Overview of complications and roles of family doctor in managing hypertension

Abdulsalam Rashed Alshehri, Jamaan Hasan Alqahtani, Majed Abdulhakim Alamoudi, Faisal Sultan Alharthi, Majed Mohammad Albogami, Hamed Abu Baker Altobaiqi

Abstract:

Hypertension is a cause of morbidity and mortality. In this review we will discuss the pathophysiology, effective treatment methods. We highlight the consequences and complication of hypertension as well as role of family doctor in managing hypertension. We performed detailed search through electronic databases; PubMed, and EMBASE, for studies published in English language and human subjects thought instant to 2017. Studies discussing the managing hypertension in primary care by family physicians, were included whether were reviews or control studies. following keywords are used in search process: “hypertension”, “complications”, “family doctors”, “primary care”, “Management”. The excessive pressure on your artery walls caused by high blood pressure can damage blood vessels, as well as organs in the body. Family doctor and their teams have an important duty to play in the recognition and management of high blood pressure, because it can cause mortality. Risk factors like smoking and obesity, can cause hypertension, in this case changing the lifestyle is important.

Introduction:

Hypertension, defined as systolic blood pressure (SBP) of 140 mm Hg or better or diastolic blood pressure (DBP) of 90 mm Hg or higher [1]. It is anticipated that it will affect up to one-third of the grown-up populace worldwide by 2025 [2]. Hypertension is the leading chronic risk aspect

for mortality, accounting for 13.5% of all deaths [3]. The occurrence is approximately 15%, 30%, and 55% in males aged 18 to 39 years, 40 to 59 years, and 60 years or older, specifically, and regarding 5%, 30%, and 65% in females in the same age groups [4] Modern arterial stiffening leads to a constant rise in SBP throughout adult life, whereas DBP plateaus in the 6th years of life and decreases thereafter, which explains the high occurrence of isolated systolic hypertension in the senior [5].

High blood pressure (BP) is an important danger variable for end-stage renal illness, coronary artery illness, congestive heart failure, and stroke. Throughout the BP variety from 115/75 mm Hg to 185/115 mm Hg, the threat of cardio occasions doubles with each 20 mm Hg increment of SBP [6]. Despite pharmacologic advancements, just one-third of American grown-up patients with hypertension in 2000 had adequate BP control, far below the Healthy People 2010 goal of 50%.

With issues about the cost, performance, and possibility for deleterious negative effects of antihypertensive medicines, there is an increased interest in behavioural interventions, including workout for the treatment and avoidance of high blood pressure [6]. Regrettably, exercise prescription is underused among clinicians, generally owing to unpredictability regarding its efficiency and uncertainty regarding workout prescription components (duration, intensity, and regularity).

Hypertension is a cause of morbidity and mortality. In this review we will discuss the pathophysiology, effective treatment methods. We highlight the consequences and complication of hypertension as well as role of family doctor in managing hypertension.

Methodology:

We performed detailed search through electronic databases; PubMed, and EMBASE, for studies published in English language and human subjects thought instant to 2017. Studies discussing the managing hypertension in primary care by family physicians, were included whether were reviews or control studies. following keywords are used in search process: “hypertension”, “complications”, “family doctors”, “primary care”, “Management”. We excluded case reports. Moreover, references of included studies were scanned for more relevant articles.

Discussion:

- **Pathophysiology**

High blood pressure is a chronic altitude of blood pressure that, in the long-lasting, causes end-organ damages and leads to raised morbidity and death [7]. Blood pressure is the item of cardiac output and systemic vascular resistance. It follows that patients with arterial hypertension may have a rise in cardiac output, a rise in systemic vascular resistance, or both. In the more youthful age group, the cardiac output is commonly elevated, while in older patients boosted systemic vascular resistance and enhanced stiffness of the vasculature play a dominant function. Vascular tone could rise because of enhanced α-adrenoceptor excitement or enhanced release of peptides such as angiotensin or endothelins. The final pathway is an increase in cytosolic calcium in vascular smooth muscle triggering vasoconstriction. A number of growth elements, including angiotensin and endothelins, cause a boost in vascular smooth muscular tissue mass termed vascular renovation. Both a rise in systemic vascular resistance and a rise in vascular stiffness

increase the load troubled the left ventricle; this induces left ventricular hypertrophy and left ventricular diastolic dysfunction [8].

In youth, the pulse pressure produced by the left ventricle is reasonably reduced and the waves reflected by the outer vasculature occur mainly after the end of systole, hence boosting pressure throughout the early part of diastole and boosting coronary perfusion. With aging, stiffening of the aorta and elastic arteries increases the pulse pressure. Reflected waves move from very early diastole to late systole [9].This causes a boost in left ventricular afterload, and results in left ventricular hypertrophy. The widening of the pulse pressure with ageing is a strong predictor of coronary heart problem.

The autonomic nervous system plays an essential duty in the control of blood pressure. In hypertensive patients, both enhanced release of, and improved peripheral sensitivity to, norepinephrine can be located. On top of that, there is raised responsiveness to stressful stimuli. One more feature of arterial hypertension is a resetting of the baroreflexes and decreased baroreceptor sensitivity [10].The renin-- angiotensin system is involved at least in some types of hypertension (e.g. renovascular hypertension) and is subdued in the presence of primary hyperaldosteronism. Elderly or black patients tend to have low-renin hypertension. Others have high-renin hypertension and these are more probable to develop myocardial infarction and various other cardiovascular issues [22].

In human important high blood pressure, and experimental hypertension, volume regulation and the relationship in between blood pressure and sodium excretion (pressure natriuresis) are unusual. Substantial evidence signifies that resetting of pressure natriuresis plays a vital function in creating hypertension. In patients with essential high blood pressure, resetting of pressure natriuresis is defined either by a parallel shift to higher blood pressures and salt-insensitive high

blood pressure, or by a decreased slope of pressure natriuresis and salt-sensitive hypertension [11].

- **Consequences and complications of hypertension**

The cardiac consequences of high blood pressure are left ventricular hypertrophy and coronary artery disease. Left ventricular hypertrophy is brought on by pressure overload and is concentric. There is an increase in muscle mass and wall thickness but not ventricular volume [12].Left ventricular hypertrophy hinders diastolic function, slowing ventricular relaxation and delaying filling. Left ventricular hypertrophy is an independent risk aspect for cardiovascular disease, specifically sudden death. The consequences of hypertension are a function of its intensity. There is no limit for complications to take place as elevation of blood pressure is related to boosted morbidity throughout the whole range of blood pressure (**Table 1**).

Table 1. Stages of hypertension (Joint National Committee VI Guideline)

Stage	Systolic	Diastolic
Optimal	<120	<80
Normal	120–129	80–84
High-normal	130–139	85–89
HT stage 1	140–159	90–99
HT stage 2	160–179	100–109
HT stage 3	>180	>110

Coronary artery disease is associated with, and accelerated by, chronic arterial hypertension, bring about myocardial ischaemia and myocardial infarction. Indeed, myocardial ischaemia is a lot more frequent in untreated or inadequately regulated hypertensive patients than in normotensive patients. Two major factors add to myocardial ischaemia: a pressure relevant boost in oxygen demand and a decrease in coronary oxygen supply arising from connected atheromatous lesions. High blood pressure is a significant threat element for fatality from coronary artery disease [13].

Heart failure is a consequence of chronic pressure overload. It could begin as diastolic disorder and progresses to overt systolic failure with cardiac congestion. Strokes are major difficulties of hypertension; they result from thrombosis, thrombo-embolism, or intracranial haemorrhage. Kidney illness, originally disclosed by micro-albuminaemia might proceed slowly and becomes evident in later years [22].

- **Role of family physician**

Improvements in analysis accuracy are crucial to attaining the goal of additional enhancing hypertension management, which could not be accomplished without buy-in from primary care carriers. To date, uptake of automated oscillometric AOBP gadgets within Canadian medical care techniques has been relatively high; nonetheless, it is unclear if they are being utilized ideally. The accessibility of ABPM has been restricted owing to cost and absence of reimbursement in lots of provinces.

In order to accomplish the goal of far better diagnosis and much better control of hypertension, family physicians must be encouraged to purchase both AOBP and ABPM devices and to utilize

them routinely and properly in professional technique. Some rural health plans already provide repayment for ABPM, and initiatives are under way in various other provinces to develop new billing codes. A "best-practice" table has been developed by CHEP to assist service providers with performing and interpreting ABPM (**Table 1**) [14]. In lots of techniques the roles of determining BP, informing patients concerning HBPM, and carrying out ABPM are undertaken by nurses and pharmacists; this is another reason for sustaining the transfer to a team-based medical house model for family medicine.

Table 1. Standardized protocol for ABPM [14].

• The appropriately sized cuff should be applied to the nondominant arm unless the SBP difference between arms is > 10 mm Hg, in which case the arm with the highest value obtained should be used
• The device should be set to record for a duration of at least 24 h, with the measurement frequency set at 20- to 30-min intervals during the day and 30- to 60-min intervals at night
• A patient-reported diary to define daytime (time awake), nighttime (time sleeping), activities, symptoms, and medication administration is useful for study interpretation
• Daytime and nighttime should preferrably be defined using the patient's diary. Alternatively, predefined thresholds can be used (eg, 8 AM to 10 PM for day and 10 PM and 8 AM for night)
• The ABPM report should include all of the individual BP readings (both numerically and graphically), the percentage of successful readings, the averages for each time frame (daytime, nighttime, 24 h), and the "dipping" percentage (the percentage the average BP changed from daytime to nighttime)
• Criteria for a successful ABPM study are as follows:
-at least 70% of the readings are successful and
-at least 20 daytime readings and 7 nighttime readings are successful

ABPM—ambulatory blood pressure measurement, BP—blood pressure, SBP—systolic blood pressure.

- **Long-term therapy**

All anti-hypertensive drugs need to act by decreasing the cardiac output, the peripheral vascular resistance, or both. The classes of medications most commonly utilized include the thiazide diuretics, β-blockers, ACE inhibitors, angiotensin II receptors antagonists, calcium channel blockers, α-adrenoceptor blockers, integrated α- and β-blockers, direct vasodilators, and some centrally acting medicines such as α2-adrenoceptor agonists and imidazoline I1 receptor agonists.

Lifestyle adjustment is the initial step in the therapy of high blood pressure; it consists of modest sodium restriction, weight reduction in the obese, decreased alcohol intake, and a rise in exercise. Medication therapy is required when the above measures have not succeeded or when hypertension is already at a harmful phase (Stage 3) when first recognized.

- **Drug therapy**

Diuretics

Low-dose diuretic treatment is effective and minimizes the threat of stroke, coronary heart disease, congestive heart failure, and complete mortality [15]. Whilst thiazides are most generally used, loop diuretics are also used effectively and the organization with a potassium sparing diuretic decreases the danger of both hypokalaemia and hypomagnesaemia. Even in tiny doses diuretics potentiate other antihypertensive drugs. The threat of untimely end is lowered when potassium-sparing diuretics are utilized. In the lasting, spironolactones reduce morbidity and mortality in patients with heart failure that is a common difficulty of long-lasting high blood pressure [16].

Beta-blockers

High sympathetic tone, angina, and previous myocardial infarction are good reasons for using β-blockers. As a reduced dose reduces the risk of fatigue (an unpleasant effect of β-blockade) enhancement of a diuretic or a calcium channel blocker is usually beneficial [17]. Nevertheless, β-blockade therapy is associated with signs of depression, fatigue, and sexual disorder. These side-effects need to be taken into consideration in the evaluation of the benefits of treatment.

Over the past few years β-blockers have been utilized significantly frequently in the management of cardiac arrest, a well-known problem of arterial hypertension. They work yet their introduction in the visibility of cardiac arrest needs to be extremely mindful, beginning with very low doses to avoid a preliminary worsening of heart failure [18].

Calcium channel blockers

Calcium channel blockers can be split right into dihydropyridines (e.g. nifedipine, nimodipine, amlodipine) and non-dihydropyridines (verapamil, diltiazem). Both team's lower peripheral vascular resistance yet verapamil and diltiazem have adverse 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 prep work of nifedipine cause much less sympathetic activation. Short-acting dihydropyridines show up to boost the threat of untimely end. However, the systolic hypertension in Europe (SYST-EUR) test which compared nitrendipine with sugar pill had to be stopped early as a result of significant benefits of active therapy [19].

Angiotensin converting enzyme inhibitors

ACE inhibitors are significantly being used as initial line treatment. They have relatively couple of side-effects and contraindications other than bilateral renal artery stenoses. Though ACE

preventions are effective in unilateral renovascular hypertension, there is danger of ischaemic atrophy. For that reason, angioplasty or surgical kidney artery reconstruction are better to long-term simply medical treatment. ACE inhibitors are first option representatives in diabetic hypertensive patients as they reduce the progression of renal dysfunction. In high blood pressure with heart failure, ACE inhibitors are additionally front runner medications [20].The HOPE trial has revealed that ramipril lowered the danger of cardiovascular occasions also in the lack of hypertension. Hence, this ACE inhibitor might exert a protective result by systems besides the reduction in blood pressure.

Angiotensin II receptor blockers

As angiotensin II stimulates AT1-receptors that cause vasoconstriction, angiotensin AT1-receptor antagonists are effective antihypertensive drugs. Losartan, valsartan and candesartan work and trigger less coughing than ACE inhibitors.

The LIFE research is the most recent landmark trial in hypertension. More than 9000 patients were randomized to get either the angiotensin receptor antagonist losartan or a β-blocker (atenolol). Patients in the losartan arm showed better decrease of mortality and morbidity, owing to better decrease in strokes [23].Losartan was additionally much more reliable in minimizing left ventricular hypertrophy, an independent effective threat factor for unfavorable outcome. In patients with separated systolic hypertension, the supremacy of losartan over atenolol was a lot more obvious compared to in those with systolic and diastolic hypertension. These favourable results brought about a content entitled: 'Angiotensin blockade in high blood pressure: a guarantee satisfied'. It needs to be kept in mind that the comparator in the LIFE research study

was a β-blocker, which, in the past, β-blockers were found to be no better compared to placebo in the senior [24].

α_1-Adrenergic blockers

Free from metabolic side-effects, these drugs reduce blood cholesterol and decrease outer vascular resistance. Prazosin is shorter acting than doxazosin, indoramin and terazosin. These medicines are extremely discerning for α1-adrenoceptors. Drowsiness, postural hypotension, and periodically tachycardia, can be problematic. Fluid retention could call for the enhancement of a diuretic. Phenoxybenzamine is a non-competitive α-adrenoceptor agonist utilized (in association with a β-blocker) in the management of patients with phaeochromocytoma, however recently doxazosin has been utilized effectively.

Direct vasodilators

Hydralazine and minoxidil are directly acting vasodilators. Their usage has declined because of the potential for serious side-effects (lupus syndrome with hydralazine, hirsutism with minoxidil)[21].

- **Risk management**

Along with pharmacological procedures for the control of high blood pressure, there need to be energetic treatment of those factors known to enhance the threat of hypertension. There are two distinctive measures. First, those that lower high blood pressure, for example weight decrease, decreased salt consumption, limitation of alcohol consumption, exercise, raised fruit and vegetable intake, and reduced complete and saturated fat consumption. Second, those that reduce

cardiovascular risk, for instance quiting smoking; changing saturated with polyunsaturated and monounsaturated fats; raised oily fish usage; and minimized overall fat consumption [25].

Due to the fact that hypertensive patients go to really high risk of coronary artery condition, various other therapeutic procedures consist of aspirin and statin therapies. Lose-dose aspirin works in the prevention of thrombotic events such as stroke and heart attack; this is also real in hypertensive patients whose high blood pressure is well controlled. The danger of extreme bleeding is really reduced offered blood pressure is minimized to below 150/90 mm Hg. The advantages of lipid-lowering drug therapy with statins are well established in coronary heart disease and in cerebrovascular illness, two problems often associated with arterial hypertension.

Conclusion:

The excessive pressure on your artery walls caused by high blood pressure can damage blood vessels, as well as organs in the body. Family doctor and their teams have an important duty to play in the recognition and management of high blood pressure, because it can cause mortality. Risk factors like smoking and obesity, can cause hypertension, in this case changing the lifestyle is important.

Reference:

1. Hackam DG, Quinn RR, Ravani P, Rabi DM, Dasgupta K, Daskalopoulou SS, et al. The 2013 Canadian Hypertension Education Program recommendations for blood pressure measurement, diagnosis, assessment of risk, prevention, and treatment of hypertension. Can J Cardiol. 2013;29(5):528–42. Epub 2013 Mar 29.
2. Hamer M. The anti-hypertensive effects of exercise: integrating acute and chronic mechanisms. Sports Med. 2006;36(2):109–16.

3. Brook RD, Appel LJ, Rubenfire M, Ogedegbe G, Bisognano JD, Elliott WJ, et al. Beyond medications and diet: alternative approaches to lowering blood pressure: a scientific statement from the American Heart Association. Hypertension. 2013;61(6):1360–83. Epub 2013 Apr 22.
4. Fagard RH. Exercise therapy in hypertensive cardiovascular disease. Prog Cardiovasc Dis. 2011;53(6):404–11.
5. Fagard RH, Cornelissen VA. Effect of exercise on blood pressure control in hypertensive patients. Eur J Cardiovasc Prev Rehabil. 2007;14(1):12–7.
6. Martin J. Hypertension guidelines: revisiting the JNC7 recommendations. J Lancaster General Hospital. 2008;3(3):91–7.
7. Action to Control Cardiovascular Risk in Diabetes Study, G, et al. Effects of intensive glucose lowering in type 2 diabetes. N Engl J Med 2008; 358:2545–2559.
8. Bangalore S, Kumar S, Lobach I, Messerli FH. Blood pressure targets in subjects with type 2 diabetes mellitus/impaired fasting glucose: observations from traditional and Bayesian random-effects meta-analyses of randomized trials. Circulation 2011; 123:2799–2810; 9 p following 810.
9. Lonn EM, Bosch J, Lopez-Jarmillo P, et al. Blood-pressure lowering in intermediate-risk persons without cardiovascular disease. N Engl J Med 2016; 374:2009–2020.
10. Bangalore S, et al. J-curve revisited: an analysis of blood pressure and cardiovascular events in the Treating to New Targets (TNT) Trial. Eur Heart J 2010; 31:2897–2908.
11. Bangalore S, Messerli FH, Wun CC, et al. What is the optimal blood pressure in patients after acute coronary syndromes?: relationship of blood pressure and cardiovascular events in the PRavastatin OR atorVastatin Evaluation and Infection Therapy-Thrombolysis In Myocardial Infarction (PROVE IT-TIMI) 22 trial. Circulation 2010; 122:2142–2151.
12. Bangalore S, Gong Y, Cooper-DeHoff RM, et al. 2014 Eighth Joint National Committee panel recommendation for blood pressure targets revisited: results from the INVEST study. J Am Coll Cardiol 2014; 64:784–793.
13. McEvoy JW, Chen Y, Rawlings A, et al. Diastolic blood pressure, subclinical myocardial damage, and cardiac events: implications for blood pressure control. J Am Coll Cardiol 2016; 68:1713–1722.
14. Dickson RC, Gaebel K, Zizzo A, Neimanis I, Bridge M, Corsini J, et al. Self-reported physician adherence to guidelines for measuring blood pressure. J Am Board Fam Med. 2013;26(2):215–7.
15. 6. Veiga EV, Nogueira MS, Cárnio EC, Marques S, Lavrador MA, de Moraes SA, et al. Assessment of the techniques of blood pressure measurement. Arq Bras Cardiol. 2003;80(1):89–93. Epub 2003 Feb 19.
16. 7. O'Brien E, Atkins N, Stergiou G, Karpettas N, Parati G, Asmar R, et al. European Society of Hypertension International Protocol revision 2010 for the validation of blood pressure measuring devices in adults. Blood Press Monit. 2010;15(1):23–38.
17. Fagard RH, Staessen JA, Thijs L, Gasowski J, Bulpitt CJ, Clement D, et al. Response to antihypertensive therapy in older patients with sustained and non-sustained systolic

hypertension. Systolic Hypertension in Europe (Syst-Eur) Trial Investigators. Circulation. 2000;102(10):1139–44.

18. 17. Stergiou GS, Kollias A, Zeniodi M, Karpettas N, Ntineri A. Home blood pressure monitoring: primary role in hypertension management. Curr Hypertens Rep. 2014;16(8):462.
19. 18. Niiranen TJ, Hänninen MR, Johansson J, Reunanen A, Jula AM. Home-measured blood pressure is a stronger predictor of cardiovascular risk than office blood pressure: the Finn-Home study. Hypertension. 2010;55(6):1346–51. Epub 2010 Apr 12.
20. James PA, Oparil S, Carter BL, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). JAMA. 2014 Feb 5. 311 (5):507-20. [Medline].
21. Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988-2000. JAMA. 2003 Jul 9. 290(2):199-206.
22. Bianchi S, Bigazzi R, Campese VM. Microalbuminuria in essential hypertension: significance, pathophysiology, and therapeutic implications. Am J Kidney Dis. 1999 Dec. 34(6):973-95.
23. Madhur MS, Lob HE, McCann LA, et al. Interleukin 17 promotes angiotensin II-induced hypertension and vascular dysfunction. Hypertension. 2010 Feb. 55 (2):500-7.
24. Webster J, Petrie JC, Jeffers TA, Lovell HG. Accelerated hypertension--patterns of mortality and clinical factors affecting outcome in treated patients. Q J Med. 1993 Aug. 86(8):485-93.
25. Culleton BF, Larson MG, Kannel WB, Levy D. Serum uric acid and risk for cardiovascular disease and death: the Framingham Heart Study. Ann Intern Med. 1999 Jul 6. 131(1):7-13.