Review Article

LIPITENSION: Interplay between dyslipidemia and hypertension

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ABSTRACT

The burden of cardiovascular disease (CVD) is increasing worldwide. The increase in the burden is a major concern in developing countries like India. It is well-established that hypertension and dyslipidemia are the two major contributing risk factors for CVD. Various epidemiological studies have shown the prevalence of the co-existence of hypertension and dyslipidemia, in the range of 15 to 31%. The co-existence of the two risk factors has more than an additive adverse impact on the vascular endothelium, which results in enhanced atherosclerosis, leading to CVD. This review emphasizes on the 'co-existence and interplay of dyslipidemia and hypertension'. The authors have termed the co-existence as, 'LIPITENSION'. The term LIPITENSION may help clinicians in easy identification and aggressive management of the two conditions together, ultimately preventing future cardiovascular events.

Key words: Cardiovascular risk factors, dyslipidemia, hypertension, lipitension

Introduction

Dyslipidemia and hypertension are established risk factors of prime importance in cardiovascular diseases. They constitute the important components of the metabolic syndrome (MS), as defined by the *National Cholesterol Education Program* (NCEP) *Guidelines* (Adult Treatment Panel III).[1]

In the broader sense, dyslipidemia includes abnormality in LDL-cholesterol, HDL-cholesterol, and triglyceride levels. The term *dyslipidemic hypertension* (DH) was first used in 1988, in the context of familial DH,[2] which was proposed as a genetic syndrome found in approximately 12% of the patients with essential hypertension and 48% of the hypertensive sibships.[3] It has been stated that insulin resistance coexists in up to 50% of the hypertensive individuals.[4] Non-familial forms of DH are more common than the familial ones.

The *Framingham Heart Study* data on the hypertensive population reported that more than 80% had at least one additional cardiovascular disease risk factor and predominantly these risk factors were atherogenic in nature. Studies have consistently indicated that hypertension and hypercholesterolemia frequently coexist, causing what is known as dyslipidemic hypertension (DH).[2,5] The risk of CVD associated with concomitant hypertension and dyslipidemia is more multiplicative than the sum of the individual risk factors.[6,7] This has been recognized in the recent treatment guidelines that emphasize the need to quantify a person's overall CVD risk.[8,9]

In this review, the coexistence of dyslipidemic hypertension has been termed as 'LIPITENSION' for the ease of active identification, diagnosis, and management of these two risk factors together, as global CV risk factors.The diagnosis of hypertension and dyslipidemia are considered as pre JNC 7 and NECP ATP III guidelines, respectively.

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The previous prevalence estimates for the existence of lipitension range from 15 to 31% in the United States.[10,11] The elderly population in the US had 31% existence of lipitension. In another study, the overall prevalence of lipitension, hypertension alone, and hypercholesterolemia alone was 30, 47, and 18%, respectively. The incidence of lipitension was 20% in women versus 16% in men ($p < 0.05$). The incidence was variable in different age groups with only 1.9% in the 20 – 39 year age group versus an increasing trend of 56% in the aged, > 80 years ($p <,0.001$). lipitension prevalence showed racial variation, with the least in Hispanics (9.8%) and highest in African–Americans (22%) ($p < 0.01$). The prevalence increased with addition of risk factors; highest in those with CVD plus DM or metabolic syndrome (69%).[12]

*Chennai Urban Population Study (*CUPS study 5) reported the prevalence of coronary artery disease (CAD) in type 2 DM south Indian patients. The highest prevalence of CAD (21.1%) was found in patients with multiple risk factors and these risk factors, in order, were diabetes, dyslipidemia, and hypertension. However data on the coexistence of dyslipidemia and hypertension together, in these diabetic patients, was not reported.[13]

Chennai Urban–Rural Epidemiology Study (CURES-38) on 2300 diabetic patients reported the prevalence of dyslipidemia, hypertension, and other CV risk factors, however, the co-existence of dyslipidemia and diabetes was not reported.[14]

Prevalence of the metabolic syndrome (METS) using IDF 2005 guidelines, in a semi-urban south Indian was 30%. Nearly 80% of these patients had systolic and 57% had diastolic hypertension, the dyslipidemia trend was high TG (38.8%) and low HDL (59%). However, the data on the co-existence of dyslipidemia and hypertension were not reported.[15]

The Interplay — Lipitension

Dyslipidemia, one of the strong predictors of cardiovascular disease, causes endothelial damage and loss of physiological vasomotor activity.[16-19] The damage may manifest as elevated systemic blood pressure (BP). Cross-sectional studies have suggested a link between abnormal lipids and hypertension.[20-22] Few prospective studies have demonstrated the relationship between plasma lipids and the future development of hypertension.[23,24]

Blood pressure has a continuous and consistent relationship with the risk of cardiovascular events; the higher the BP, the higher the chance of CVD. The presence of each additional risk factor multiplies the risk for hypertension.[25] However, there is limited data demonstrating the effect of elevated blood pressure on lipid levels.

This interplay of lipitension was observed in a large epidemiological study like MRFIT (*Multiple Risk Factor Intervention Trial),* with 356,222 men, followed up for 12 years. The study results emphasized that even mild-to-moderate levels of both hypertension and dyslipidemia had a multiplicative adverse impact on the risk for coronary heart disease (CHD), and the risk was similar or greater due to a severe elevation of either one of the risk factors. Furthermore, the *Framingham Study* results also reflected that moderately elevated blood pressure and cholesterol had a similar 10-year risk of CHD, as those with highly elevated systolic BP or LDL cholesterol alone.[26]

This physiological interplay produces a marked increase in CVD risk.[27,28] Reduction in the prevalence of lipitension can significantly improve the outcomes.

Lipitension and Atherosclerosis

The renin-angiotensin aldosterone system (RAAS) promotes atherogenesis. Angiotensin II, a major villain of the RAAS pathway, promotes atherogenesis through stimulation of the angiotensin type 1 receptor (AT1), which increases lipid uptake in cells, vasoconstriction, and free radical production, to foster both hypertension and atherosclerosis.[29]

Hypertension, a major component of lipitension, damages the endothelium through altered shear stress and oxidative stress, resulting in increased endothelial cell synthesis of collagen and fibronectin, reduced nitric oxide-dependent vascular relaxation, and increased permeability to lipoproteins.[30,31] Hypertension is also associated with an upregulation of lipid oxidation enzymes.[32] LDL, especially oxidized LDL, is a major cause of endothelial dysfunction.[33] Hence, lipitension contributes to atherosclerosis and the resultant vascular risk, through its effects on the level of the endothelium.

Microalbuminuria has been identified in hypertensive patients as a marker of glomerular dysfunction and a predictor of coronary artery disease.[34,35] Microalbuminuria has also been associated with lipid abnormalities, including high levels of low-density lipoprotein cholesterol (LDL-C) and triglycerides, low levels of HDL-C, and elevated levels of lipoprotein (a).[36] This area will merit further attention, because it suggests a possible role for circulating lipoproteins in the small vessel organ damage associated with hypertension. It is also noted that the presence of one condition could enhance the development of the

other; hypertension and dyslipidemia may also interact synergistically to enhance the atherosclerotic process.

Parallel Management of Comorbidity

It is intuitive that simultaneous treatment of two or more risk factors should provide at least additive benefits in preventing atherosclerotic vascular events. The analysis mandates urgent steps to be implemented toward the identification and treatment of undetected hypertension and dyslipidemia, which will reduce cardiovascular complications in the long run. Today, most CVD prevention efforts are focused on treating blood pressure, lowering LDL cholesterol, increasing HDL cholesterol, encouraging tobacco cessation and physical activity, and providing medical nutrition therapy. There is strong evidence to support risk factor reduction in these areas. However, there are still significant improvements needed to achieve both primary and secondary prevention of CVD. Data from the cross-sectional National Health and Nutrition Examination Survey 2001 – 2002, estimated the proportion of individuals not at goal for blood pressure, lipids, and hemoglobin A_{1c} (A1C). Overall, 50.2% were not at goal for A1C, 64.6% did not achieve the LDL cholesterol goals, 52.3% did not hit the HDL cholesterol targets, and 53% failed to attain target blood pressure levels.[37]

Few recent clinical studies have made an effort to document the benefits of multiple risk factor interventions. Two large studies have attempted to modify both hypertension and dyslipidemia simultaneously. The first of these is the *Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial* (ALLHAT).[38] This trial had 40,000 patients on various antihypertensive therapies. Out of these, 10,000 were put on pravastatin 40 mg daily dose. At the end of five years, unfortunately, the LDL-C difference in the group taking pravastatin versus the group taking only hypertensive therapy was only 16.7%, which was clinically insignificant. Furthermore, this study, understandably, did not show a statistically significant difference in death (the primary endpoint), fatal or nonfatal CHD events or strokes.

Results of the second study, the ASCOT-LLA trial suggested that, in addition to good BP control, patients with hypertension and additional CVD risk factors required a more intensive lipid-lowering therapy than previously recommended, to decrease future CV events.

In a study of patients taking an ACE inhibitor (enalapril or lisinopril), the degree of systolic blood pressure lowering was doubled, and the degree of diastolic blood pressure lowering increased by 25% when a statin (either lovastatin or pravastatin) was added to the regimen. The mean cholesterol levels fell by 38% and these results could be attributed to a direct statin effect or to cholesterol lowering.[39] Such differences in blood pressure between patients treated with lipid-lowering therapies and those given a control intervention were not seen in HPS, ASCOT, ALLHAT or several other large clinical trials. Whether blood pressure–lowering drugs have any effect on the circulating lipid levels has been the subject of intense debate for many years.

Effect of Lipid-Lowering Therapy on Blood Pressure

There is some evidence to suggest that treating dyslipidemia has beneficial effects on blood pressure (BP). Studies by Borghi *et al*.,[36,40] have found that patients receiving concomitant antihypertensive and statin therapy experienced a reduction in BP that could not be explained solely by the lipid-lowering effect of the statin or the effect of the antihypertensive medication. These results suggest that the use of statins in combination with antihypertensive drugs may improve BP control in patients, with uncontrolled hypertension and high serum cholesterol levels.

The pathophysiological basis for the apparent beneficial effect of statin therapy on BP may be that statins have positive effects on endothelial or vascular smooth muscle cell functions or both. Hypercholesterolemia can also influence BP by potentiating the effects on the endothelium of the vasoconstrictors endothelin-1 and Angiotensin II.[41-43] Thus, a reduction in nitric oxide production, coupled with a heightened vasoconstrictor response, will tend to increase BP in patients with dyslipidemia.

Clearly, further large-scale trials are needed to examine the BP-lowering effects of the lipid-lowering therapy and to distinguish between the effects that can be directly attributed to reduced serum cholesterol, the pleiotropic effects of lipid-lowering agents or situations in which lipid-lowering medications potentiate the action of concomitant antihypertensive agents.

Effect of Blood Pressure–Lowering Therapy on Lipid Levels

Blood pressure–lowering drugs have a certain impact on the lipid levels. These changes in lipid levels are important in hypertensives, as up to 40% of the newly diagnosed hypertensives have at least one lipid abnormality.[44] Among the anti-hypertensive drugs, beta blockers and thiazide diuretics have shown changes in the lipid parameters. The beta blocker effect on the lipid level depends on the

pharmacological properties. Selective blockers (beta-1) and newer beta blockers have little impact on the total cholesterol levels. The thiazide diuretics' effect on cholesterol parameters becomes evident at higher doses.

However, these two classes of drugs have shown protection in patients with comorbidities such as coronary artery disease, if the BP is well controlled.[45]

Considering the effect on plasma lipid alpha-blockers, the Renin Angiotensin Aldosterone System (RAAS) blocker or the calcium channel blocker may be the preferred initial therapy in patients with underlying hyperlipidemia.[46]

Lipitension and Coronary Artery Disease

Lipitension, as a combination, is an important risk factor for the progression of atherosclerosis in CAD patients. A study by Adnan *et al*., looked at the impact of the optimal control of LDL-C and Systolic BP on the progression of atheroma in the coronary arteries by using the Intravascular Ultrasound (IVUS). The study concluded that patients with very low LDL-C (< 70 mg / dl) and normal SBP (≤ 120 mmHg) had the least progression of percentage atheroma ($P < 0.001$) and more frequent plaque regression ($P < 0.01$). These findings suggest the need for the intensive control of lipitension as a component of global risk factors in patients with CAD.[47]

On the other hand trials like the ß-Blocker Cholesterol-Lowering Asymptomatic Plaque study (BCAP) and ELVA have shown that beta blocker therapy has beneficial effects on reducing early atherosclerosis progression and intima-media thickness (IMT) thickening, in asymptomatic carotid plaque patients, along with BP reduction. This in turn also results in reducing CVD mortality.[48,49]

Discussion

Dyslipidemia and Hypertension are well-established risk factors for CVD, and the coexistence of both these conditions have proved to have adverse outcomes (1) Prevalence of coexistence is variable across the globe. Interaction between these two risk factors occurs at the vascular endothelial level. This results in increased oxidative stress, endothelial dysfunction, and progression of atherosclerosis, and ends up with a major CV event.[29-32] Hypertension individually with a well-known rule of half; we know that half of the total cases are diagnosed, among the diagnosed, half get treated and of these treated only half achieve the goals.[50] On other side, the dyslipidemia picture is more disappointing.[51] Data on the management of these two conditions together is lacking.

When we treat the metabolic syndrome the usual focus remains more on insulin resistance and lipid levels, ignoring blood pressure.[52] Thus, there is a need for a new approach to tackle the coexistence of hypertension and dyslipidemia. The term lipitension may help in identifying patients with hypertension and dyslipidemia.

Studies have demonstrated that the treatment of dyslipidemia, particularly LDL-C lowering, has favorable effects on both coronary and cerebrovascular event rates, over and above the benefits of blood pressure lowering itself.[39,41] Similarly blood pressure control has favorable effects both on the coronary and cerebrovascular event rates. Even as the cholesterol-lowering drugs may favorably affect blood pressure, some blood pressure–lowering drugs, such as RAAS blockers and CCBs have a neutral or some beneficial effect on serum lipid levels.[53]

The management of these disorders, particularly in high-risk patients, requires multiple interventions, including dietary and pharmacological. The need for a different treatment approach in CV medicine has emerged, due to combined problems of an aging population and poor adherence to complex drug regimens. This has led to a reconsideration of combination pills. This approach can be related to the treatment of hypertension and dyslipidemia together.

There is a need to increase the awareness, both in the medical and patient communities, for early detection and treatment of these two conditions. Few proposed approaches are always screening for another risk factor, when one risk factor is found, as also family screening of patients with these disorders. These approaches may have a rich yield of additional patients requiring treatment.

Our hope is that the information discussed here and the concept of the term lipitension will expand the knowledge in this area, and help to proactively identify individuals who may benefit from further investigations or treatments, and refer complex cases for specialized care.

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