Assessment of Pentraxin 3 (PTX3) and Sclerostin (SOST) Levels in Serum of Patients with Chronic Kidney Disease

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Abstract

In the present study, 70 of patients were 35 males and 35 females were suffering from chronic kidney disease, and 20 of control group. The samples were collected from Specialized Center for Diseases and Kidney Transplant in AL-Sadder Medical City/Najaf Governorate/Iraq, during the period from July till August, 2019. The ages of control and patients ranged 30 to 69 y old. The patients group was divided into subgroup according to the age and gender, Patients without a complete medical record were excluded and those with other diseases were excluded.

The present study revealed a significant enhance ($p<0.05$) inserum PTX3 and SOST levels in CKD compare with control group, while a significant decline ($P<0.05$) in serum HDL level of CKD in comparison with control group. The result indicated no significant differences ($p>0.05$) in serum of PTX3 and SOST levels between female and males groups of CLD patients. Also, The results showed a significant increase ($p>0.05$) in serum PTX3 and SOST levels there was a significant increase($p<0.05$)among different groups ages. There is a positive association between PTX3 and SOST concentrations of CKD patients

Conclusion: The current study conducted that PTX3 and SOST levels were good markers for diagnosis and detection of chronic kidney disease in both genders the males and females.

Keywords: *Chronic Kidney disease (CKD), Pentraxin 3 (PTX3) and Sclerostin (SOST).*

Introduction

The chronic kidney disease (CKD) is refer to along term loss function of kidney, Its identified via the presence of an abnormality of kidney function or structure or both for at least three months [1,2]. The inflammation is might to assume an important role in both atherogenesis and advancement of chronic kidney disease[3, 4].

Pentraxins (PTXs) a superfamily of evolutionarily conserved proteins characterize via acyclic multimeric structure[5]. The Pentraxin-3 (PTX3) is protein encoded by PTX3 gene and produced via avariety of cells and tissues, and especially via innate-immunity cells in responses to endothelial cells and proinflammatory signals [6,7]. as a consequence of this extra- hepatic synthesis, and also in contrast to C-reactive protein(CRP), the PTX3 levels are thought to be atrue independent marker of disease activity produce at site of the inflammations[8].

Sclerostin (SOST) is a190 amino acid residue glycoprotein encodevia SOST gene, with a molecular mass of 24 kilo dalton and asequence homology analogous to that of other bone morphogenetic protein antagonist[9, 0]. Sclerostin (SOST) is one of biomarkersas the link between vascular and bone disease[11.] Sclerostin is inhibitors of Wingless Int (Wnt) signaling produced via osteocytes and potentiall play important role in obstruct bone formation[12]. Sclerostin may perhaps affect bone metabolism through chronic kidney disease and the endstage renal undergoing maintenances dialysis[13,14,15.]

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Method and Materials

Healthy and Patients groups: In the present study, 70 of patients were 35 males and 35 females were

suffering from chronic kidney disease, and 20 of control group. The samples were collected from Specialized Center for Diseases and Kidney Transplant in AL-Sadder Medical City/Najaf Governorate/Iraq, during the period from July till August, 2019. The ages of control and patients ranged 30 to 69 y old. The patients group was divided into subgroup according to the age and gender, Patients without a complete medical record were excluded and those with other diseases were excluded.

Blood samples collection: Five ml of venous blood was acquired by antecubital venipuncture utilizing needle drained from CKD and control subjects between 8:30- 10 AM following 12 hour fasting. The blood was permitted to clot in plain test tube at room temperature. The serum was suctioned after centrifugation at 3000rpm for 10min, divided into aliquots in epindroff tubes and stored at -20°c.

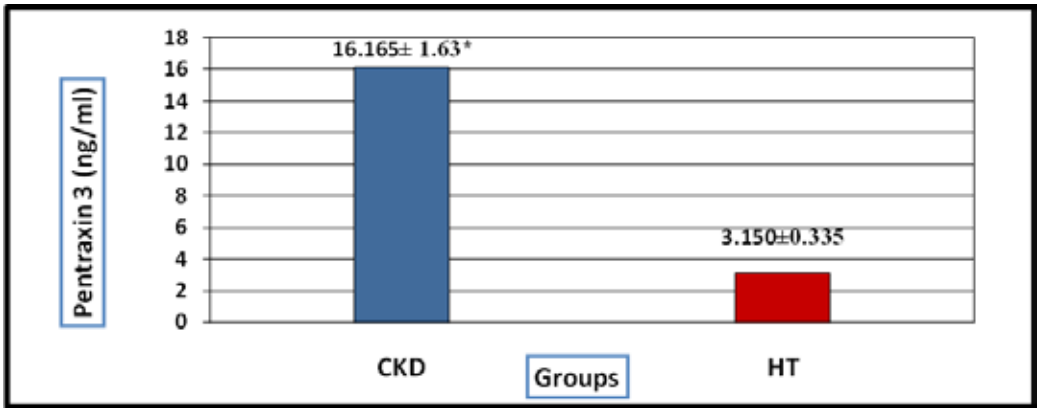
Determination of serum Pentraxin3 (PTX3) level: Human Pentraxin3 Elisa kit (PTX3) was supplied via Bioassay technology laboratoryCo., Ltd. A Catalog No: E1938Hu .

Determination of serum Sclerostin (SOST) level: Human sclerostin Elisa kit (SOST) was supplied viaBioassay technology laboratoryCo., Ltd. A Catalog No: E3068Hu

Statistical analysis: The data of present study were articulated as (Mean±Standard Error), the statistical analysis (Descriptive statistics, Correlation coefficients, P-value) were calculated by using Graphpad prism. The comparison between two groups were analyzed by t-test and the comparison among subdivided groups were analyzed by one-way ANOVA. when P-value < 0.05 was statistically a significant**.**

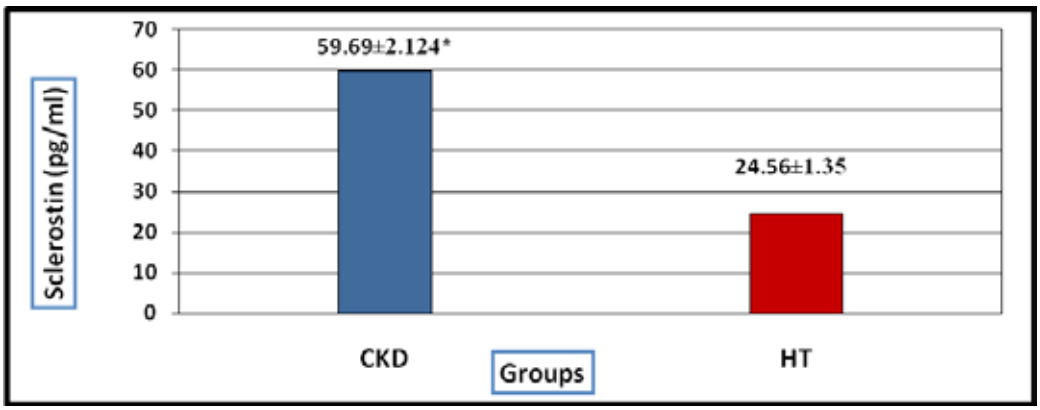
Results

Evaluation of serum level Pentraxin 3 and sclerostin: The result in figure (1) and (2) exhibit a significant increased ($p<0.05$) in serum levels of pentraxin 3 and sclerostin in CKD group compared with in HT group.



(*) :Statistically significant differences ($p<0.05$).

Fig (1): Serum level of Pentraxin 3 between chronic kidney disease Patients and control groups.



(*) :Statistically significant differences ($p<0.05$).

Fig (2): Serum level of Sclerostin between chronic kidney disease patients and control groups.

Evaluation of serum levels pentraxin 3 and sclerostin in CKD patients between males and females groups: In Table (1), The results of present study revealed there is no significant differences($p>0.05$) in of pentraxin 3 and sclerostin levels of CKD between female and males groups.

Evaluation of serum levels pentraxin 3 and sclerostin in CKD patientsat different ages groups: Table (2), The result showed a significant increased ($p<0.05$) in pentraxin 3 and sclerostin levels of CKD at different ages groups .

Table (1): Comparison of serum levels pentraxin 3 and sclerostin between males and females groups of CKD patients.

Groups Markers	Mean±S.E.	
	Males	Females
Pentraxin 3 (ng/ml)	15.163±0.086	17.32±0.148 ns
Sclerostin (pg/ml)	58.235±2.316	61.145±2.913 ns

(NS): Statistically mean no significant differences ($p>0.05$).

Table (2): Evaluation of serum levels pentraxin 3 and sclerostin CKD patients at different ages groups .

Groups Markers	Mean±S.E.			
	30-39y	40-49y	50-59y	60-69y
Pentraxin 3 (ng/ml)	7.583±0.516 a	13.023±0.871 b	18.563±1.245 c	25.493±1.89 d
Sclerostin (pg/ml)	40.813±1.261 a	53.023± 1.87 b	64.613±2.041 c	80.313±2.54 d

The different letters mean significant differences ($P<0.05$).

Correlation between serum pentraxin 3 and sclerostin levels: Results of the correlation coefficient between pentraxin 3 and sclerostin concentrations at the chronic kidney disease patients revealed in figure (3):

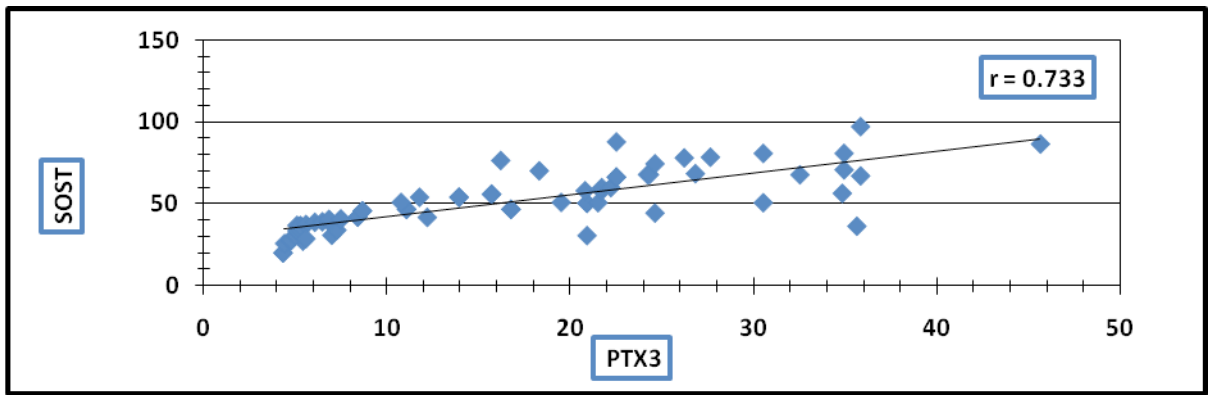


Fig (3): The Correlation between PTX3 and SOST in patients with CKD.

Discussion

The present study revealed a significant enhanced ($p<0.05$)in serum level pentraxin 3 and sclerostin in CKD patients in comparison with healthy group**.** Also the study indicateda positive association between PTX3 and SOST concentrations of CKD patients.

Many studies have confirmed that PTX3 was significantly related with kidney dysfunction and with activity or severity of the inflammatory, autoimmune and cardiovascular diseases[16-19]. The pentraxin3 (PTX3) is "acute phase protein belongs to the same family of C-reactive protein(CRP)", the inflammatory marker broadly used in clinical laboratories, unlike CRP, the PTX3 rapidly produced at inflamed sites[20]. Many recent studied confirmed that PTX3 was a sensitive and rapid marker of inflammation in CKD patients[21]. High systemic PTX3 levels were related with increment risk of cardiovascular mortality and morbidity in CKD patients[22- 24]. The study of[25] indicated that PTX3 affect on lipid accumulation promoting uptake of oxLDL via macrophages, supporting aproatherogenic mechanism for the PTX3. Other studies are confirm the actual of the balance between pro and antiatherogenic mechanism

of PTX3 in progression and development ofthe atherosclerosis in CKD patients, Also PTX3 was shown tobe innate involve in repair and tissue remodeling under an-acidic environment and hypoxiaconditions, which may perhaps support aprotective role of PTX3 through renal damage[26]. The increment in PTX3 levels are related with decrement of Glomerular filtration rate (GFR) and independently predict occurrencethe CKDin elderly women and men, the inflammatory processes are activated in early stages of the CKD and impairment of kidney function[24], [27].Thegradual increment of PTX3 associated to the decrment in Glomerular filtration rate may possiblydue to PTX3, is" a large molecular weight substance (molecular weight 40.6 kDa)" characterizevia amultimeric, generally pentameric structure, also be explainvia an "enhanced the release and synthesis and stimulation in the peripheral tissues and decline infunctioning kidney"[28].

During the studies that indicated the correlationCKD with MBD and demonstrated that sclerostin (SOST), a glycoprotein derived from osteocyte, the SOSTserve as asoluble inhibitor of Wingless Int(Wnt) signaling-pathway and it's have important physiological role to reduced the increment in bone formation, The increment in serum SOST level related with progression of chronic kidney disease[29,30]. These changes are probable explain via changes in creation of SOST in bone[31] and also explain the changes in kidney function . The research of Fang and et al.[32] who indicated, that increment in "osteocytic-protein release, vascular osteoblastic-transition and vascular calcification which happen in early phaseof chronic kidney disease. Previous studies indicated a positive correlations between serum SOST and MBD in CKD patients [33,34]. The Mutations in sclerostin structure, especially in SOST gene that encoding of SOST and enhanced Wnt signaling-pathawy which lead to aphenotype characterizevia marked increase in clinical indications and symptoms of bone disease, coinciding with an increase in levels of sclerostin[30]**.** And enhanced in circulating of SOST have revealed to predict the increment of mortality and decrement of mortality[35,36]**.**

The bone volume, Mineral density, and bone formation [37]. Many physiological factors that increase the circulating SOST level including men gender and older age [38]. Also the increment of SOST levels were related positively with Body mass index, BMD, and serum uric acid levels But negatively related with total Kt/V for urea.[38-41]. Multiple regression analysis in study of [38] indicated that SOST increment were the associated with other factors including, the GFR, serum calcium and serum phosphorus [42]. Other Studied have indicated that serum SOST increases with ageing[38], the Ageingis an independent risk factor for vascular disease and chronic kidney disease, And many traditional risk factors for CKD such smoking, diabetes, hypertension, and hyperlipidemia, nontraditional risk factors such as oxidative stress,inflammation, anemia, mineral bone abnormalities[43-49].

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Ethics Clearance: This article does not contain any studies with human participants directly or animals.

Conflict of Interest: The others declare that there is no conflict of interest

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Data Availability: All data were analyzed during this work are included in the manuscript.

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