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A study on correlates of chronic kidney disease

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Abstract
CKD is associated with increased activity of the RAAS. There is reduced blood flow in peritubular capillaries downstream of sclerosed glomeruli. As a result of this reduced effective (perceived) blood flow, glomeruli in these regions hyper secrete renin, thereby increasing circulating angiotensin II levels. Angiotensin II has a direct vasoconstrictor effect, which increases systemic vascular resistance and BP. A pre-structured proforma was prepared be used to collect demographic data which included age, gender, occupation history of present illness, Past history, family history and personal history including history of smoking, alcohol. Pearsons correlation (two tailed with 95% CI) for CKD stage and hemoglobin levels shows significant negative correlation with r value of -0.6176analysis shows with a P value of <0.0001. Pearsons correlation (two tailed with 95% CI) for CKD stage and serum creatinine levels shows positive correlation with r value of 0.9517, which is statistically significant with a P value of <0.0001.

Keywords: Chronic kidney disease, correlates, ESRD

Introduction
Chronic kidney disease (CKD) covers a wide spectrum of pathology, from early, subclinical changes in renal function in patients with multiple co-morbidities to end-stage renal disease (ESRD) where renal replacement therapy is required to sustain life.
CKD has also been recognised as a risk factor for cardiovascular disease independent of other conventional risk factors for cardiovascular disease.
Individuals with early CKD are more likely to die of cardiovascular (CV) disease than they are to progress to ESRD [1].
Patients with end-stage renal disease (ESRD) are at a much higher risk of CV disease than the general population. Premature cardiovascular disease is a significant cause of morbidity and mortality among patients with CKD [2].
CKD is associated with increased activity of the RAAS. There is reduced blood flow in peritubular capillaries downstream of sclerosed glomeruli. As a result of this reduced effective (perceived) blood flow, glomeruli in these regions hypersecrete renin, thereby increasing circulating angiotensin II levels.
Angiotensin II has a direct vasoconstrictor effect, which increases systemic vascular resistance and BP. Because there are fewer functioning glomeruli in CKD, each remaining glomerulus must increase its glomerular filtration rate (GFR): increasing systemic arterial pressure helps bolster perfusion pressure and GFR [3].
Angiotensin II also promotes sodium reabsorption in the proximal tubule and (through aldosterone) the collecting duct. Moreover, net loss of overall GFR impairs sodium excretion, which also leads to sodium retention. Sodium retention causes hypertension through volume dependent and volume-independent mechanisms.
Excess extracellular volume leads to increased perfusion of peripheral tissues, which stimulates vasoconstriction, increases peripheral vascular resistance, and therefore increases BP. Extracellular volume expansion also leads to the production of ouabain-like steroids that induce vasoconstriction and therefore increase peripheral vascular resistance. Volume-independent mechanisms include increased vascular stiffness [4].
Overactivity of the SNS in CKD stimulates renin production by the renal juxtaglomerular cells. Beyond SNS activation by sodium retention, renal ischemia also leads to renal afferent nerve excitation through adenosine. Finally experimental and clinical studies suggest that angiotensin II levels (which are higher in patients with CKD as detailed above) directly stimulate SNS activity.

Endothelial dysfunction (including impaired nitrous oxide production), oxidative stress, and elevated endothelin levels are also implicated in the pathogenesis of hypertension in patients with CKD.

Methodology

- It was hospital based cross sectional study.
- Cases selected randomly
- A pre-structured Proforma was prepared be used to collect demographic data which included age, gender, occupation history of present illness, Past history, family history and personal history including history of smoking, alcohol.
- General examination and cardiovascular examination was done according to the proforma.
- All relevant investigations were done for the all the participants. all the samples were sent to the respective laboratory departments.
- GFR was calculated using Cockcroft-Gault equation corrected to the body surface area

General examination

- Blood pressure was measured thrice and the average was taken.
- PR- was recorded regularly.
- RR – was recorded regularly.
- All the Patients was examined for Pallor, icterus, Cyanosis, Clubbing, Koilonychia and lympadenopathy.

BP monitoring

- Systolic and diastolic BP were measured in all of the subjects.
- The diagnosis of hypertension was based on a systolic BP of 140 mmHg or higher and a diastolic BP of 90 mmHg or higher.

Procedure

With the patient in a seated position and after a 5-minute rest, BP will be measured on the right arm with a mercury sphygmomanometer (cuff size, 12.5x40 cm). The systolic pressure and diastolic pressure will be read to the nearest 2 mm Hg with Phase I of Koroskoff sound corresponding to SBP and Phase V to DBP.

Systemic examination

Included Inspection, Palpation, Percussion and Auscultation

CVS Examination: Looked for S1 and S2, and for any abnormal heart sounds

Respiratory System: looked for bronchial sounds.

Per Abdomen Examination: For splenomegaly and hepatomegaly.

Results

Table 1: Correlation for CKD stage and creatinine clearance

CKD staging	Creatinine clearance	
	r value	P value
	-0.8991	<0.0001

Pearsons correlation (two tailed with 95% CI) for CKD stage and creatinine clearance shows negative correlation with r value of -0.8991, which is statistically significant with a P value of <0.0001****.

Table 2: Hb % AND CKD

Stages of CKD	Hemoglobin(g/dl)			
	< 7g/dl	7 to 10 g/dl	> 10 g/dl	Total
Stage II	-	01	05	06 (6%)
Stage III	04	30	20	54 (54%)
Stage IV	10	20	-	30 (30%)
Stage V	06	04	-	10 (10%)
TOTAL	20	55	25	100 (100%)

Table 3: Correlation for CKD stage and hemoglobin levels

CKD	Hemoglobin levels	
	r value	P value
	-0.6176	<0.0001

Pearsons correlation (two tailed with 95% CI) for CKD stage and hemoglobin levels shows significant negative correlation with r value of -0.6176analysis shows with a P value of <0.0001****.

Table 4: CKD & serum Phopshate

Stages of CKD	Serum Phosphate			Total
	<4.5 mg/dl	4.5-6.5 mg/dl	>6.5 mg/dl	
Stage II	06	-	-	06
Stage III	-	32	22	54
Stage IV	-	20	10	30
Stage V	-	05	05	10
Total	06	57	37	100

Table 5: Correlation for CKD stage and serum phosphate levels

CKD	Serum phosphate	
	r value	P value
	0.2058	0.04*

Pearsons correlation(two tailed with 95% CI) for CKD stage and serum phosphate levels shows a positive correlation with r value of 0.2058,analysis shows with a P value of 0.0400 *, which is mildly significant.

Table 6: CKD and GFR

Stages of CKD	GFR (ml/min)				Total
	60-89	30-59	15-29	<15	
Stage II	06	-	-	-	06
Stage III	-	54	-	-	54
Stage IV	-	-	30	-	30
Stage V	-	-	-	10	10
TOTAL	06	54	30	10	100

Table 7: Correlation for CKD stage and GFR

CKD	GFR	
	r value	P value
	-0.9580	0.04*

Pearsons correlation (two tailed with 95% CI) for CKD stage and GFR shows negative correlation with r value of -0.9580 which is highly statistically significant with a P value of <0.0001****.

Table 8: Distribution of serum creatinine levels according to stages of CKD

Stages of CKD	< 1.4	1.4 to 2.4 mg/dl	2.4 to 5 mg/dl	5 to 10 mg/dl	>10mg/ dl	Total
Stage II	-	06	-	-	-	06
Stage III	-	-	54	-	-	54
Stage IV	-	-	-	30	-	30
Stage V	-	-	-	-	10	10
Total	-	06	54	30	10	100

Table 9: Correlation for CKD stage and serum creatinine levels

CKD	Serum creatinine levels	
	r value	P value
	0.9517	0.04*

Pearsons correlation (two tailed with 95% CI) for CKD stage and serum creatinine levels shows positive correlation with r value of 0.9517, which is statistically significant with a P value of <0.0001****.

Discussion

The term ‘‘CKD-associated mineral and bone disorders’’ comprises abnormalities in bone and mineral metabolism and/or extra skeletal calcification secondary to CKD pathophysiology.

Renal osteodystrophy is the spectrum of histologic changes that occur in bone architecture of patients with CKD. The kidney is the primary site for phosphate excretion and 1-a-hydroxylation of vitamin D [5].

CKD patients develop hyperphosphatemia as a result of inadequate 1,25 dihydroxy-vitamin D levels that reflect reduced synthesis from parenchymal scarring. In addition, renal phosphate excretion is reduced. Together, both processes cause serum calcium levels to fall resulting in increased secretion of parathyroid hormone (secondary hyperparathyroidism). Parathyroid hormone has a phosphaturic effect. It also increases the calcium levels by increasing bone resorption and promoting 1-a-hydroxylation of 25-hydroxy vitamin D synthesized by the liver (limited effect because of reduced kidney reserve from scarring) [6].

Rising phosphorus levels are almost universally observed in stage 3 CKD patients. However, secondary hyperparathyroidism often begins to distort bone architecture earlier before serum phosphorus is noted to be abnormal, indicating that phosphate binder therapy needs to be initiated when eGFRs have declined below 50 mL/min per 1.73 m2.

Changes in bone architecture can be caused by either a high bone turnover state or a low bone turnover state.

Four types of bone phenotypes (renal osteodystrophy) can be diagnosed in CKD patients:

Osteitis Fibrosa cystic (high bone turnover with secondary hyperparathyroidism),

Osteomalacia (low bone turnover and inadequate mineralization, primarily related to diminished vitamin D synthesis), Adynamic bone disorder (low bone turnover from excessive suppression of the parathyroid glands), and

The predominant type of renal osteodystrophy and CKD-mineral and bone disorder differs between predialysis and end stage renal disease patients. In predialysis patients, high bone turnover bone disease is most prevalent. In contrast, low bone turnover predominates in dialysis patients. Patients with low turnover disease represent most cases of renal osteodystrophy. The cause of this prevalent bone phenotype results from over suppression of parathyroid hormone and high calcium dialysate concentrations [7].

Acidosis, the suppressive effect of phosphate retention on renal synthesis of 1,25 dihydroxy-vitaminD synthesis, and absence of the physiologic inhibitory effect of vitamin D on parathormone secretion are also minor factors that contribute to the low turnover bone disease in CKD patients [8].

Conclusion

CKD-associated mineral bone disorders significantly increase mortality in CKD patients. In fact, hyperphosphatemia is one of the most important risk factors associated with cardiovascular disease in CKD patients. The exact mechanism underlying this association remains unclear. It is believed to be related to hyperparathyroidism [52] and vascular calcification, which results from high phosphorus levels.

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