Cystatin C Levels & Peripheral Arterial Disease in Type 2 Diabetes Mellitus Regarding Nephropathy

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PAD is an atherosclerotic blockage found in the lower extremities, it is a spectrum of macrovascular complications with higher in diabetic patients than those without diabetes [1]. The higher prevalence of PAD in diabetic patients in general is due to the nature of the disease itself, but other factors such as the longer average life span, and a longer disease duration should not be underestimated [2]. The serum cystatin C or cystatin C-based eGFR is suggested to be associated with PAD in the general population, moreover, subjects with the highest quintile of cystatin C (>1.27 mg/L) at baseline were 2.5 times more likely to have a new onset of PAD than those with the lowest (≤0.9 mg/L), independent of traditional risk factors, where as serum Cr and Cr-based eGFR were not associated with PAD. However, there are few studies that have investigated the association between cystatin C and PAD in patients with type 2 diabetes [3].

A cross-sectional study by Ji Hye Huha et al. [4] Published in Diabetes Research and Clinical Practice entitled "Serum cystatin C levels are associated with asymptomatic peripheral arterial disease in type 2 diabetes mellitus patients without overt nephropathy" with great interest demonstrated that type 2 diabetic patients with peripheral artery diseases PAD have have a lower eGFR and higher 24 H urinary Albumin and cystatin C compared to type 2 diabetic patients without Peripheral Arterial Diseases. PAD is associated with cystatin C in type 2 diabetes mellitus patients with normal renal function or mild renal impairment after adjustment for age, gender, waist circumference, current smoking, hypertension, HDL cholesterol, hsCRP, albuminuria, and Cr-based eGFR and a 1-SD increase in cystatin C was associated with about 2 times increased risk for the presence of PAD independent of age, gender, current smoking, and hypertension. Moreover, the AUC with cystatin C was higher than the AUC with other two markers for predicting the presence of PAD. Thus, serum cystatin C may be a more reliable marker than albuminuria or Cr-based eGFR for the early detection of PAD in type 2 diabetes mellitus patients without overt nephropathy.

This study by Ji Hye Huha et al. is a clinically important work proved the independent association of cystatin C with PDA in Type 2 Diabetes Mellitus without overt nephropathy.

First; the causal link between Cystatin C and PDA could not be proved in cross-sectional design and thus a clinically large sample sized longitudinal study is recommended.

Second, although Ji Hye Huha et al. excluded those with overt nephropathy, the link between cystatin C and PAD could be attributed to the involvement of a variable range of renal impairment from normal to subclinical KD.

Third; the diagnosis of PAD by an Arterial Brachial Index (ABI) was unreliable and the small sample size mainly those with PAD thus the patients could not be further divided to several subgroups based on low ABI values.

Finally; hypertension, age, glycosylated albumin, and duration were not evaluated as risk factors for PAD by binary logistic regression analysis.

References

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