International Journal of Medical and Health Research
ISSN: 2454-9142
Impact Factor: RJIF 5.54
www.medicalsciencejournal.com
Volume 3; Issue 12; December 2017; Page No. 08-10

Estimation of levels of serum prolactin in chronic kidney disease

Dr. Anil Kumar Mahto
Senior Resident, Department of Nephrology, Indira Gandhi Institute of Medical Sciences, Patna, Bihar, India

Abstract
Raised levels of serum prolactin which arises in CKD may contribute to vascular derangements. This might lead to worse cardiovascular consequences amongst CKD patients. Hence the present study was planned to assess the serum prolactin levels in CKD patients.
The study has planned in IGIMS Patna, The 50 patients diagnosed with chronic kidney disease were enrolled in to the study. The age group of the patients are from 25-65 years.
The data generated in the present study showed that Hyperprolactinemia is detected in 52% of patients with CKD. From the above findings it can be concluded that CKD is associated with increased serum levels of hormone prolactin (Hyperprolactinemia).

Keywords: hyperprolactinemia, prolactin, chronic kidney disease

Introduction

Chronic kidney disease (CKD) is progressive loss in kidney function over a period of months or years. The symptoms of worsening kidney function are not specific, and might include feeling generally unwell and experiencing a reduced appetite. Often, chronic kidney disease is diagnosed as a result of screening of people known to be at risk of kidney problems, such as those with high blood pressure or diabetes and those with a bloodline relative with CKD. This disease may also be identified when it leads to one of its recognized complications, such as cardiovascular disease, anemia, pericarditis or renal osteodystrophy (the latter included in the novel term CKD-MBD) [1, 2]. CKD is a long-term form of kidney disease; thus, it is differentiated from acute kidney disease (acute kidney injury) in that the reduction in kidney function must be present for over 3 months. CKD is an internationally recognized public health problem affecting 5–10% of the world population [3, 4].

Chronic kidney disease is identified by a blood test for creatinine, which is a breakdown product of muscle metabolism. Higher levels of creatinine indicate a lower glomerular filtration rate and as a result a decreased capability of the kidneys to excrete waste products. Creatinine levels may be normal in the early stages of CKD, and the condition is discovered if urinalysis (testing of a urine sample) shows the kidney is allowing the loss of protein or red blood cells into the urine. To fully investigate the underlying cause of kidney damage, various forms of medical imaging, blood tests, and sometimes a kidney biopsy (removing a small sample of kidney tissue) are employed to find out if a reversible cause for the kidney malfunction is present [1].

Previous professional guidelines classified the severity of CKD in five stages, with stage 1 being the mildest and usually causing few symptoms and stage 5 being a severe illness with poor life expectancy if untreated. Stage 5 CKD is often called end-stage kidney disease, end-stage renal disease, or end-stage kidney failure, and is largely synonymous with the now outdated terms chronic renal failure or chronic kidney failure; and usually means the patient requires renal replacement therapy, which may involve a form of dialysis, but ideally constitutes a kidney transplant. Recent international guidelines reclassified CKD based on cause, glomerular filtration rate category (G1, G2, G3a, G3b, G4 and G5), and albuminuria category (A1, A2, A3) [5].

The most common recognized cause of CKD is diabetes mellitus. High blood pressure is also a very common cause of chronic kidney disease. Other causes of CKD include idiopathic (i.e. unknown cause, often associated with small kidneys on renal ultrasound) and glomerulonephritis [6]. Together, these cause about 75% of all adult cases.

Historically, kidney disease has been classified according to the part of the kidney anatomy involved [7].

- Vascular disease includes large vessel disease such as bilateral renal artery stenosis and small vessel disease such as ischemic nephropathy, hemolytic-uremic syndrome, and vasculitis.
- Glomerular disease comprises a diverse group and is classified into:
  - Primary glomerular disease such as focal segmental glomerulosclerosis and IgA nephropathy (or nephritis)
  - Secondary glomerular disease such as diabetic nephropathy and lupus nephritis
- Congenital disease such as polycystic kidney disease.
- Tubulointerstitial disease includes drug- and toxin-induced chronic tubulointerstitial nephritis, and reflux nephropathy.
- Obstructive nephropathy is exemplified by bilateral kidney stones and diseases of the prostate such as benign prostatic hyperplasia.
- On rare cases, pinworms infecting the kidney can also cause nephropathy.
- Nontraditional causes of CKD (CKDu) are denoted if the common causes of CKD are not present:

- CKD of unknown cause is the subject of study by the Sri Lanka Ministry of Health and the World Health Organization 2009–2012 [8].
- Mesoamerican nephropathy, a form of CKDu, is "a new form of kidney disease that could be called agricultural nephropathy" [9].

Prolactin (PRL), also known as luteo tropic hormone or luteotropin, is a protein that is best known for its role in enabling mammals, usually females, to produce milk. It is influential in over 300 separate processes in various vertebrates, including humans. Prolactin is secreted from the pituitary gland in response to eating, mating, estrogen treatment, ovulation and nursing. Prolactin is secreted in pulses in between these events. Prolactin plays an essential role in metabolism, regulation of the immune system and pancreatic development.

There are numerous works showed recently are display that prolactin may have several biologic actions that contribute in the atherosclerotic progression and leads to insulin resistance. It is also connected with endo the lial dysfunction. Hyperprolactinemia is establishing in patients with essential hypertension, acute phase of coronary syndrome, during ischemic stroke, and transient ischemic attacks and in preeclampsia.

Raised levels of serum prolactin which arises in CKD may contribute to vascular derangements. This might lead to worse cardiovascular consequences amongst CKD patients. Hence the present study was planned to assess the serum prolactin levels in CKD patients.

Methodology

The study has planned in IGIMS Patna. The 50patients diagnosed with chronic kidney disease were enrolled in to the study. The age group of the patients are from 25-65 years. The patients visited to Out Patient Department (OPD) and in-patient department (IPD) of IGIMS Patna, were considered in the study. All the patients are informed consents. All the patient's clinical history were collected. The approval of the institutional ethical committee is taken for the planned study.

The following was the Inclusion Criteria for the present study:

- Symptoms of uremia for 3 months or more Elevated blood urea, serum creatinine, and decreased creatinine clearance
- Patients with established CKD on maintenance dialysis irrespective of etiology

The following was the Exclusion Criteria for the present study:

- Pregnant Females
- Patients of seizure disorder
- Patients of Chronic liver diseases
- Thyroid patients

Blood test and urine tests were performed and data were collected. After selecting the patients, about 5 ml of blood sample is collected in a nonheparinized bottle, and quantitative determination of serumprolact in was done by fully automated bidirectionally interfaced chemiluminescent immunoassay (CLIA).

The reference range for serum prolactin is given as follows:

- Normally menstruating females: 2.8-29.2 ng/ml
- Pregnant women: 9.7-208.5 ng/ml
- Postmenopausal women: 1.8-20.3 ng/ml
- Men: 2.1-17.7 ng/ml.

Results & Discussion

The data from the 50 enrolled patients is collected and presented as below. Out of the 50 patients 45 were males and 5 patients were females. The various serum and renal markers were measured in the enrolled group of patients.

The creatinine clearance is observed as below.

Table 1: creatinine clearance

Creatinine clearance	30-60 ml/min	15-30 ml/min	Less than 15 ml/min
Chronic Kidney Disease Stage	Stage III	Stage IV	Stage V
Number of patients	4	20	26

Out of 50 study cases stage III CKD patients are 4 this are having creatinine clearance of 30-60 ml/min. 20 patients who are in Stage IV of CKD were having the creatinine clearance of 15-30 ml/min. The Stage V CKD patients are 26 having creatine clearance of Less than 15 ml/min.

Table 2: Blood Urea Levels

Blood Urea mg/dl	Number of Cases
Less than 60	4
60-80	10
80-100	6
100-120	14
120-140	10
More than 140	6

The maximum number of 14 cases are observed with the blood urea level 100-120 mg/dl. Thereafter 10 cases are having blood urea level of 60-80 mg/dl and 120-140 mg/dl.

Table 3: Serum Creatinine

Serum Creatinine mg/dl	Number of Cases
Less than 5	24
5-10	18
10-15	6
15-20	2

The highest 24 cases are having serum creatinine less than 5 mg/dl. There are 18 cases are showing the serum creatinine 5-10 mg.dl.

Table 4: Serum Prolactin

Observations	Number of Cases
Positive	26
Negative	24

A Similar study of evaluating serum prolactin levels in chronic renal failure patients, CKD patients on hemodialysis, and transplant recipients was conducted by Peces *et al.* In this study, Peces *et al.* conducted serum prolactin estimation in twelve patients with CKD who were on conservative line of treatment and thirty patients with CKD who were on hemodialysis, and nineteen patients with CKD who were post-transplant recipients with a functioning kidney [10].

In this study, Peces *et al.* alsohave shown that Serum prolactin

levels remain normal in those CKD patients who have received a transplant with functioning kidney. In thisstudy, authors have attributed the increased serum prolactin hormone levels to decreased renal catabolism and impaired hypothalamo–pituitary regulation [10].

A similar study of evaluating hyperprolactinemia and impaired pituitary response to suppression and stimulation in patients with CKD was conducted by V.S. Lim, S.C. Kathpalia, and L. Frohaman [11]. In this study, authors also analyzed the reversibility of the above-mentioned abnormalities with renal transplantation. In this study, authors have demonstrated increased basal serum prolactin levels in patients with CKD. In this study, authors also have demonstrated that prolactin hormone showed a lack of responsiveness to suppressive as well as stimulatory agents. They attributed this lack of responsive ness to pathology at the pituitary either at the level of receptor binding or apost-receptor level.

Conclusion

The data generated in the present study showed that Hyperprolactinemia is detected in 52% of patients with CKD. From the above findings it can be concluded that CKD is associated with increased serum levels of hormone prolactin (Hyperprolactinemia).

References

1. National Kidney Foundation. K/DOQI clinical practice guidelines for chronic kidney disease. Retrieved, 2002-2008.
2. KDIGO. Kidney Disease Improving Global Outcomes. KDIGO Clinical Practice Guideline for the Diagnosis, Evaluation, Prevention, and Treatment of Chronic Kidney Disease-Mineral and Bone Disorder CKD-MBD PDF. Kidney Int., 2009; 76(Suppl 113).
3. Eknoyan G, Lameire N, Barsoum R *et al.* The burden of kidney disease: improving global outcomes. Kidney Int., 2004; 66:1310-14.PMID 15458424.doi:10.1111/j.1523-1755.2004.00894.x.
4. A Martínez-Castelao, JL Górriz, J Bover *et al.* Consensus document for the detection and management of chronic kidney disease. Nefrologia. 2014; 34(2):243-62.PMID 24658201.doi:10.3265/Nefrologia.pre2014.Feb.12455.
5. KDIGO Kidney Disease Improving Global Outcomes. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease PDF. Kidney Int Suppl. 2013; 3(1):1-150.2016.
6. United States Renal Data System USRDS. Archived from the original, 2007.
7. Rahman, Mahboob, Smith, Michael C. Chronic renal insufficiency: A diagnostic and therapeutic approach. Archives of Internal Medicine. 1998; 158:1743-52.PMID 9738603. doi:10.1001/archinte.158.16.1743.
8. Redmon JH, Elledge MF, Womack DS, Wickremashinghe R, Wanigasuriya KP, Peiris-John RJ *et al.* Additional perspectives on chronic kidney disease of unknown aetiology CKDu in Sri Lanka - lessons learned from the WHO CKDu population prevalence study. BMC Nephrology. 2014; 15(1):125.PMC-4120717.PMID 25069485.doi:10.1186/1471-2369-15-125.
9. Orantes CM, Herrera R, Almaguer M, Brizuela EG, Núñez L, Alvarado NP *et al.* Epidemiology of chronic kidney disease in adults of Salvadoran agricultural communities. MEDICC Rev. 2014; 16(2):23-30.PMID 24878646.
10. Peces R, Casado S, Frutos M, Horcajada C, López-Novoa JM, Hernando L. Prolactin in chronic renal failure, haemodialysis,and transplant patients. Proc Eur Dial Transplant Assoc, 1979; 16:700-2.
11. Lim VS, Kathpalia SC, Frohman LA. Hyperprolactinemia and impaired pituitary response to suppression and stimulation in chronic renal failure: Reversal after transplantation. J Clin Endocrinol Metab. 1979; 48(1):101-7.