



Vascular Medicine

ALTERED CALCIFICATION MEDIATORS OF BONE MORPHOGENETIC PROTEIN-2 AND FETUIN-A PROTEIN EXPRESSION AND MEDIAL ARTERY CALCIFICATION IN PATIENTS WITH PERIPHERAL VASCULAR DISEASE, WITH OR WITHOUT DIABETES MELLITUS AND CHRONIC KIDNEY DISEASE: IMPLICATIONS IN LESION PROGRESSION

Moderated Poster Contributions

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Background: Medial artery calcification (MAC) is increased in diabetic mellitus (DM) patients with peripheral vascular disease (PVD), and Chronic Kidney Disease (CKD). These patients may have altered homeostasis of calcification mediators. Bone Morphogenetic Protein-2 (BMP-2), a calcification inducer, and Fetuin-A, a calcification inhibitor were quantified in patients with symptomatic PVD with/without DM and CKD, and correlated with the degree of MAC.

Methods: Fifty symptomatic patients with PVD undergoing directional atherectomy were evaluated. Plaque specimens were grouped by no DM/no CKD (n=14), DM alone (n=12), CKD alone (n= 10), and DM+CKD (n=14). BMP-2 and Fetuin-A protein density were quantified by immunohistochemistry. MAC was quantified by Alizarin Red stain.

Results: The BMP-2 protein density was progressively increased, and Fetuin-A protein density was progressively decreased in DM-CKD group when compared to other groups (p=0.0001 for both comparisons). Incremental increase in medial artery calcification was observed in DM+CKD group. (p<0.02) (See Figure and Table).

Conclusions: Altered calcification mediators of bone morphogenetic protein-2 and Fetuin-A protein expression are associated with progressive increments of medial artery calcification in DM patients with PVD and CKD. Careful screening, early detection, and aggressive therapy may reduce progression of MAC and PVD in DM patients with CKD.

