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Review Article

A review on biomarkers of hypertension

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ABSTRACT

Hypertension is the most prevalent chronic medical condition seen in primary care. It is a silent disease because of its asymptomatic nature and is usually diagnosed at advanced stage. It effects various organs like heart, kidney, etc. It is responsible for 10.8% deaths in India. Over the years many biomarkers are identified to understand the pathophysiology of hypertension. Biomarkers have unique role in prognosis as the level rises before the onset of overt hypertension. Clinically it is beneficial as it helps in identifying the high risk patients for better treatment and prognosis. In this review we have highlighted the importance of various biomarkers of hypertension in early diagnosis, before the onset of overt hypertension, which is associated with long-term end-organ diseases.

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1. Introduction

Hypertension (HTN) is a major global health burden. It is the most prevalent chronic medical condition seen in primary care.[1] It is responsible for 10.8% of all deaths in India.[2] It effects various organ like heart, kidney, brain, eyes, etc. HTN is directly responsible for 57% of all stroke deaths and 24% of all coronary heart disease deaths in India. It is a controllable disease. A 2mmHg population-wide decrease in BP can prevent 151,000 stroke and 153,000 coronary heart disease deaths in India.[3]

HTN is divided into 2 groups: primary (or essential) and secondary hypertension. Essential hypertension has no clear etiology and accounts for 95% of cases. Secondary hypertension accounts for the remaining 5% of cases.[4] It has many clinical patterns and/or etiopathological forms which includes: essential HTN, secondary HTN, white-coat HTN, isolated systolic HTN, masked HTN, pulmonary HTN, pregnancy related HTN.[1] Clinical or office measurement of blood pressure (BP) is the classical diagnosis of HTN. It is defined as persistent elevation in office systolic BP ≥ 140 mmHg and or diastolic BP ≥ 90 mmHg. This is equivalent to a 24 hr ambulatory BP monitoring average of ≥ 130/80 mmHg or a home BP monitoring average ≥ 135/85 mmHg.[5]

Regardless of the device used to assess BP, the conventional practice only provides us with values and patterns. A significant proportion of hypertensive patients are diagnosed at an advanced stage of the disease, because of the "silent" nature of the disease in most cases.[6] According to the latest European guidelines hypertension is diagnosed when BP is >140/90 mm Hg, whereas according to the American guidelines hypertension is diagnosed when BP >130/80 mm Hg. It is proven that in Indian population BP levels between 130 to 139/80 to 89 mm Hg can lead to higher risk of CVD, stroke, and premature mortality.[7] Thus the newer (lower) thresholds for goal BP according to American guidelines might seem unattainable but if the country adopts it can be protective to Indian population.

Gold standard for blood pressure measurement is using mercury sphygmomanometer.[8] But poor standardized

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techniques of BP measurement, poor calibration of device and diagnosis based on single measurement are few challenges in India. Therefore, laboratory tests could predict not only the risk of development of HTN, but also in treatment of individual patients by helping to choose the right drug for the right patient, and also can help in monitoring the control of HTN throughout the treatment period, help in risk stratification of the patients by prognosis, and predict and prevent hypertensive end organ damage.[9]

Multiple biomarkers for HTN have been identified over the years and may shed light on the underlying processes involved in the development and progression of HTN. Here, we discuss the important biomarkers of HTN to help better evaluate their utility and to better understand the pathophysiology, diagnosis, progression, and therapeutic efficacy of EH.

2. Materials and Methods

A Pubmed and google scholar search was performed, using "hypertension", "biomarkers" and "markers" as search parameters, and also their relevant references were reviewed. No limitation was placed on years included in the study. A total of 150 articles were read and analysed with particular focus on usefulness of the biomarker. Studies were excluded if they were not relevant and practically feasible. 64 remaining articles were found relevant and reviewed further. The included studies described number of biomarkers of which five biomarkers i.e., C-reactive protein, cytokines, uric acid, urine albumin excretion and nitric oxide were considered for this study because of its feasibility.

3. Discussion

Genetic and environmental factors contribute to development of HTN. Also, a complex interactions of various hormones, local vascular factors, and neural mechanisms have a role to play.[10] The cause of secondary HTN includes primary hyperaldosteronism, Cushing's syndrome, etc. They have specific markers for diagnosis such as plasma metanephrine measurements, plasma aldosterone level, etc.[1] On the other hand, though essential hypertension accounts for 95% of all cases of HTN, its etiology is not clear. Different patients have different etiology for high blood pressure.[11] Endothelial dysfunctions, oxidative stress, and inflammation play a major role in its development Figure 1.[12–16]

In this section we discuss various biomarkers of hypertension. Of the many available markers C-reactive protein, cytokines, uric acid, urine albumin excretion and nitric oxide gives us better understanding of the progression of HTN.

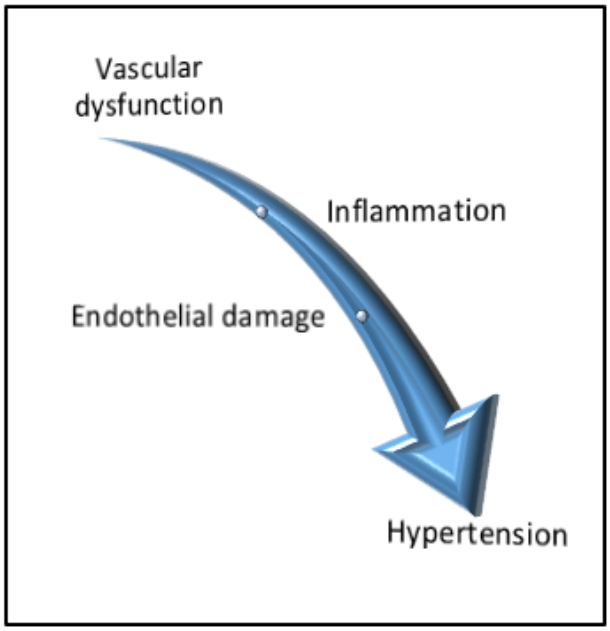

Fig. 1: Schematic diagram showing relationship between inflammation & HTN

3.1. C-reactive protein (CRP)

CRP is an inflammatory marker and is increased in several conditions like diabetes.[17] Normally minimal amounts of CRP is present in plasma and the level increases to 100 fold when there is any injury, infection, or inflammation. CRP is the first acute phase protein to be described.[18,19] It is mainly synthesized by liver in response to interleukin-6 and interleukin-1ß. Its relation to essential HTN is extensively studied.[20–22] Sesso et al. in their study demonstrated that with increased levels of CRP, risk of developing HTN increases.[15] Similar findings were shown in another study by Wang et al.[23]

CRP increases the blood pressure by various mechanisms:

1. It decreases the production of nitric oxide by endothelial cells,[24,25] andthus indirectly inhibits vasodilatation.
2. It increases leukocyte adhesion, platelet activation, oxidation, and thrombosis.[24,25]
3. It also up regulates the angiotensin type-1 receptor, thus mediating the angiotensin-II mediated increase in blood pressure.[11]

CRP is very stable and have half-life of 19 hours with very little variation in values between fresh and frozen forms.[26] Also it has a longer plasma half-life making it more reliable indicator of chronic inflammation.[27] The only drawback is that it is not detectable at a very low level (i.e., <3 mg/l). Instead hsCRP (detectable at lower than

3 mg/l) can be used to detect pro-inflammatory state at the earliest. Sinha et al. in their study found increased hsCRP level positively related with increase in diastolic blood pressure in prehypertensives.[28] Ki Chul Sung et al. in another study found hsCRP to be an independent risk factor for development of hypertension in Korean population.[29]

3.2. Cytokines

Cytokines are signalling proteins, which regulate a wide range of biological functions including innate and acquired immunity, haematopoiesis, inflammation and repair, and proliferation through mostly extracellular signalling. Pro-inflammatory cytokines include IL-1β, IL-6, and TNFα.[30]IL-6 is a central mediator of the acute-phase response and a primary determinant of hepatic production of CRP, plus, IL-6 modulates the production of tumour necrosis factor and regulates the immune response.[31] Circulating IL-6 also stimulates the hypothalamic– pituitary–adrenal axis, which, when activated, is also associated with hypertension.[32]

There are studies that have demonstrated increased cytokine (IL-1β, IL-6, TNF-α) levels in hypertensive patients compared to normotensive patients.[30–33] The link between cytokines and hypertension maybe:

1. Various hemodynamic changes due to hypertension leads to endothelial dysfunction, and increase in levels of inflammatory markers such as interleukin-6 (IL-6), intracellular adhesion molecule 1 (ICAM1), P-selectin, and tumor necrosis factor-α (TNF-α).[12,13]
2. The renin-angiotensin system and sympathetic nervous system, play an important role in regulating blood pressure. They stimulate the release of proinflammatory cytokines (IL-6, TNF- α) and serve its source.[34,35] These cytokines in turn induce structural as well as functional alterations in endothelial cells.[36,37]
3. CRP by stimulating monocytes also release pro-inflammatory cytokines such as IL-6, IL-1β and TNF-α which further promotes inflammation and effects endothelial function.[37]
4. There is increasing evidence that excess adiposity, which is a major risk factor for hypertension, is characterized by broad inflammatory response.[1,38] This induces adipose tissue to release various cytokines and adipokines proteins[39] and to create a chronic state of inflammation that may lead to hypertension.

3.3. Uric acid (UA)

UA is an end product of purine metabolism.[40] It is 5% plasma protein bound, is freely filtered at the glomerulus, is 99% reabsorbed in the proximal tubule, secreted by the distal tubule, and subjected to considerable post secretory reabsorption. Fractional secretion of uric acid is about 7% to 10%.[41] Serum UA serves as a useful marker of inflammation and oxidative stress in HTN.[42] An elevated UA level is observed in nearly 90% of adolescents with essential hypertension of recent onset.[43]

A lot of studies have shown a significant association between uric acid levels, HTN and its cardiovascular complications in their study.[44,45] A study by Feig et al. found that by administering drugs such as allopurinol (used for treatment of hyperuricemia) in obese adolescents with pre-hypertension, resulted in marked BP control and reduction in systemic vascular resistance.[43] Scheepers et al. in their study have also focussed on importance of uric acid and purine catabolism, and their potential association with essential hypertension.[46] Association between hypertension and increased UA was first seen in 1957.[47]

UA is thought to play a role in HTN via mechanisms like inflammation, vascular smooth muscle cell proliferation in renal microcirculation, endothelial dysfunction, down regulation of nitric oxide (NO) production and activation of the rennin – angiotensin – aldosterone system.[48,49]This hypertension type is salt-resistant in that it occurs even in the presence of a low-salt diet, and it responds to lowering of UA.[49] The role played by UA in pathogenesis of early onset HTN decreases with increase in age. With increase in age, stiffening of the aorta, activation of RA- system and renal vasoconstriction have a role to play.[50] Various studies have found a higher mean UA concentration in pre-hypertensives who were averagely younger.[45,49,51]

3.4. Urine albumin excretion (UAE)

The detection and quantification of albumin in urine is commonly used for screening of diabetic and hypertensive nephropathy, as well as preeclampsia. It's an early marker of renal damage.[52] UAE predicts the development of hypertension, independent of BP and other widely known risk factors for development of hypertension.[53] There are studies which found risk of developing HTN was highest with increase in UAE.[54] This can be explained by following mechanisms:

1. UAE reflects damage to the renal microvasculature which causes generalized endothelial dysfunction, thus causing hypertension.[55,56]
2. Reduction in number of nephrons also provides a link between higher albumin excretion and the development of hypertension.[57]
3. GFR is mildly reduced when there is reduction in glomerular filtration surface area thereby limiting excretion of sodium and causing hypertension.[58]

3.5. Nitric oxide

Nitric oxide(NO) is generated from its precursor L-arginine by nitric oxide synthase (NOS). Inflammation has been shown to downregulate NOS activity.[59] Research data

suggests impaired NO activity is associated with HTN. Increased ROS and an altered balance between NO and ROS lead to impaired bioavailability of NO, resulting in decreased endothelium-dependent vasodilation, which, in turn, causes or exacerbates hypertension.[60,61] Impaired NO dependent vasodilation due to an imbalance between vasoconstrictors and vasodilators precedes hypertension.[62]

4. Conclusion

HTN is a chronic disease which causes severe complications like CAD, heart failure, stroke, etc. Inflammation can lead to the development of hypertension and oxidative stress and endothelial dysfunction which is associated with inflammation contributes to hypertension, by exacerbating the inflammatory response. Thus, biomarkers like C-reactive protein, cytokines, uric acid, urine albumin excretion and nitric oxide gives us better understanding of the progression of HTN.

5. Source of Funding

None.

6. Conflict of Interest

None.

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