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A Study of Metocarpocortical Index (MCI) in Chronic Renalfailure

KEYWORDS: Metocarpocortical index, chronic renal failure, renal osteodystophy

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ABSTRACT *Objectives: To document renal osteodystrophy in chronic renal failure patients by measuring metacarpocortical index (MCI)*

Methods: A hospital based prospective observational study of metacarpocorticalindex in chronic renal failure patients calculated by taking X-ray of second metacarpal bone.

Results: A total 50 patients diagnosed with chronic renal failure were studied. Mean Metacorpocortical index in study group chronic renal failure patients was 0.387 whereas in controls group was 0.575. Metacorpocortical index was decreased by rise of serum creatinine, urea, phosphorus, serum alkaline phosphatase and serum uric acid. Metacorpocortical index was increased by increase of serum calcium.

Conclusions: The study revealed that renal osteodystophy in CRF patients can be measured by simple reliable and accessible method of calculating metacarpocortical index.

INTRODUCTION

Chronic renal failure is a pathophysiological process with multiple etiologies resulting in the inexorable attrition of nephron number and function and frequently leading to end stage renal disease.

Uremia is the clinical and laboratory syndrome reflecting dysfunction of all organ systems as a result of untreated or under treated acute or chronic renal failure.

Bone disease is observed in 75-100% of patients with chronic renal failure as the glomerular filtration rate (GFR) falls below 60 ml/minute. Renal osteodystrophy is an almost universal consequence of chronic renal insufficiency and is associated with rickets in child hood and osteomalacia in adults, hyperparathyroidism, osteosclerosis and osteoporosis.

In normal bone, the remodeling process of removal and replacement is tightly coupled. Osteoblasts are responsible for the production of bone matrix constituents such as collagen and ground substances which become mineralized and the multinucleated osteoclasts which are in contact with calcified bone surfaces reabsorb bone.

End-stage renal disease (ESRD) may result in abnormal turnover, coupling, and mineralization. As nephron loss causes the glomerular filtration rate (GFR) to fall below 60 ml/minute, phosphate is retained inducing a rise in parathyroid hormone (PTH) and a decline in 1,25-dihydroxyvitamin D levels/1'^ As described in 1969 and termed the "trade-off hypothesis."

The PTH rise reduces renal phosphate reabsorption so that phosphate balance is restored. Calcium levels are maintained by the rise in PTH as is 1, 25-dihydroxyvitamin D homeostasis, providing adequate renal mass remains. In 50% of patients, abnormal bone histology is present at this level of renal function.® With further reduction in the GFR to 20-40 ml/minute, 1,25-dihydroxyvitamin D levels fall below normal[(i)] and calcium and phosphate homeostasis cannot be maintained. Skeletal PTH resistance also increases with worsening uremia and in the immediate predialysis period almost all patients have abnormal bone histology.

Hyperparathyroid (high turnover) bone disease is found most frequently followed by mixed osteodystrophy, low-turnover bone disease, and osteomalacia. With advancing renal impairment, "skeletal resistance" to parathyroid hormone (PTH) occurs. To maintain bone turnover, intact PTH (iPTH) targets from two to four times the upper normal range have been suggested, but whole PTH (1-84) assays indicate that amino-terminally truncated fragments, which accumulate in end-stage renal disease (ESRD), account for up to one-half of the measured iPTH. PTH levels and bone-specific alkaline phosphatase (BSAP) provide some information on bone involvement but bone biopsy and histomorphometry remains the gold standard. Calcitriol and calcium salts can be used to suppress PTH and improve osteomalacia but there is growing concern that these agents predispose to the development of vascular calcification, cardiovascular morbidity, low-turnover bone disease and fracture. Newer therapeutic options include less calcemic vitamin D analogues, calcimimetics and bisphosphonates for hyperparathyroidism, and sevelamer for phosphate control. Calcitriol and hormone-replacement therapy (HRT) have been shown to maintain bone mineral density (BMD) in certain patients with end-stage renal disease (ESRD). After renal transplantation, renal osteodystrophy generally improves but BMD often worsens. Bisphosphonate therapy may be appropriate for some patients at risk of fracture.

When renal bone disease is assessed using a combination of biochemical markers, histology and bone densitometry, early intervention and the careful use of an increasing number of effective therapies can reduce the morbidity associated with this common problem.

One of the earliest radiological changes in chronic renal

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failure is metacarpocortical index (MCI). It is sum of medial +lateral cortical thickness of second metacarpal bone at mid point divided by total thickness of second metacarpal bone.

This study is conducted to measure bone density by calculating metacarpocortical thickness of second metacarpal bone by X-ray which is simple and reliable method to predict bone changes (MCI index) in comparison with biochemical parameters like serum creatinine, urea, calcium, phosphorus, alkaline phosphatase and uric acid.

AIM OF THE STUDY

1) To calculate metacarpocortical index (MCI) and predict quantitative bone changes in Chronic Renal failure (CRF) patients.
2) Comparison between metacarpocortical index in CRF with biochemical parameters like blood urea, serum creatinine, calcium, phosporus, alkaline phosphatase and uric acid.

MATERIALS AND METHODS

This study was conducted on 50 patients of chronic renal failure admitted in Gandhi Hospital, Secunderabad, in the department of General medicine and Nephrology. A control group of 50 persons was studied

Inclusion Criteria:

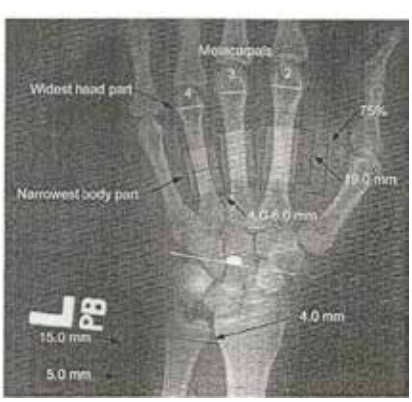
Study group:	Controlgroup:
a)CRF of any cause	a) No evidence of CRF
b)Both male and female	b) Apparentlyhealthy
c)Age between 18yrs-50yrs in male	c) Age between 18yrs-50yrs in male
d) Age between 18yrs-45yrs in female	d)Age between 18yrs-45yrs in female

Exclusion Criteria:
Patients with

a) Acute renal failure
b) Bones changes other than CRF
c) Rickets
d) Drug intake (steroids)

METHODS OF STUDY:

This study was done by doing the simple X-ray of right hand anteroposterior view to detect metacarpoarticortical index by measuring medial plus lateral cortical thickness in the mid shaft of the second metacarpal bone divided by the total thickness of the mid shaft of second metacarpal bone. Metacarpocortical index (MCI) was correlated with simple biochemical parameters like serum levels of creatinine, urea, calcium, phosphorus, alkaline phosphatase, uric acid.

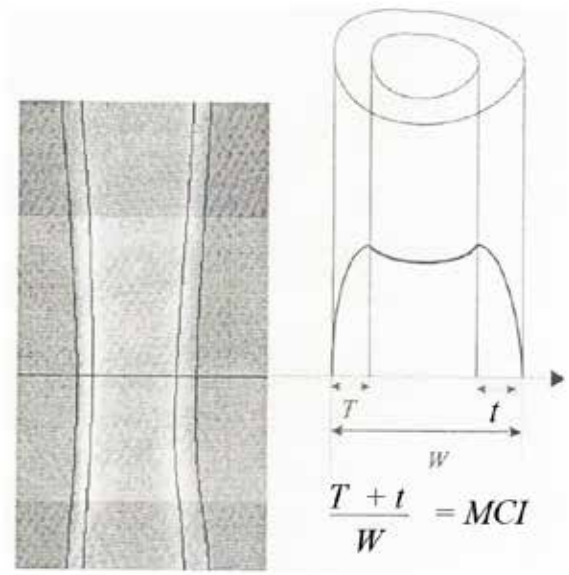


Calculation of MCI

X-ray of AP view of right hand was taken.

MCI =

Medial + lateral cortical thickness of second metacarpal bone at midpoint / Total thickness of second metacarpal-bone at mid point



Blood was drawn from study group (CRF) in the fasting and sent to Biochemistry laboratory of Gandhi Hospital for the following investigations, such as Serum creatinine, Blood urea, Serum calcium, Serum phosphorous, Alkaline phosphatase, Uric acid.

Results were tabulated and analyzed.

RESULTS AND ANALYSIS

The metacarpoarticortical index (MCI) value in the study group (CRF) was 0.3876

The metacarpoarticortical index (MCI) in control group was 0.575.

Serum Creatinine vs Metacarpocortical Index (MCI)
TABLE 1

S .No	Serum Creatinine (mg/dl)	Metacarpocortical Index (MCI)
1	<1 .4(control group)	0.575
2	1.4-5	0.478
3	5-10	0.40
4	10-15	0.37
5	>15	0.34

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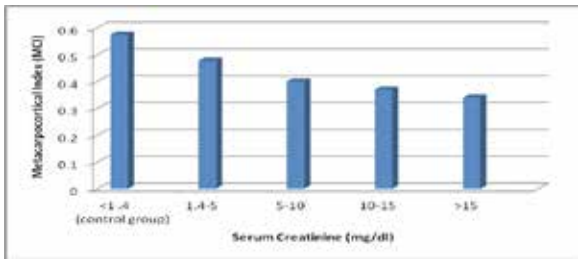
GRAPH 1



In graph 1 and table1, with the increase in serum creatinine levels, there decline in the MCI value.

Blood Urea vs Metacarpocortical Index (MCI)
TABLE 2

S.No	Blood Urea (mg/dl)	Metacarpocortical Index (MCI)
1	<20	0.575
2	20-99	0.453
3	100-199	0.392
4	200-249	0.389
5	>250	0.376

GRAPH 2

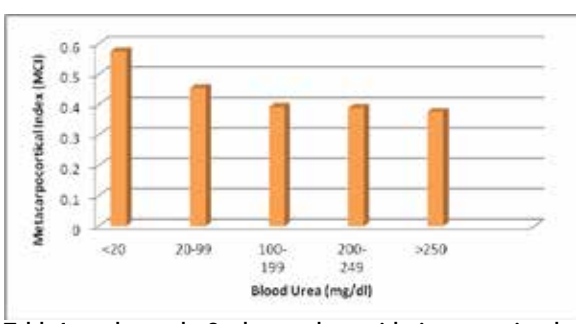
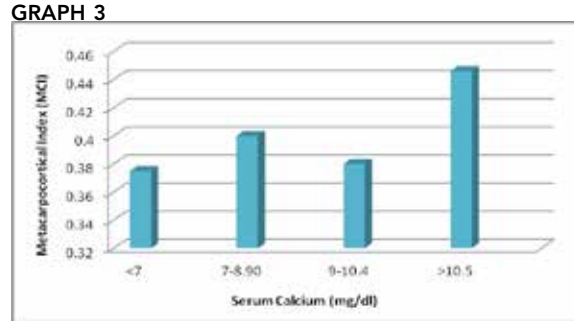


Table1 and graph 2 shows that with increase in the blood urea levels there is decline in the MCI value.

Serum Calcium vs Metacarpocortical Index (MCI)
TABLE 3

S.No	Serum Calcium (mg/dl)	Metacarpocortical Index (MCI)
1	<7	0.3748
2	7-8.90	0.40
3	9-10.4	0.38
4.	>10.5	0.446

GRAPH 3



The table3 and graph 3 is showing that with increase in the serum calcium levels there is increase in the MCI value.

Serum Phosphorus Level vs Metacarpocortical Index (MCI)

TABLE 4

S.No	Serum Phosphorus (mg/dl)	Metacarpocortical Index (MCI)
1	<4.5	0.5056
2	4.5-8	0.4594
3	8.1-12	0.396
4	>12	0.3478

GRAPH 4

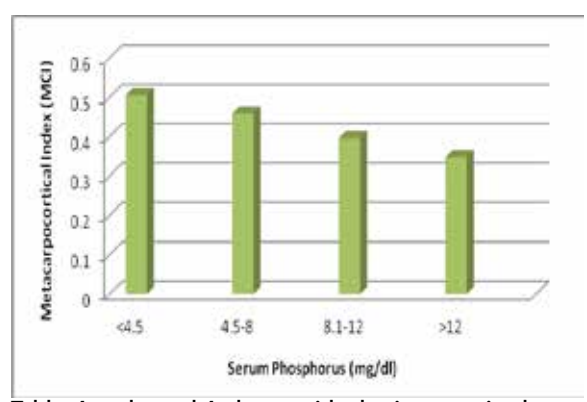


Table 4 and graph4 shows with the increase in the serum phosphorus levels, there is decline in the MCI.

Serum alkaline phosphatase vs Metacarpocortical Index (MCI)

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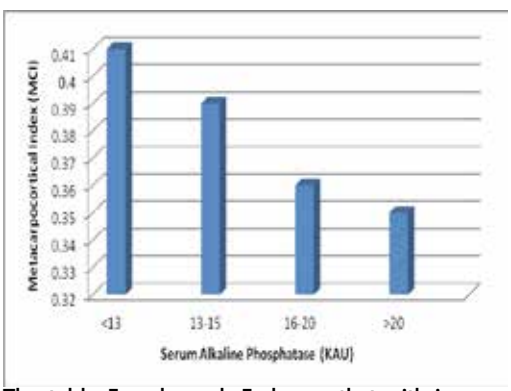
TABLE 5

S.No	Serum Alkaline Phosphatase (KAU)	Metacarpocortical Index (MCI)
1	<13	0.41
2	13-15	0.39
3	16-20	0.36
4	>20	0.35

GRAPH 5

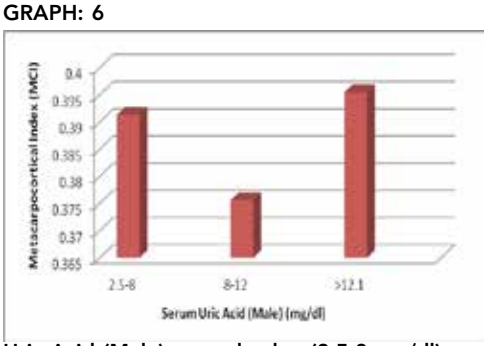


The table 5 and graph 5 shows that with increase in the serum alkaline phosphatase levels there is decline in the MCI.

Uric acid vs Metacarpocortical Index (MCI)
TABLE 6

S.No	Uric Acid (Male) (mg/dl)	Metacarpocortical Index (MCI)
1	2.5-8	0.3913
2	8-12	0.3756
3	>12.1	0.39548

GRAPH: 6

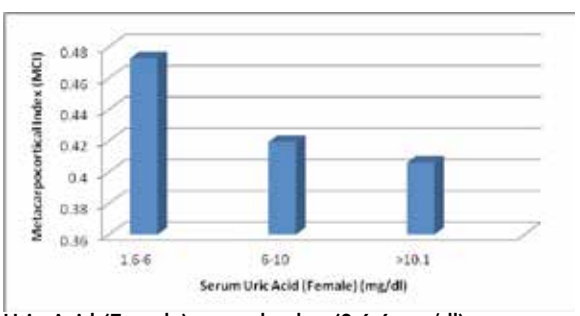


Uric Acid (Male) normal value (2.5-8 mg/dl)
The table6 and graph 6 shows that with increase in the serum uric acid levels there is decline in the MCI index.

Uric Acid (Female) vs Metacarpocortical Index (MCI)
TABLE 7

S.No	Uric Acid (Female) (mg/dl)	Metacarpocortical Index (MCI)
1	1.6-6	0.4725
2	6-10	0.419
3	>10.1	0.4059

GRAPH: 7



Uric Acid (Female) normal value (0.6-6 mg/dl)

The table7and graph 7 shows that with increase in the serum uric acid levels there is decline in the MCI.

Discussion

This study was conducted in patients with chronic renal failure to document the osteodystrophy by measuring metacarpocortical index (MCI) of second metacarpal bone of right hand and to correlate the MCI index with serum levels of creatinine, urea, calcium, phosphorus, alkaline phosphatase, uric acid.

MCI was calculated from 50 persons (control group) both male and female and this mean MCI index was taken as reference and compared with MCI calculated in study group (CRF).

The results showing the MCI value in the study group (CRF) (0.38766) is declined in comparison with control group (0.575), indicating bone changes.

The results showed that as the serum creatinine values increases a decline in the MCI was observed in the study group.

This study compared MCI in CRF patients with blood urea levels and found the MCI was decreased in proportional raise in the levels of blood urea.

This study compared MCI in CRF patients with serum calcium levels and found the MCI was increased in proportional elevated levels of serum calcium.

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This study compared MCI in CRF patients with serum phosphorus levels and found the MCI was decreased with elevated levels of serum phosphorus.

This study compared MCI in CRF patients with serum alkaline phosphatase levels and found the MCI was decline with elevated levels of serum alkaline phosphatase.

This study compared MCI in CRF patients with serum uric acid levels and found that as the uric acid levels elevated there is decline in the MCI is observed.

Conclusion

This study concludes that

1) Quantitative bone changes in chronic renal failure patients can be measured by calculating metacarpocortical index from second metacarpal bone of right hand by the X-ray technique which is an effective method.
2) Comparison between MCI in CRF patients with biochemical parameters like serum creatinine, urea, calcium, phosphorus, alkaline phosphatase, uric acid concluded that MCI has been found to be decline with elevated levels of serum creatinine, urea, phosphorus, alkaline phosphatase and uric acid levels and MCI has been in found to be increased with elevated serum calcium levels.
3) X-ray of right hand for calculating MCI from second metacarpal bone can predict quantitative bone changes which is useful in preventing complications of osteodystrophy (ex : fractures).
4) Quantitative bone changes occurring in CRF patients by measuring MCI can be useful in the management and treatment in CRF. Thus MCI, is a simple, reliable, noninvasive and accessible method in predicting renal osteodystrophy early in asymptomatic stage and helps in preventing grave complications of renal osteodystrophy by earlyintervention and treatment.

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