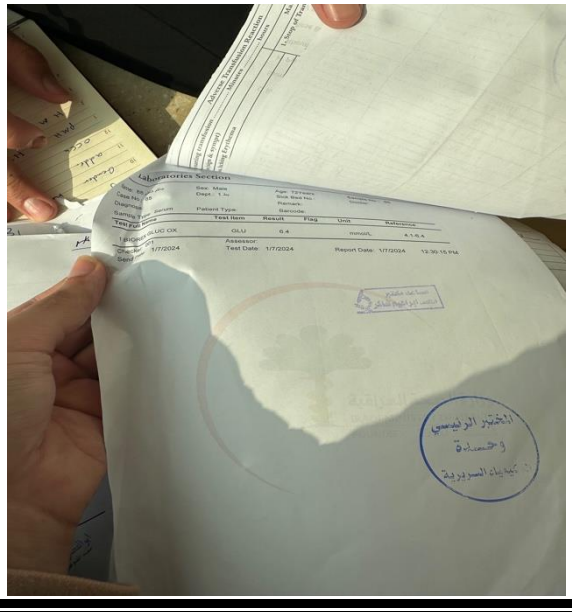
Some pictures from work inside the hospital

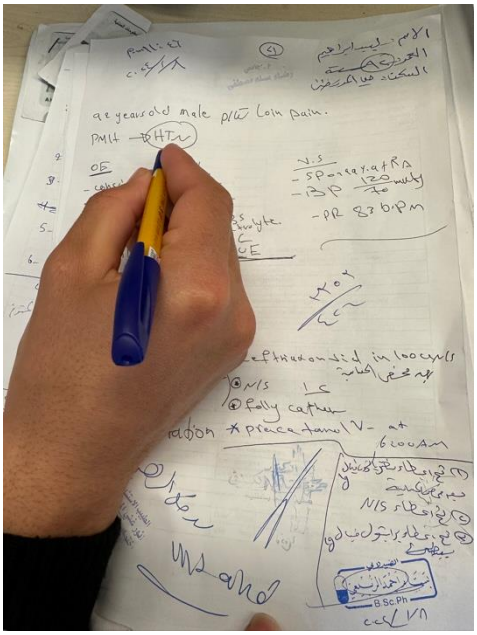




		Result		Ref. Range	Unit
1	WBC	5.98		3.50-9.50	10^9/L
2	Neu#	3.02		1.80-6.30	10^9/L
3	Lym#	2.28		1.10-3.20	10^9/L
4	Mon#	0.38		0.10-0.60	10^9/L
5	Eos#	0.28		0.02-0.52	10^9/L
6	Bas#	0.02		0.00-0.06	10^9/L
7	Neu%	50.6		40.0-75.0	%
8	Lym%	38.1		20.0-50.0	%
9	Mon%	6.3		3.0-10.0	%
10	Eos%	4.7		0.4-8.0	%
11	Bas%	0.3		0.0-1.0	%
12	RBC	4.03		3.80-5.80	10^12/L
13	HGB	113	↓	115-175	g/L
14	HCT	35.1		35.0-50.0	%
15	MCV	87.0		82.0-100.0	fL
16	MCH	28.2		27.0-34.0	pg
17	MCHC	324		316-354	g/L
18	RDW-CV	14.3		11.5-16.0	%
19	RDW-SD	46.1		35.0-56.0	fL
20	PLT	333		125-350	10^9/L
21	MPV	8.8		6.5-12.0	fL
22	PDW	15.7		9.0-17.0	fL
23	PCT	0.294	↑	0.108-0.282	%
24	P-LCC	55		30-90	10^9/L
25	P-LCR	16.4		11.0-45.0	%







11-Conclusions

Hypertension is a chronic medical condition that leads to severe damage to numerous organs in the body (e.g., heart, kidney, and retina) and eventually results in cardiovascular disease. Further, hypertension increases the risk of developing cognitive impairment, including an increased risk for vascular dementia. With good reason, understanding the

causes of hypertension is an important area of scientific inquiry that will assist in the development of effective interventions by healthcare providers. Untreated hypertension is a serious global health problem. Most people are familiar with having their blood pressure checked during visits to their healthcare providers. By adopting the recommended assessment protocol using an occluding cuff and stethoscope, healthcare providers can measure arterial pressures reliably and easily. However, in certain circumstances, blood pressure measures obtained in clinics bear little resemblance to those that occur during daily life; in these situations, ambulatory blood pressure measurements have proven to be quite valuable. Although few medical reasons for discrepant blood pressure values measured in clinic and life settings exist, there are psychological reasons why such differences occur. Foremost among these is the well-established association between exposure to stress during daily life and elevated blood pressure. Evidence from both animal and human studies has revealed that exposure to a range of chronic stressful environments is associated with hypertension. The mechanisms responsible for this association, however, are not clear. Nevertheless, several pathways have been proposed through which stress may lead to hypertension, including the propensity for hypertensive patients to differ from persons with normal blood pressures regarding:

(1) the experience of negative affect (anxiety, anger, or depression);

(2) the hostile expression or suppression of anger;

(3) the denial or defensiveness associated with the experience of emotion or pain;

(4) specific cognitive or intellectual capabilities; and

(5) the magnitude and patterning of cardiovascular responses to and recovery from acute environmental stressors.

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