Cardiovascular diseases: Are we overlooking some cardiovascular disease risk factors/ markers?

Abstract

Despite of credible advances in the field of cardiovascular health promotion contributed by scientific research over the past several decades, cardiovascular disease (CVD) remains to be the leading cause of premature deaths across the globe. Low and middle income countries are more vulnerable to deaths due to CVD. Significantly elevated concentrations of hs-CRP, lipoprotein (a), ischemia modified albumin (iscMA), glycated hemoglobin (Gly-Hb), fibrinogens and decreased HDL-C associated arylesterase activity are also associated with CVD risk. Higher levels of oxidants dominating the antioxidants can be additive risk of CVD. Current studies have suggested additional risk factors namely coronary artery calcium scores, homocysteine levels, periodontal disease, ankle brachial index, B type natriuretic peptide (BNP), N-terminal pro BNP (NT-pro BNP) and carotid intima-media thickness. In addition, albuminuria, inflammatory markers (IL-6), (IL-18), endothelial dysfunction markers (PTX3),(VCAM) are also indicative of CVD which are associated with chronic kidney disease (CKD). Adiponectin levels also influence insulin resistance, dyslipidemia, abdominal obesity and coronary artery disease.

Key words: cardiovascular risk factors, ischemia modified albumin, paraoxonase, oxidants, antioxidants.
Despite of credible advances in the field of cardiovascular health promotion contributed by scientific research over the past several decades, cardiovascular disease (CVD) remains to be the leading cause of premature deaths across the globe [1]. Research and epidemiological studies have concluded that 17.3 million people died from CVD in 2008. Of the entire death takes place due to CVD, 80% of the deaths are from low-middle income countries. It has been projected that by the year 2030, >23.3 million people will die annually from CVDs [2].

Low and middle income countries are more vulnerable to deaths due to CVD, since they are more exposed to risk factors such as tobacco, less access to health care facilities and lack of awareness.

Until, the late twenties, we were aware of the risks factors, such as smoking, hypertension, dyslipidemia, diabetes, obesity, sedentary life style and dietary factors were responsible for the premature deaths due to CVD [2]. To be more appropriate, the risk factors are classified as modifiable and non-modifiable ones. The non-modifiable risks are, sex, age, and familial history and among the modifiable ones are hypertension, tobacco usage, elevated blood glucose, lack of physical activity, unhealthy diet, dyslipidemia, overweight and obesity.

When the prevalence of cardiovascular disease were not explained completely on the basis of modifiable and non-modifiable ones, the search of additional markers and risks were enlisted which might answer and unveil the relationship and could be of great help in risk stratification and improving treatment directed towards specific group or population.

Our research revealed the fact that even in the absence of dyslipidemia; there are chances of getting acute myocardial infarction (AMI). We focused our research mainly on those patients who suffered from AMI but were having absolutely normal lipid profiles. Our study analyzed the additional risk factors which could move the patients to elevated or higher risks in spite being normolipidemic.

Our analytical parameters included the evaluation of additional markers which could answer to the query of being devoid of dyslipidemia, why patients have suffered from AMI. It was found that there are additional risks which are missed out or not evaluated in normal routine follow up in patients. Our study revealed that the patients had significantly elevated concentrations of analytical parameters like hsCRP, Lipoprotein (a), Ischemia modified albumin, glycated hemoglobin, caeruloplasmin, fibrinogens, and decreased HDL-C associated arylesterase activity [1, 3, 4, 5].

Additionally it was found that there is interplay of oxidants and anti-oxidants and balance between these two components were disrupted leading to higher levels of conjugated dienes, malondialdehyde and lower levels of antioxidants namely uric acid, bilirubin, superoxide dismutase, glutathione peroxidase and catalase [5].

Current studies have suggested additional risk factors in addition to our findings, namely coronary artery calcium scores, homocysteine levels [6], periodontal disease, ankle brachial index, B type natriuretic peptide (BNP), N-terminal pro BNP (NT-pro BNP) and carotid intima-media thickness. In addition, albuminuria, inflammatory markers (IL-6), (IL-18), endothelial dysfunction markers (PTX3),(VCAM) are also indicative of CVD which are associated with chronic kidney disease (CKD) [7].

Research studies have also linked adiponectin levels with insulin resistance, dyslipidemia, abdominal
obesity and coronary artery disease. It is noted that adiponectin regulates the body energy metabolism, by stimulating the fatty acid oxidation, lowering TG levels, and improves glucose metabolism through increase in insulin sensitivity [8].

In a tertiary health care setup, apart from analyzing lipid parameters, one should look for additional risk factor analysis which could be feasible and cost effective for patients so that the risk stratification can be evaluated well in advance and proper advice can be conveyed to the patients.

In order to overcome the risk of cardiovascular disease, we need to change the life style which is an important area to look upon. At least 30-35 minutes of brisk walking 3-4 time a week, >4 servings/day of fruits and vegetables, depending on natural nutrients instead of supplements could reduce the risk of future CVD. As indicated, homocysteine levels are related to higher risk of CVD and stroke. We must incorporate more of folic acid and vitamin B₁₂ and B₆ which can breakdown or decrease homocysteine levels in circulation. Green leafy vegetables and grain products must be included in our diet. It is recommended to reduce the future risks of CVD by modifying the modifiable risks associated with it. If we improve and work on the modifiable risks, than the CVD associated deaths can be delayed, thus reducing the global burden of deaths due to CVD. In order to prevent the heavy cost incurred in Intensive coronary care unit and the agony of death in family members, we can take precautionary measures and improve our quality of life style.

References

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