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LETTER TO EDITOR

Serum Paraoxonase (Arylesterase) activity in chronic renal failure

Dear Editor,

Paraoxonase (PON) is a calcium dependent polymorphic enzyme. In serum, PON circulates as high-density lipoprotein cholesterol (HDL-C) component, tightly bound to apolipoprotein (apo) A1 with its hydrophobic N-terminal domain. Biochemical studies have shown that antioxidant activity of HDL-C resides in its enzymes, particularly PON as a major contributor to its antiatherogenic role [1]. In chronic renal failure (CRF), there is an increase susceptibility to oxidation of LDL-C and a significant reduction in serum PON activity, explaining the cause for a high and premature incidence of atherosclerosis in these individuals [2]. Arylesterase activity is a measure of PON activity, in which p-nitro phenyl acetate is used as a substrate. The aim of this study was to measure the serum PON - arylesterase activity and phenotypic distribution in patients with CRF (both on conservative management and on hemodialysis) and healthy individuals.

The study was carried out on 44 CRF patients on conservative management, 33 CRF patients on hemodialysis and age-matched 50 healthy subjects in PSG Hospitals, Coimbatore, India. Serum PON activity was measured spectrophotometrically using p-nitro phenyl acetate as a substrate [3]. Levels of serum HDL-C and apo A were determined by Immunoturbidimetry in Roche Integra 400 using dedicated kits. The Pearson stimulation of PON was calculated as (salt stimulated pon activity-basal pon activity/basal pon activity) x 100%.

Individuals were classified for PON phenotype using the antimode of PON as proposed by Eckerson et al. [4]. The results were expressed as mean ± SD. and a p value of < 0.05 was considered statistically significant. Statistical analysis was performed using the Statistical Package for Social Sciences (SPSS), Version 10. Student's t-test was used to compare mean values. Pearson's Correlation coefficient analysis was used to find the degree of correlation between parameters. PON, HDL standardized PON activity, serum HDL-C and apo A were significantly lower ($p<0.05$) in CRF (on conservative management and on hemodialysis) compared to controls (Table-1). CRF patients either on conservative management or hemodialysis, 43 and 30 subjects respectively had AA Phenotype (low PON activity) in comparison to only 17 among the healthy subjects. Studies have reported a decrease in serum PON activity in uremia. This is due to the retention of uremic toxins, "middle molecules" like advanced glycated end products (AGE) as well as high concentrations of HOCl that severely oxidize serum proteins and tryptophan residues in the active site of PON, decreasing PON arylesterase activity in serum. In CRF patients, overproduction of HOCl that leads to high concentrations of severely oxidized proteins and increased oxidants in plasma could contribute to low serum PON arylesterase activity [2]. It has also been reported that serum PON activity decreases with increasing severity of renal failure and in patients on hemodialysis [5]. A reduction in serum PON activity as seen in our study may increase the oxidative stress leading to an increase susceptibility of LDL-C as well as all other serum lipids including HDL-C to oxidation. HDL-C oxidation may impair its ability to induce cellular cholesterol efflux from macrophages while oxidized-LDL- C is atherogenic and cytotoxic leading to stimulation of thrombotic and inflammatory events. Hence in CRF, the quality of HDL-C maintained by its protective enzymes e.g. PON is altered rather than the absolute levels of HDL-C. It was found that the HDL–C standardized enzyme activities (HDL-C/PON Arylesterase activity) were lower in CRF, $p<0.05$ (both on conservative management and hemodialysis) (Table1). Similarly a significant positive correlation was found between serum basal PON activity, salt stimulated PON activity, HDL-C and apo A levels in CRF ($p<0.01$). These data suggest that PON activity changes are not entirely dependent on HDL-C concentration in CRF patients. In conclusion the serum HDL-C level is not a good reflection of the functional property of HDL-C. Even with a normal level of HDL-C, a measure of antioxidant capacity of HDL-C will yield additional information, which improves the predictive accuracy of atherosclerotic coronary artery disease in chronic renal failure. It may also provide new strategies for the prevention and treatment of accelerated atherosclerosis in chronic renal failure.

Table 1 Mean basal PON, salt stimulated PON activity, HDL-C and apo A in healthy individuals and CRF (on conservative management and hemodialysis)

Parameter	Mean ± SD	p*
Basal arylesterase activity (U/L)		
Control		
On conservative management	114.35±35.68	
On hemodialysis	89.27± 20.64	0.003
	39.05± 10.35	0.001
Salt stimulated arylesterase activity (U/L)		
Control	137.5± 45.29	
On conservative management	103.4± 26.34	0.006
On hemodialysis	122.3±36.10	0.033
HDL cholesterol		
Control	52.97±35.87	
On conservative management	34.60± 12.83	0.005
On hemodialysis	29.89±11.20	0.001
Apo A		
Control	151.30±51.89	
On conservative management	110.58±29.83	0.05
On hemodialysis	83.66±32.76	0.03
Basal arylesterase/HDL-C ratio		
Control		
On conservative management	2.54±1.00	0.001
On hemodialysis	3.20±2.46	0.002
	4.59±3.27	
Salt stimulated arylesterase/HDL -C ratio		
Control	3.05± 1.28	
On conservative management	3.73± 3.02	0.047
On hemodialysis	5.19± 3.49	0.002

*Student's t-test

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Competing Interests

The authors declare that they have no competing interests.

Sincerely,

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