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Review article

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Cardiovascular Risk Factors

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Abstract

Cardiovascular illnesses (CVDs) stay a main cause of morbidity and mortality worldwide. Various danger factors contribute to the improvement and development of CVDs, encompassing each modifiable and non-modifiable element. This abstract pursues to spotlight the important thing cardiovascular chance factors and their impact on coronary heart fitness.

***Age and Gender:** Advancing age and being male are non-modifiable risk factors associated with increased CVD threat. Guys are usually at a better chance than premenopausal women; but, this difference decreases put up-menopause. High blood pressure: extended blood strain is a sizeable modifiable chance issue for CVDs. control hypertension damages blood vessels, selling atherosclerosis and increasing the threat of heart assault, stroke, and coronary heart failure. Dyslipidemia: high degrees of LDL cholesterol and triglycerides, coupled with low stages of HDL cholesterol, make contributions to atherosclerosis and plaque formation, main to coronary artery disorder and other cardiovascular complications.*

***Smoking:** Cigarette smoking is a main modifiable risk element for CVDs. It damages blood vessels, accelerates atherosclerosis, and decreases oxygen delivery to tissues, heightening the risk of heart disease and stroke. Diabetes Mellitus: each type 1 and kind 2 diabetes drastically raises the danger of CVDs because of insulin resistance, inflammation, and metabolic abnormalities that adversely affect blood vessels and the heart.*

***Obesity: -** extra body weight, specifically abdominal adiposity, increases the likelihood of CVDs using contributing to insulin resistance, hypertension, dyslipidemia, and inflammation. Bodily state of being inactive: Sedentary lifestyle and shortage of regular bodily pastimes are connected to weight problems and numerous metabolic disturbances that sell CVD improvement. Circle of relatives records: A high-quality own family record of premature CVD increases an individual's chance, suggesting a capacity genetic predisposition to heart disease.*

***Weight-reduction plan:** - consuming a diet excessive in saturated and Tran's fat, salt, and introduced sugars even as missing fruits, veggies, and whole grains can make contributions to the CVD threat. Strain and intellectual health: chronic pressure, depression, and anxiety can impact CVD risk via numerous mechanisms, consisting of unhealthy coping behaviors and hormonal imbalances. Alcohol consumption: whilst mild alcohol consumption may also have some cardiovascular blessings, immoderate ingesting can boost blood strain and make contributions to coronary heart muscle damage. Efforts to mitigate cardiovascular hazard factors should recognition on lifestyle adjustments, consisting of ordinary workouts, a heart-wholesome diet, smoking cessation, stress management, and blood pressure and cholesterol control. Early identification of chance factors and their effective control can play an important role in decreasing the burden of cardiovascular illnesses and improving typical coronary heart health.*

Keywords: Cardiovascular Health, Risk Factors for Heart Disease, Identifying Cardiovascular Risks Heart Disease Risk Factors, Modifiable Cardiovascular Risk Factors, Non-Modifiable Cardiovascular Risk Factors, Managing Cardiovascular Risk Preventing Heart Disease, Understanding Heart Health Risks, Cardiovascular Risk Assessment

Introduction

Hypertension often accompanies diabetes mellitus, both type 1 (T1DM) and type 2 (T2DM). The association between the two conditions has long been recognized. In 1923, the Swedish physician Eskil Kylin described a syndrome of diabetes, hypertension, and hyperuricemia, which are now regarded as aspects of the broader "metabolic syndrome" that has been linked to insulin resistance (IR) [1-3]. The relationship between diabetes and hypertension is complex. Both are common and so are likely to be associated by chance, but in some instances, they may have a common cause; moreover, hypertension can develop as a consequence of diabetic nephropathy, while some drugs used to treat hypertension can induce diabetes in susceptible subjects. Hypertension is important because, like diabetes, it is a major cardiovascular risk factor and one that synergizes with the deleterious effects of diabetes. It is also a risk factor for microvascular complications: nephropathy and retinopathy. The management of hypertension in diabetes has been widely debated, and there is still a need to agree on treatment targets and strategies. During the last decade, several well-constructed trials have added considerably to the evidence base [4-8]. Demonstrating convincingly the benefits of lowering blood pressure (BP), but also highlighting how difficult this can be to achieve in practice.

Size of the problem: - Hypertension is widely defined according to the World Health Organization/International Society of Hypertension (WHO/ISH) criteria (Table 40.1). People with diabetes are still at risk of macro vascular and microvascular complications at BP levels below these thresholds and the treatment target range are therefore lower (130 – 140/80 – 85 mmHg). Overall, hypertension (according to the WHO criteria) is up to twice as common in people with diabetes as in the general population [9]. In white Europeans, 10 – 30% of subjects with T1DM and 60 – 80% of those with newly diagnosed T2DM are hypertensive [10]. There are racial and ethnic differences in the Pre valence of hypertension, which presumably are at least partly genetically determined: for example, hypertension (and macro vascular disease) is less frequent among the Pima Indians and Mexican - Americans [11]. Impaired glucose tolerance (IGT) is also associated with hypertension (20 – 40% of cases), perhaps reflecting the common origins of these aspects of the metabolic Syndrome [12]. There is evidence that the true prevalence of hypertension is increasing in the diabetic population (especially T2DM) after allowing for the greater number of cases identified.

Through improved screening and the lowering of thresholds for treatment of BP [13]. The causes probably include the rising prevalence of obesity and longer survival of older people with diabetes. Causes of hypertension in diabetes Associations among high blood pressure and diabetes are indexed in Table 40.2. Vital hypertension and remoted systolic hypertension are each common among the non-diabetic populace (particularly in the elderly). Its miles anticipated that important high blood pressure money is owed for about 10% of cases in people with diabetes. Other crucial reasons are hypertension that coexists with IR, obesity, and IGT inside the metabolic syndrome, and high blood pressure secondary to diabetic nephropathy, as discussed in detail below Hypertension in the metabolic syndrome.

This syndrome includes IR, IGT (which includes T2DM), and characteristic dyslipidemia. Hypertriglyceridemia, low high - density lipoprotein (HDL) cholesterol, and raised low-density lipoprotein (LDL), with an extra of small dense LDL debris-truncal obesity, pro coagulant modifications (raised plasminogen activator inhibitor 1 and fi fibrinogen stages) and hyperuricemia [2, 14, 15]. As those abnormalities are all chance factors for atherogenesis, the syndrome is completed through a marked tendency to vascular aging main to macro vascular ailment, in particular coronary heart disorder (CHD) and stroke (Figure 40.1). IR has been proposed via Reaven [2]. DeFronzo and Ferrannini [14] and others [15] to be an essential reason for high blood pressure and cardiovascular sickness (CVD) as well as T2DM. IR is partially genetically determined, and bought factors which include obesity, physical inactivity and perhaps malnutrition in utero and during early infancy may also contribute [16]. In support of the latter, family studies have revealed a correlation between the BP of the mother and her offspring that appear to be non - hereditary in origin; early growth retardation is suggested to program abnormal development of the vasculature as well as the tissues that regulate glucose homeostasis IR is closely associated with high BP in both humans and animals. Experimental induction of IR (e.g. feeding rats with fructose) is accompanied by a rise in BP. More persuasively, an inverse relationship has been demonstrated in humans between BP and insulin sensitivity [17] (Figure 40.2). Various mechanisms have been proposed to explain how IR and/or the hyperinsulinemia that accompanies it could increase BP (Figure 40.3). First, there is some evidence that insulin is an endothelium-dependent vasodilator, releasing nitric oxide (NO) from the endothelium, which relaxes vascular smooth muscle [18, 19]. Blunting of this the effect, caused by insensitivity to the action of insulin on the endothelium as well as on metabolically important tissues, could contribute to the increased peripheral resistance that is the hallmark of hypertension in obesity and T2DM. Impaired endothelium-mediated vasodilation is associated with IR states and may have a key role in the initiation and progression of atherosclerosis [20]. By contrast, insulin also has several actions that tend to raise BP and there is some evidence that these are accentuated in IR states, presumably because sensitivity is preserved to the effects of the raised insulin levels. Insulin acts on the distal renal tubule to retain Na + ions and water [20, 21]. An effect that still operates in IR subjects [22]. And so could contribute to the rise in total body Na + content that occurs in obesity and T2DM [23]. Insulin also stimulates the cell membrane Na + – K + ATPase, which would raise intracellular Na + concentrations in vascular smooth muscle and, increasing systolic Ca 2+ levels would enhance contractility and increase peripheral resistance [22, 23]. Through its effects on the CNS, insulin may stimulate sympathetic outflow. Theoretically, this could also increase BP, although direct evidence in humans is lacking [22, 24]. Finally, insulin may stimulate the proliferation of vascular smooth muscle cells, which could lead to medial hypertrophy and

increased peripheral resistance [22, 25].

Hypertension and diabetic nephropathy this association is most obvious in young patients with T1DM, in whom the presence of hypertension is strikingly related to renal damage and even minor degrees of proteinuria. BP begins to rise when the urinary albumin excretion (UAE) enters the Microalbuminuria range (> 30 mg/24 hours) and is usually over the WHO threshold when UAE reaches the Microalbuminuria stage (> 300 mg/24 hours) [26]. The association may be partly genetically determined: subjects with diabetes and Microalbuminuria commonly have parents with hypertension and may also inherit over activity of the cell - membrane Na + – H + pump (indicated by increased Na + – Li + counter-transport in red blood cells),which would tend to raise intracellular Na + concentrations and thus increase vascular smooth muscle tone [27].

The basic mechanisms of hypertension include decreased Na +excretion with Na + and water retention. Peripheral resistance is increased, which raised intracellular Na + will contribute. The role of the renin-angiotensin-aldosterone system (RAS) is uncertain, as both increased and decreased activity has been reported [28, 29]. These discrepancies may be explained by differences in diet, treatment, metabolic control, and the type and duration of diabetes. Na + retention and hypertension would be predicted to suppress the RAS, while renin levels may be influenced by other complications of diabetes: renal tubular acidosis type 4 causes hyporeninemic hypo aldosteronism and neuropathy can also lower plasma renin, while renin may be raised in retinopathy and advanced nephropathy. Patients with Microalbuminuria who are insulin-resistant appear to be particularly susceptible to hypertension [30]. Impact of hypertension on diabetes a large proportion of hypertensive people with diabetes show signs of cardiovascular aging and target-organ damage [10]. Hypertension, as an independent risk factor for atherogenesis, synergizes with the effects of diabetes and significantly increases the development and progression of CHD, cerebrovascular and peripheral vascular disease. Overall, the effects of hypertension on deaths from CHD are increased by 2 – 5 times in people with diabetes, with the greatest increase occurring at the lowest BP levels (Figure 40.4). The deleterious effects of hypertension on left ventricular function are also accentuated by the presence of diabetes. These include impaired left ventricular relaxation [31]. And increased left ventricular mass [32].

The latter being an independent predictor of premature death from CHD. Hypertension also predisposes to the development of certain microvascular complications, particularly nephropathy and end - stage renal failure (ESRF), for which the risk is increased by 2 – 3times Hypertension is also a risk factor for retinopathy, as has been confirmed by the beneficial effects of improved BP control in patients with T2DM, reported by the UK Prospective Diabetes Study (UKPDS) [4].

Screening for hypertension in diabetes: - As the two conditions are so commonly associated, people with diabetes must be regularly screened for hypertension and vice versa. Hypertensive patients, especially if obese or receiving treatment with potentially diabetogenic drugs, should be screened for diabetes at diagnosis and during follow-up. Should hyper guycamera be detected, potentially diabetogenic antihypertensive drugs should be reduced or changed to others or used in combinations that do not impair glucose tolerance, and normoglycemia can then often be restored.

All people with diabetes should have their BP checked at diagnosis and at least annually thereafter. This is especially important in those with other cardiovascular risk factors, such as nephropathythy (which is associated with a substantial increase in the cardiovascular mortality rate), obesity, dyslipidemia, smoking, or poor glycemic control.

Measurement of blood pressure: - BP should be measured with the patient in the supine or sitting position, with an accurate sphygmomanometer and a cuff of appropriate size (i.e. wider for obese subjects with an arm circumference of > 32 cm). Systolic and diastolic BP should be recorded, to the nearest 2 mmHg if using a manual sphygmomanometer, from phases I and V (i.e. appearance and final disappearance of the sounds of Korotkoff). Usual precautions should be taken to ensure reliability and avoid " white coat " stress effects which can acutely raise BP. Conditions should be quiet and relaxed, and at least two readings should be taken initially and then repeated at intervals over weeks or months to determine the subject's typical values and any trend to change.

Office BP could be complemented by repeated home BP recordings. BP should also be checked with the patient in the upright position (1 minute after standing), because there may be a significant postural fall (> 20 mmHg systolic) in patients with diabetic autonomic neuropathy, the elderly, or those treated with vasodilators or diuretics. Marked postural hypotension, which can coexist with supine hypertension, may indicate the need to change or reduce antihypertensive medication, especially if symptoms are provoked.

Ambulatory BP monitoring over 24 hours may be useful in some cases to exclude "white coat" effects, and in patients with early nephropathy who have nearly normal BP during the day, but who may be at risk of hypertensive tissue damage because they fail to show the physiologic BP dip during sleep [33].

Diagnosis of hypertension in diabetes the criteria issued in 1999 by WHO and ISH [34] define hypertension as an office BP exceeding 140/90 mmHg (Korotkoff I – V), and borderline hypertension as being below these limits but above 130 mmHg systolic and/or 85 mmHg diastolic (Figure 40.5) [34]. Established hypertension is diagnosed when readings consistently exceed 140/90 mmHg over several weeks, or when the BP is very high (diastolic BP > 110 mmHg), or when there are clinical signs of tissue organ damage from long-standing hypertension.

It is clear from numerous epidemiologic studies that the WHO/ ISH threshold is too high in people with diabetes because of their

the additional risk of both macro vascular and microvascular disease, and that there are definite benefits to treating micro albuminric subjects whose diastolic BP is < 90 mmHg [35]. Various other expert bodies have suggested alternative, generally lower target levels (Figure 40.5). A consensus would be to aim for a BP of less than 130 – 140 mmHg systolic and below 80 – 85 mmHg diastolic, and to treat any subject whose BP is consistently above one or both of these thresholds.

Management of hypertension in diabetes: - Strict BP control is the primary goal of treatment. In recent years, target treatment levels have declined progressively to the current recommendation of a mean office BP less than 130 – 140/80 – 85 mmHg, for all patients who can tolerate this without side effects such as orthostatic reactions or compromising arterial circulation in critical vascular beds. Recent observations indicate that subgroups of susceptible patients might exist who will not tolerate a dramatic BP reduction below 130 mmHg systolic BP and so caution should be exercised. Management begins with lifestyle modification, but few patients respond to this alone, and most will require more than one antihypertensive drug to control BP adequately [4, 5].

Non - pharmacologic Treatment: - The treatment for high blood pressure in sufferers with diabetes needs to be primarily based on dependent lifestyle intervention. This means weight discount or weight stabilization inside the overweight, sodium limit, weight-reduction plan modification, and ordinary bodily exercise (moderate intensity, forty – 60 mins, 2 – three times weekly). Nutritional consumption of saturated fats has been related to impaired in insulin sensitivity and must therefore be reduced [37]. Alcohol needs to be constrained to 2–3 units/day in guys and 2 units/day in girls but left out altogether if high blood pressure proves difficult to manipulate. Smoking causes an acute boom in blood pressure and a greater variability average [38]. Smoking cessation is especially critical, as smoking now not simplest speeds up the development of atherosclerosis and vascular growing old, but also impairs insulin sensitivity [39]. and worsens albuminuria [40]. Treatment with nicotine supplementation for 4 – 6 weeks (chewing gum or patches), bupropion, or varenicline may be useful.

When followed in completely by using the patient, way of life modification can be extraordinarily powerful. The above measures can decrease systolic and diastolic BP by using eleven and 8 mmHg, respectively [41] – as much as many antihypertensive drugs – and every so often enough to obviate the need for drug therapy. Weight reduction in overweight sufferers can similarly reduce BP.

Antihypertensive drug therapy: - Numerous drugs are available to lower BP, but some are better suited than others to the particular needs of subjects with diabetes because of their favorable or neutral effects on glucose metabolism and other factors. Most patients (at least two-thirds) will require combinations of antihypertensive drugs to control BP – an average of around three different drugs in two large studies [4, 5]. Accordingly, the clinician must be able to use a wide variety antihypertensive pills and choose combinations that achieve maximum pharmacological synergy. Usually aggregate therapy the way lower doses of character pills can often be used, accordingly reducing the risk of their harmful effects.

Diuretics: - Diuretics are often powerful antihypertensives for human beings with diabetes, in which the total body sodium burden is increased and increased extracellular fluid volume [42]; but, diuretics that increase urinary losses of potassium and magnesium can worsen hyperglycemia because insulin secretion is impaired by potassium depletion and insulin sensitivity in peripheral tissues may additionally also, reduce [43]. taking excessive doses of thiazide diuretics - equivalent to ≥ 5 mg/day bendroflu umethiazide (bendrofluazide) – it is said to increase the likelihood of developing diabetes in hypertensive patients up to threefold; this does not happen low doses (up to 2.5 mg/day of bendroflumethiazide) [44]. Potassium depletion is particularly excessive with an overdose of chlorthalidone (Chlorthalidone), less so with furosemide (Frusemide) and bedroll ume-thiazide and reportedly negligible with indapamide.

This mechanism is irrelevant for C - peptide-negative subjects with T1DM who are dependent on exogenous insulin.In addition, thiazides may worsen dyslipidemia [45], even when it is low doses may pose little danger. Thiazides were extra associated with gout and impotence and usually avoided middle-aged men with diabetes and hyperuricemia or erectile disorder; however, some evidence suggests that the chances of erectile dysfunction may have been overestimated. Diuretics can induce hyperosmolar hyperglycemia syndrome and must be prevent or use at the lowest effective dose in patients with a records of this hardship. Diuretics are effective in preventing CVD in old subjects with T2DM and systolic high blood pressure [46]. Butone observational view suggested that the use of diuretics increased cardiovascular mortality in hypertensive patients with T2DM who were still hyperglycemic regardless of treatment [47].

By default, these pills are strong and safe when used in diabetic patients. Diuretics suitable for use in diabetic high blood pressure include furosemide, bendroflumethiazide (≤ 2.5 mg/day), hydrochlorothiazide, spironolactone, and indapamide. Low doses must be used, sometimes mixed with potassium supplements or potassium-sparing tablets, including amiloride. If diuretics are ineffective, they should be mixed with some other first-line drug (e.g. angiotensin II receptor inhibitor or antagonist [ARB]).then given in an extended dose. Spironolactone is exceptional when not combined with an ACE inhibitor, as this increases the risk of hyperkalemia. Furosemide is useful in patients with impaired renal function (serum creatinine > 150 μ mol/L) or edema.

During this, serum urea, creatinine, and potassium should be checked initiation of diuretic therapy and every 6 – one year thereafter, as risk disorders of plasma potassium levels may develop, specifically in patients with diabetes and impaired renal function.
β - Adrenergic blocking agents Beta-blockers may significantly lower BP levels in patients with diabetes and hypertension, even

though renin release (a major the target for these drugs) is commonly reduced in diabetes because of Na + and fluid retention. These drugs are often ineffective in Afro - Caribbean patients, who commonly have low renin hypertension. Other mechanisms of action that reduce BP include reductions in heart rate and cardiac output via interaction with β1 - and β2 - receptors in the myocardium and the vessel wall.

Like diuretics, beta-receptor blockers may aggravate both hyperglycemia and dyslipidemia [48]. These effects depend on both the dosage and the degree of selectivity of the individual drug. The hyperglycemic effect is attributed to the inhibition of β2 - adrenergic-mediated insulin release and decreased insulin action in peripheral tissues; the long-term risks of a person without diabetes developing the disease may be increased by sixfold [49] and even more, if given together with thiazides. Some studies suggest that the hazards of both hyperglycemia and hyperlipidemia have been exaggerated and maybe both dose-dependent and secondary to weight gain [50]. The metabolic side effects of beta-blockers can be reduced by using low dosages combined with other agents, particularly dihydropyridine calcium channel antagonists (CCAs), or by intensifying non - pharmacologic efforts to decrease weight and improve physical activity.

Beta-blockers have other side effects relevant to diabetes. They may interfere with the counter-regulatory effects of catecholamines released during hypoglycemia, thereby blunting manifestations such as tachycardia and tremor and delaying recovery from hypoglycemia [51]. In clinical practice, however, this rarely presents a serious problem, especially when cardio selective β1 - blockers are used. Beta-blockers may also aggravate impotence, and are generally contraindicated in second - or third-degree atrioventricular (AV) heart block, severe peripheral vascular disease, asthma, and chronic airway obstruction. Recent studies have shown that certain beta-blockers such as metoprolol and carvedilol [52, 53]. Can be used favorably in cardiac failure in patients with diabetes, as shown in the Metoprolol CR/XL Randomized Intervention Trial in congestive heart failure (MERIT - HF) study, in which 25% of the patients had diabetes [52].

Atenolol is a commonly used drug, as it is cardio selective and water-soluble, which reduces CNS side effects and renders its metabolism and dosage more predictable. It is mostly effective as a single daily dose, which probably encourages compliance. In the UKPDS, its effect was comparable to that of the ACE inhibitor, captopril [54]; however, it should be kept in mind that the stroke preventive effect of atenolol is 16% less than other antihypertensive drugs, based on data from meta-analyses. Metoprolol is an alternative, in moderate dosages. Both non-selective and selective beta-blockers are effective in the secondary prevention of myocardial infarction (MI) after an initial event in patients with diabetes [55]. Metoprolol or carvedilol may be indicated in patients who also have heart failure [52, 53]. And beta-blockers in general, are useful in patients who also have angina or tachyarrhythmias.

Calcium-channel antagonists: - These useful vasodilator agents do not generally worsen metabolic control when used at conventional dosages, although sporadic cases of hyperglycemia have been reported after starting a calcium - channel antagonist (CCA) of the dihydropyridine class [56]. This may be caused by the inhibition of insulin secretion (a calcium-dependent process) in susceptible patients or a compensatory sympathetic nervous activation, which antagonizes both insulin secretion and action, following vasodilation. CCAs have a slight negative inotropic effect and are contraindicated in significant cardiac failure; they often cause mild ankle edema, but this is caused by relaxation of the peripheral precapilary sphincters and raised capillary pressure rather than to right ventricular failure. Because of their potent vasodilator properties, these drugs can cause postural hypotension and can aggravate that was brought about by autonomic neuropathy. Non -dihydropyridine CCAs (e.g. verapamil) reduce proteinuria in diabetic nephropathy, but this effect is not seen with dihydroxy-riding derivatives such as nifedipine, amlodipine, felodipine and isradipine [57].

Because of their other cardiac actions, these drugs are particularly indicated in hypertensive patients who also have angina (e.g. sustained-release nifedipine and diltiazem) or supra ventricular tachycardia (e.g. verapamil). Their vasodilator properties may also be beneficial in peripheral vascular disease. CCAs are ideally combined with selective β1 - blockers, but the specific combination of verapamil and beta-blockers (especially together with digoxin) must be avoided because of the risk of conduction block and asystole. Overall, CCAs appear less or similarly cardioprotective but better at preventing stroke than either beta-blockers or thiazide diuretics [58, 59]. Amlodipine given once daily is an evidence-based and convenient preparation for general use, and felodipine, isradipine and sustained-release nifedipine is a suitable alternative.

Angiotensin-converting enzyme inhibitors:-

ACE inhibitors may be used in diabetic hypertension, even in cases where the general RAS is not activated as the drugs may interfere with local angiotensin action in specific target tissues. When used alone, however, these agents have a limited hypotenusesive action in many black patients, who tend to have suppressed RAS activity.

ACE inhibitors have no adverse metabolic effects and may even improve insulin sensitivity [60]; hypoglycemia has rarely been reported [61]. These drugs are particularly beneficial in diabetic nephropathy by reducing albuminuria and possibly delaying the progression of renal damage [62]. Their antiproteinuric effect may be caused specifically by the relaxation of the efferent arterioles in the Glomerulus, which are highly sensitive to vasoconstriction by angiotensin II, thus reducing the intra glomerular hypertension that is postulated to favor albumin filtration; however, the importance of this mechanism remains controversial [63]. ACE inhibitors are also indicated in cardiac failure, in combination with relatively low dosages of diuretics. A dry cough is reported by 10 – 15% of patients treated with ACE inhibitors, because these drugs also interfere with the breakdown of kinins in the bronchial epithelium.

Changing to another.

ACE inhibitor or an ARB may avoid this problem. ACE inhibitors occasionally precipitate acute renal failure, particularly in the elderly and in subjects taking non - steroidal anti-inflammatory drugs (NSAIDs), or who have bilateral renal artery stenosis. Other side effects (rashes, neutropenia, taste disturbance) are unusual with the low dosages currently recommended, but become more prominent in renal failure. Because ACE inhibitors cause potassium retention, they should not generally be taken concurrently with potassium-sparing diuretics (spironolactone and amiloride) or potassium supplements. Serum creatinine and potassium levels should be monitored regularly, especially in patients with renal failure or type 4 renal tubular acidosis, in whom hyperkalemia can rapidly reach dangerous levels.

Ramipril, enalapril, captopril, lisinopril, and perindopril are all established ACE inhibitors that are suitable for use in people with diabetes; enalapril, lisinopril, perindopril, and ramipril are given once daily for hypertension. The first dose of an ACE inhibitor should be small and taken just before bedtime to minimize postural hypotension, which may be marked in subjects receiving diuretics or on a strict sodium-restricted diet. The same problem may arise in patients with autonomic neuropathy. ACE inhibitors are now recommended in patients with left ventricular dysfunction following MI (see Chapter 41). Ramipril has been shown to prevent cardiovascular morbidity and mortality in high-risk patients with diabetes, with or without pre - existing ischemic heart disease [64].

Angiotensin II type 1 receptor blockers: - This promising new class includes losartan, irbesartan, valsartan, candesartan, and telmisartan, which act on the AT1 receptor to reduce BP. They are metabolically neutral [65] and unlike ACE inhibitors, do not cause cough. They are effective antihypertensive agents in people with diabetes [66] and have been demonstrated to slow the progression of nephropathy in patients with diabetes and different degrees of albuminuria (at RENAAL, IDNT and PRIME - 2 studies) [67-69]. Losartan has also been shown (in a subgroup of the LIFE study) to be superior to atenolol in the reduction of both cardiovascular endpoints (by 25%) and overall mortality (by 40%) in high-risk T2DM patients with hypertension and left ventricular hypertrophy [70]. Interestingly, the combination An ACE inhibitor (lisinopril) with an AT1 antagonist (candesartan) was more effective in lowering BP than either agent alone and SAE in patients with T2DM [71]. But lately In the ONTARGET study, no extra benefits on cardiovascular endpoints were noted for the combination of telmisartan and ramipril compared to monotherapy [72].

α1 - Adrenoceptor antagonists: - α1 - Blockers can lower BP effectively and also improve dyslipidemia and insulin sensitivity. Doxazosin is normally well tolerated, especially in combination therapy; side effects include nasal congestion and postural hypotension. Doxazosin has been reported to be inferior to the diuretic chlortalidone in the prevention of stroke and heart failure [73].

Treatment strategies: - In general, lifestyle modification should be tried initially for 3 months or so. If moderate hypertension (diastolic BP > 100 mmHg, or systolic BP > 160 mmHg) or signs of hypertensive tissue damage are present, then drug therapy should be started at the outset. Initially, monotherapy with one of the first - line drugs suggested above should be used, the choice being influenced by other factors such as the coexistence of angina, heart failure, or nephromathy. All drug treatment should aim for being evidence-based and cost-effective in the individual patient.

Hypertension in T1DM:- ACE inhibitors are especially suitable if the patient has albuminuria or more advanced stages of diabetic nephropathy. Diuretics, β1 - selective blockers and CCAs are equally valid alternatives with regard to BP reduction. If renal function is moderately impaired (serum creatinine values > 150 μ mol/L), thiazide diuretics become less effective, and furosemide or other loop diuretics should be-used instead; however, in established ESRF (serum creatinine > 500 μ mol/L) furosemide may be toxic, and dialysis must be started. In some patients, hypoglycemia attacks may be masked by the use of beta-blockers.

Hypertension in T2DM:- BP control is generally more important than the choice of individual drugs. First-line agents, according to evidence from clinical studies, are ACE inhibitors, ARBs, beta-blockers, low-dose thiazide diuretics (in the elderly), furosemide and CCAs [4-8]. Ramipril has evidence-based support for its use in patients with T2DM because of its high cardiovascular risk [64]. Beta - blockers (in combination with low-dose aspirin) are indicated as secondary prevention for patients who have had a MI, as long as no serious contraindications are present. Low doses of thiazide diuretics are useful in elderly patients with diabetes, as this class of drugs has proven efficacy in preventing stroke and all-cause mortality in elderly hypertensive patients [8].

α1 - Blockers may be used as part of combination therapy, especially in patients with dyslipidemia (high triglycerides and low HDL cholesterol levels) and prostatic hyperplasia. Indapamide is well tolerated and has no metabolic side effects. Spironolactone may also be of value [74]. Especially for elderly obese female patients with hypertension and hypovolemia with a low renin profile.

Combination therapy: - Combination therapy is needed in most people with diabetes (especially those with T2DM) to achieve satisfactory BP control [4, 5]. It is often better to use low-dose combinations than to increase dosages of single agents, as side effects are commonly dose-dependent. As already mentioned, potassium-sparing agents (spironolactone and amiloride) should not be combined with an ACE inhibitor, because of the increased risk for hyperkalemia. Certain combinations of antihypertensive drugs have proved very safe and effective in low to moderate doses, e.g. ACE inhibitor plus a diuretic, for example in the ADVANCE study [75]. CCA plus ACE inhibitor, for example in the ACCOMPLISH study [76]. selective β1 - blocker plus CCA; or β1 - blocker plus α1 - blocker. In some high-risk patients, a combination treatment could also be considered as initial therapy.

Special considerations in ethnic groups: - Hypertension in diabetes represents a serious medical problem in many ethnic groups, such as African - Americans [77]. In non - white European patients, beta-blockers, and ACE inhibitors are often less effective at lowering BP because the RAS is already underactive. Diuretics and CCAs are often drugs to be preferred, particularly in African - Americans [78]. The outcome of treating hypertension in diabetes it has long been recognized that effective treatment of hypertension can slow the progression of diabetic nephropathy, lowering UAE and decreasing the rate of fall of the GFR [79].

The assumptions that improved BP control would improve cardiovascular and other prognoses in T2DM have been confirmed by the UKPDS [4]. In this study, tighter BP control (averaging 144/82 mmHg) for over 8 years led to significant improvements in several outcomes, compared with less strict control that averaged 154/87 mmHg (Table 40.4). Interestingly, the most powerful effects were related to microvascular complications (retinopathy and nephropathy), although significant reductions were seen in the risk of stroke (44%) and heart failure (56%). MI and peripheral vascular disease showed non - significant reductions (Table 40.4; Figures 40.8 and 40.9).

Overall, therefore, tight BP control has been proven to provide substantial benefits for hypertensive patients with diabetes. Moreover, this treatment strategy seems to be cost-effective, at least according to the health economics analyses in the UKPDS [80]. However, it must be kept in mind that these benefits will not lastly, if a continuous BP reduction cannot be achieved long-term, as shown by the 10-year follow-up of the UKPDS [81].

2. Research method

Research on cardiovascular threat factors has in all likelihood involved a combination of observational research, medical trials, and records evaluation. The intention of the studies changed into picking out and taking a look at various factors that contribute to the development and improvement of cardiovascular ailments.

3. Result

Studies effects may additionally display screen several key cardiovascular risk factors which might be associated with an extended probability of developing coronary heart sickness or the prevalence of unfavorable cardiovascular events. These danger factors

3.1 Might also encompass

Excessive blood stress (excessive blood strain): improved blood pressure levels are a massive danger issue for cardiovascular sickness together with heart assault and stroke.
Excessive LDL cholesterol: multiplied stages of LDL cholesterol ("terrible" LDL cholesterol) and reduced degrees of HDL LDL cholesterol ("correct" LDL cholesterol) can contribute to the improvement of atherosclerosis and coronary artery disease.

Smoking: Tobacco smoking is strongly related to an improved threat of cardiovascular ailment due to its risky outcomes on blood vessels and the coronary coronary heart.
Diabetes: each kind 1 and sort 2 diabetes is related to an improved chance of cardiovascular complications.

Weight problems: - more frame weight, particularly inside the belly area, can reason high blood stress, insulin resistance, and exceptional metabolic problems that growth cardiovascular danger. Sedentary manner of lifestyles: loss of normal bodily interest can contribute to weight gain, excessive blood pressure, and different threat elements for coronary heart illness.

Circle of relatives statistics: - A circle of relatives facts of cardiovascular sickness can also mean a genetic predisposition to such conditions. Dangerous diet plan: A healthy eating plan excessive in saturated fats, Trans fats, salt, and brought sugars can make contributions to various threat elements for cardiovascular disorder. Pressure: chronic stress can bring about dangerous coping mechanisms (eg, overeating, smoking) and advanced blood pressure, every of that may impact cardiovascular fitness.

4. Discussion

The discussion of the research results would involve interpreting the findings and placing them in the context of existing knowledge in the field. Researchers would likely discuss the strength of the associations between each risk factor and cardiovascular diseases, as well as the potential mechanisms through which these risk factors exert their effects. Additionally, the research might explore how multiple risk factors can interact and compound their effects on cardiovascular health. For example, obesity may worsen the impact of hypertension or diabetes on the heart. Understanding these interactions is crucial for developing effective prevention and treatment strategies.

Furthermore, researchers might discuss the public health implications of their findings. Identifying modifiable risk factors can guide policymakers and healthcare professionals in designing interventions to reduce the burden of cardiovascular diseases in the population. These interventions could include lifestyle modifications, smoking cessation programs, promoting physical activity, and healthier eating habits. It's important to note that the research findings may have limitations, such as potential confounding factors, sample size issues, or the specific population studied. Researchers would likely acknowledge these limitations and suggest avenues for further investigation to strengthen the evidence. Overall, the research on cardiovascular risk factors is vital for enhancing our understanding of the disease's pathophysiology and guiding preventive measures and treatments to reduce the global burden of cardiovascular diseases.

5. Conclusions

The diagnosis and treatment of hypertension are of great importance for the person with diabetes [34, 36, 82, 84]. The treatment targets are demanding and require considerable effort from both patient and physicians, but the benefits are now undisputed. New antihypertensive drugs are constantly being introduced but have to

prove themselves for both efficacy and tolerability. Even some antidiabetic drugs appear to lower BP as well as blood glucose [85]. But safety concerns are important. In the future, the application of cardiovascular genomics may substantially change the approach to treating hypertension in diabetes [86]. Aiming at tailoring treatment according to the genotype of the individual patient. In addition, further large-scale studies with large numbers of hypertensive patients with T2DM are awaited [87]. In the recent ACCORD - Blood pressure study [88].

There was no significant the difference in the primary composite outcome of cardiovascular events between patients randomized to achieve a systolic blood pressure goal below 120 mmHg versus below 140 mmHg, even if a reduction in stroke was noticed (secondary end-point) in the intensive arm. This means that the optimal blood pressure goal for patients with hypertension and T2DM is still not established [89]. Finally, it takes a multifactorial approach to address and to treat all major cardiovascular risk factors, not only BP, to achieve lasting cardiovascular protection, as evidenced by the Steno - 2 trial [90].

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