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Research Article

**ANALYSIS OF SERUM LEVELS OF CYSTATIN-C FOR THE
EARLY DETECTION OF PRE-HYPERTENSIVE
NEPHROPATHY**Dr Nadia Umar¹, Dr Waqar Shad², Dr Kiran Sadiq³¹Ayub Medical College, Abbottabad, ²Latin American School of Medicines, Cuba., ³WMO at BHU Kot Sai Singh Jhangh.

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Abstract:

Introduction: Chronic kidney disease (CKD) is common in geriatric cats, with a prevalence from 30% up to 60% in cats older than 10 years.

Aims and objectives: The main objective of the study is to analyse the serum levels of Cystatin-C for the early detection of pre-hypertensive nephropathy.

Material and methods: This cross sectional study was conducted in Ayub Medical College, Abbottabad during November 2018 to March 2019. The data was collected from 100 nephropathic patients. The data was collected through non-probability sampling technique. Those with slightly raised serum creatinine and those with blood sugar levels within pre-diabetes range on previous reports were excluded, and so were those who gave history of antihypertensive medication or use of steroid. Informed consent was taken from all study participants.

Results: The study subjects included 80 patients, 48 of them were male (60%) and 32 were female (40%). With a mean age of 59.85 ± 9.976 and age range of 41-80 years. All patients underwent elective CAG or PCI. According to the presented data and to the definition CIN and its grading in our study, CIN occurred in 19 patients 23.8%. The incidence of CIN was 0.0% in patients with serum creatinine increase <25% above baseline. While incidence in those with serum creatinine increase $\geq 25\%$.

Conclusion: It is concluded that cystatin C is the leading biomarker in the early detection of pre hypertensive nephropathy. Diagnostic markers which reflect renal impairment at early stage is important as early intervention can slow the loss of kidney function and reduce adverse clinical outcomes.

Corresponding author:**Dr. Nadia Umar,**

Ayub Medical College, Abbottabad.

QR code



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INTRODUCTION:

Chronic kidney disease (CKD) is common in geriatric cats, with a prevalence from 30% up to 60% in cats older than 10 years. Since CKD is an irreversible and progressive disease, early detection and treatment is of major importance, aiming to slow down disease progression and to improve quality of life and longevity. Glomerular filtration rate (GFR) is considered the gold standard method to evaluate kidney function, but measurement is time-consuming and is not routinely used [1]. Therefore, the indirect GFR markers, serum creatinine (sCr), and urea, are routinely measured to estimate GFR. However, these markers are insensitive. It is widely reported but poorly documented that their serum concentration only increases when approximately 75% of the functional renal mass is lost [2].

Moreover, they are both influenced by muscle mass, age, feeding status, sex, and intra individual variation. All those disadvantages support the need for new indirect biomarkers that can be measured easily and reliably. Cystatin C (CysC), a 13 kDa protein, is a proteinase inhibitor, produced in every nucleated cell at a constant rate, that is responsible for intracellular protein catabolism [3].

Most of the properties required for an ideal endogenous GFR marker apply for CysC.12 Compared to sCr, several human and canine studies have shown a better correlation of sCysC with GFR [4]. In addition, urinary Cystatin C (uCysC) is a biomarker for tubular damage in humans and dogs. In a pilot study, our group observed a significant difference in sCysC and uCysC concentration between CKD and healthy cats. We also validated the human particle enhanced nephelometric immunoassay (PENIA) for CysC measurement in cats and established a reference interval (RI) of 0.58–1.95 mg/L for sCysC [5]. In addition, we demonstrated that there is no influence of breed, age, and sex on feline sCysC and that it is not mandatory to withhold

food in cats prior to evaluation of feline sCysC. These findings make sCysC a promising marker to estimate GFR in feline medicine [6].

Aims and objectives:

The main objective of the study is to analyse the serum levels of Cystatin-C for the early detection of pre-hypertensive nephropathy.

MATERIAL AND METHODS:

This cross sectional study was conducted in Ayub Medical College, Abbottabad during November 2018 to March 2019. The data was collected from 100 nephropathic patients. The data was collected through non-probability sampling technique. Those with slightly raised serum creatinine and those with blood sugar levels within pre-diabetes range on previous reports were excluded, and so were those who gave history of antihypertensive medication or use of steroid. Informed consent was taken from all study participants. Five millilitres of blood was collected from antecubital vein of each subject under aseptic conditions and serum was subsequently obtained through ultracentrifugation which was subsequently stored at -80 degrees Celsius in Eppendorf tubes for the measurement of biochemical parameters i.e.; Cystatin-C and creatinine.

Statistical analysis:

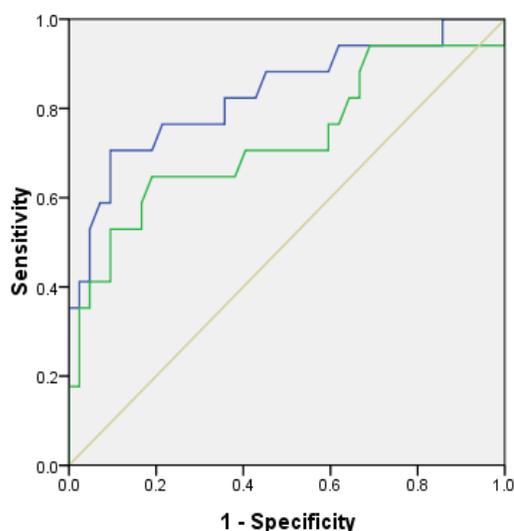
Probability (P) value were considered significant when it is less than 0.05%, and highly significant if it is less than 0.01%.

RESULTS:

The study subjects included 80 patients, 48 of them were male (60%) and 32 were female (40%). With a mean age of 59.85 ± 9.976 and age range of 41-80 years. All patients underwent elective CAG or PCI. According to the presented data and to the definition CIN and its grading in our study, CIN occurred in 19 patients 23.8%. The incidence of CIN was 0.0% in patients with serum creatinine increase <25% above baseline. While incidence in those with serum creatinine increase $\geq 25\%$.

Table 01: Changes of serum and urinary markers between CIN and non-CIN groups.

Markers	CI	Non-CIN	P Value
S. Urea (mg/dl)			
At 0 hours	36.54 ± 12.91	31.64 ± 7.03	0.036*
After 24 hours	50.20 ± 25.06	39.20 ± 13.23	0.015*
S. Creatinine (mg/dl)			
At 0 hours	0.77 ± 0.22	0.88 ± 0.18	0.037*
After 24 hours	1.30 ± 0.29	0.98 ± 0.19	0.001***
S. Cystatin C (ng/ml)			
At 0 hours	4.66 ± 5.31	2.56 ± 2.40	0.018*
After 24 hours	22.94 ± 20.73	9.46 ± 10.25	0.001***
eGFR			
At 0 hours	102.47 ± 43.04	82.52 ± 19.94	0.006**
After 24 hours	56.57 ± 18.42	72.11 ± 15.89	0.001**

**Figure 01:** ROC curve of statin therapy in patients**DISCUSSION:**

Acute kidney injury is an important topic because it has a significant impact on morbidity and mortality and hospital survival for patients. CIN is one of the main causes of AKI. It is known that occurrence of CIN depends on the level of serum creatinine. According to the definition, CIN was defined as an increase of more than 25% from the baseline value of serum creatinine or an absolute increase of at least 0.5 mg/dL within 48 h after the administration of contrast media [7].

In this study 80 patients who underwent elective PCI and CAG, the incidence of CIN was occurred in 19 (2 diagnostic and 17 therapeutic) patients frequent 23.8%

after 24 h. The results showed even patients with normal kidney function have a CIN after exposure to contrast media. Numerous studies have different results about percentage of development of CIN [8]. Nassir in 2014 studied 42 patients with type 2 diabetes mellitus who developed CIN in following PCI. He found the incidence of CIN, in diabetic patients approximately 2.6 times compared with non-diabetic 48 h after the procedure [9]. A recent study was performed by Wang et al. that include 300 patients and found that only 29 patients of them developed of CIN [16]. Another report Alharazy et al. includes 100 patients with chronic kidney disease undergoing coronary angiography. The frequency of CIN in them was 11% and 1 patient required dialysis [10]. While

Ribichini et al. studied 166 patients and he measured their serum creatinine and cystatin C at baseline and at 12, 24 and 48 h after exposure to contrast media. He found that CIN occurred in 30 patients (18%) [11]. A prospective study of consecutive 87 patients who underwent elective PCI and CAG, 31 patients had a moderate kidney disease, CIN occurred in 18 patients and was more frequent 42.0%. The reason for the difference in incidence rates of CIN among different studies including this study may be due to differences in the definitions of CIN [12].

The pre-hypertensive Pakistani population has been documented to have higher circulating levels of various stress hormones like cortisol and aldosterone. Higher circulating levels of stress hormones have been attributable to stressful life at a younger age which will ultimately develop into full-blown hypertension and its associated complications like renal dysfunction [9]. The earlier stages of reversible kidney disease have been coined as pre-chronic kidney disease that manifests itself with the excretion of low amount of albumin protein in the urine known as microalbuminuria [10]. Hypertension leads to pathophysiological.

CONCLUSION:

It is concluded that cystatin C is the leading biomarker in the early detection of pre hypertensive nephropathy. Diagnostic markers which reflect renal impairment at early stage is important as early intervention can slow the loss of kidney function and reduce adverse clinical outcomes.

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