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REVIEW ARTICLE

RENAL PARENCHYMAL CALCIFICATON – A DIAGNOSTIC DILEMA

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ABSTRACT

Introduction: Nephrocalcinosis may be defined as deposition of calacium (in the form of calcium phosphate and calcium oxalate) in the substance of the kidney, a process that can impair the function of the kidney function. The disorder may be symmetric or, in anatomic disorders such as medullary sponge kidney, involve only a single kidney. Nephrocalcinosis is caused by a number of conditions including the excess excretion of calcium by the kidney, renal tubular acidosis, medullary sponge kidney, hypercalcemia, renal cortical necrosis, and tuberculosis. ***Case Details:*** A 50-year-old male presented with loin to groin pain for the last 2 months. It was not associated with fever, dysuria or hematuria. He has been suffering from hypertension and dyslipidemia for last 5 years. No other significant past history was found. Multiple USG, X-Ray KUBP, CECT KUB with urogram and Urinary PCR was performed, all of tests revealed same finding of having two calculi in left kidney. The patient complained about having repeated episode of nephrolithiasis in renal colic. Later with detailed study a renal biopsy was performed and findings of left renal parenchymal calcification was confirmed. ***Conclusion:*** Patients with medullary nephrocalcinosis may develop nephrolithiasis leading to repeated episodes of renal colic. Treatment of the underlying cause can prevent recurrent renal calculus formation and hence reduce the morbidity associated with the disease.

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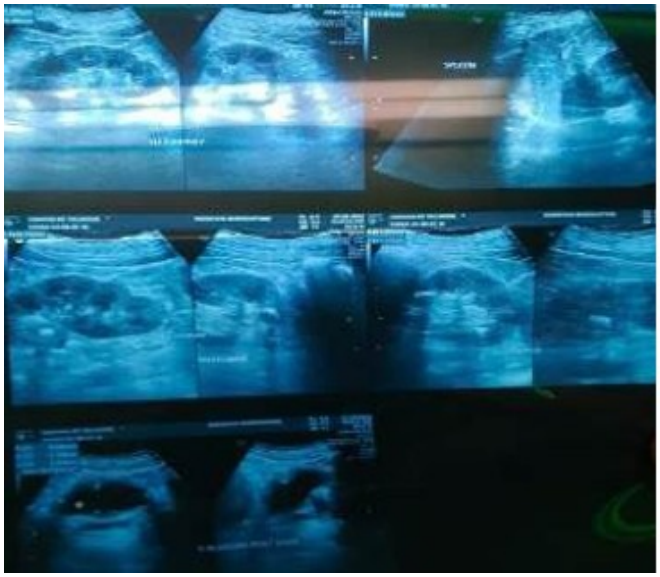
INTRODUCTION

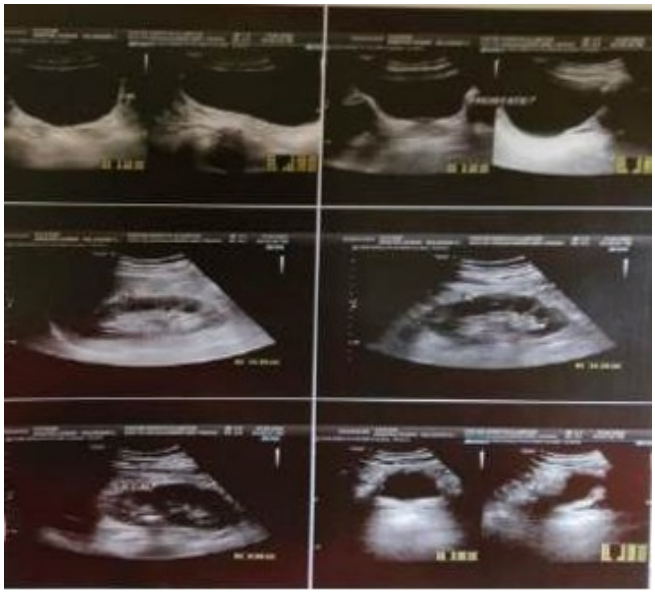
Renal parenchymal calcifications, or nephrocalcinosis, may occur as a result of specific lesions within the cortex or medulla or as a consequence of hypercalcemia or hypercalciuria. The calcifications may be more diffusely distributed in the renal cortex and medulla, including the papillae. A number of the diseases that cause nephrocalcinosis are also capable of causing renal calculi, notable among these is primary hyperparathyroidism. Other conditions associated with hypercalcemia, including secondary hyperparathyroidism, malignancy, and excessive calcium or vitamin D intake are also probable causative factors.

CASE PRESENTATION

A 50-year-old male presented with loin to groin pain for the last 2 months. It was not associated with fever, dysuria or hematuria. He has been suffering from hypertension (on Telmisartan 40mg OD) and dyslipidemia (on Tab Atorvastatin 20 OD) for last 5 years. No other significant past history was found. Multiple USG was done to detect the causative factor. In USG (Fig. 1), a non obstructive calculus (0.67 cm) was noted in mid calyx of left kidney. No hydronephrosis was detected. All other USGs done from reputed institutes corroborate with the similar findings. Ray KUBP was done showing presenced of radio-opaque calculi at the left region of left kidney. CECT KUB with urogram (Fig. 2), in this case, showed two calculi/cortical calcification in the left kidney. Small calculi was seen at the upper pole calyx of left kidney. Urinary PCR was done to rule out Genitourinary tuberculosis using Cepheid GeneXpert Systems (Gurgaon, India). Machines used for thyroid estimation used was *Seimens Advice centaur cp*, other biochemistry parameters were tested in *Randox RX imola* & serum electrolytes were measured in *ST-200 aqua electrolyte analyzer*.



(A)



(B)

Figure 1. (a) USG of left kidney showing multiple calculi,1.17cm in upper calyx & 0.71cm in lower mild calyx (b) USG of both kidneys showing few echogenic foci with shadowing in left kidney in upper &lower calyx, largest 9.9 mm in upper calyx

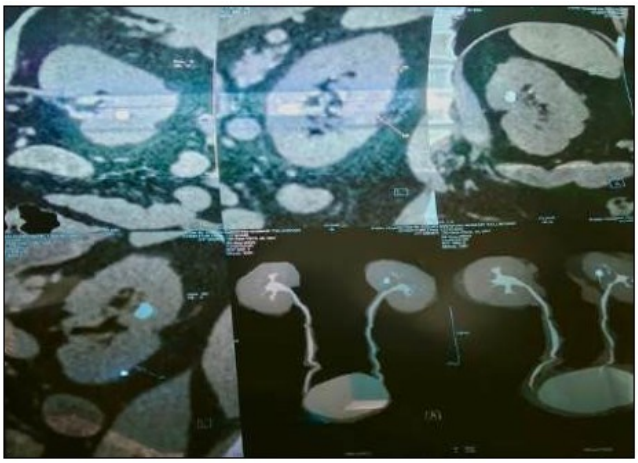


Figure 2. CECT KUB with Urogram showing two calculi/cortical calcification in the left kidney, small calculus upper pole calyx of left kidney, mild and generalised wall thickening of the urinary bladder.

Biochemical Parameters

Table 1. Biochemical parameters depicting an increased level of serum Calcium

Parameter	Measured values
Serum TSH	1.15 micro IU/ml
Uric acid	5.7 mg/dl
Serum Calcium	18mg/dl
Fasting Blood sugar (FBS)	86mg/dl
Postprandial Blood Sugar (PPBS)	136mg/dl
Prostate Specific Antigen (PSA)	0.477ng/ml
Serum sodium	137mEq/l
Serum potassium	4.5mEq/l
Creatinine	0.76mg/dl

Surgical Intervention: After diagnosis of renal calculi, the patient was taken for surgery. Cystoscopy + Retrograde Pyelogram+ Flexible Ureteroscopy + Ureteric catheterization was done under general anaesthesia. In left Retrograde Pyelogram, two radiopaque shadows, both out of PCS was noted, one in relation to upper calyx and another in relation to middle calyx, which did not corroborate with the features of renal calculi and no additional obstructive uropathy features were noted during the operative procedure. A 6-Fr U- Cath was placed. A 14-Fr Foley's Catheter was placed in the bladder and the patient was discharged in a haemodynamically stable condition with suggestive of renal parenchymal calcification. Serum PTH level assessed to diagnose it was a case of hyperparathyroidism. But serum PTH level was within normal limit (32.45pg/ml).

Patho-Physiology: The specific mechanism of nephrocalcinosis is complex and vary depending on the underlying cause. Some mechanisms include:

Tubule Damage: Calcium deposits within the tubules can damage tubular cells and impair their function. Interstitial Fibrosis: Calcium deposition in the interstitium can lead to scarring and fibrosis[1]. Reduced Renal Function: Nephrocalcinosis can impair the kidney's ability to filter blood and excrete waste products. Hypercalciuria: Increased calcium excretion in the urine can contribute to the formation of calcium deposits in the kidneys. Hypercalcemia: Elevated blood calcium levels can lead to calcium deposition in various tissues, including the kidneys. Genetic Factors: Some genetic disorders can predispose individuals to nephrocalcinosis[2].

Differential Diagnosis: Bilateral medullary nephrocalcinosis with left nephrolithiasis Cortical nephrocalcinosis. Renal tuberculosis Renal papillary necrosis. Primary/Secondary Hyperparathyroidism Malignancy (PTH-rP related)

DISCUSSION

Left renal parenchymal calcification is a pattern of renal injury characterized by abundant renal tubular and interstitial deposits of calcium phosphate. The calcium phosphate deposits are associated with acute tubular injury, as well as chronic, irreversible scarring leads to tubular atrophy and interstitial fibrosis. The finding of nephrocalcinosis on renal biopsy should prompt investigation into conditions associated with hypercalcemia, including hyperparathyroidism, malignancy, chronic renal disease and excessive calcium or vitamin D intake. Alternatively, nephrocalcinosis may result from

exposure to OSP (Oral Sodium Phosphate) bowel preparations prior to colonoscopy [3]. Cortical and medullary calcifications are not cleearly evident on pelvic x-rays, ultrasonographic studies and CT scans. Ultrasonogram is the preferred modality of choice for the evaluation of mild to moderate nephrocalcinosis [4]. However, in severe cases of nephrocalcinosis with repeated calculi formation and concern about the urinary tract as well, CT urography needs to be undertaken. The main concern with CT urography is the radiation dose that can best be avoided in mild to moderate cases by using the alternative modality of images, i.e, ultrasonography. On abdominal X-rays, cortical nephrocalcinosis may appear as thin peripheral band of calcification or diffuse punctate calcifications. Medullary calcification on the other hand is central in location and spares the cortex. It is usually bilateral with stippled calcification . Ultrasonographic study shows diffusely echogenic renal medulla without posterior shadowing [5]. CT scan shows calcific deposits along the renal medulla that are usually bilateral and symmetrical [6]. Nephrolithiasis is a common complication of nephrocalcinosis and should always be looked for. They can be in the pelviclyceal system, in the ureter or in the urinary bladder. Associated hydronephrotic changes might also be present but here no such finding.

CONCLUSION

Medullary nephrocalcinosis is a form of intrarenal calcification that occurs in the medulla and commonly involves bilateral kidneys. Patients with medullary nephrocalcinosis can develop nephrolithiasis leading to repeated episodes of renal colic. Treatment of the underlying cause can prevent recurrent renal calculus formation and hence reduce the morbidity associated with the disease

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