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An association of serum vitamin D, IL-4 level and *V*DR gene polymorphism in CAD with and without T2DM

Mamta P Sumi
Maulana Azad Medical College, India

Introduction: Coronary Artery Disease (CAD) is a leading cause of death in developed countries and is rapidly assuming epidemic proportions in developing countries as well. It has been shown that lower vitamin D levels appear to predict an increased risk of CAD mortality in patients with Type 2 Diabetes Mellitus (T2DM). Coronary atherogenesis leading to CAD is an immunological phenomenon caused by foam cells i.e. transformed macrophages at the lesion site. Apart from the traditional role of vitamin D in calcium homeostasis lot of recent experimental evidences are available on role of vitamin D levels, *VDR* gene polymorphism, vitamin D binding protein gene polymorphism in immune reaction as immunomodulators and now-a-days are being considered as risk factors in generating coronary atherogenesis leading to CAD particularly in association with T2DM. Recent studies also provide that IL-4 exerts proinflammatory effects on vascular endothelium and may play a critical role in developing coronary atherosclerosis.

Aims: So we set our aims for this study to investigate the association of vitamin D, *VDR* gene polymorphism and serum IL-4 levels in CAD with or without T2DM.

Materials & Methods: The study involved two groups of patients suffering from CAD with T2DM (n=40) and CAD without T2DM (n=40) attended emergency or coronary care unit of Lok Nayak Hospital, New Delhi. A total of 6ml of blood sample was collected for estimation of serum vitamin D and IL-4 levels by chemiluminescence immunoassay method and VDR gene polymorphism (exon ll, rs2228570) by PCR-RFLP using Fok1 restriction enzyme. Other relevant routine blood biochemistry tests were done by Beckman coulter fully automated analyzer using commercially available kits.

Results & Discussions: Serum vitamin D levels decreased in both groups of patients, more significantly decreased in the presence of T2DM in CAD patients. Serum IL-4 levels were significantly higher in CAD with T2DM group as compared to CAD without T2DM group. No association was found between VDR gene polymorphism (Fok1) and risk of CAD in T2DM and non T2DM individuals. No significant correlation was found between vitamin D and IL-4 levels in the patients of both groups. No significant association was observed between low 25-hydroxy vitamin D levels with VDR genotypes (Fok1) in both groups of patients.

Conclusions: The association between VDR Fok1 polymorphism, vitamin D and inflammatory markers needs to be further explored in diabetic CAD patients. A bigger study involving a much larger number of patients would help to generalize the results of this study.

mamtasumimamc@gmail.com