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Rare Case of Cortical Nephrocalcinosis In Post Pulmonary Tuberculosis.

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ABSTRACT

Nephrocalcinosis is a rare condition that consists of deposition of calcium salts in the renal parenchyma. It is of three types - cortical nephrocalcinosis when it involves the cortex, medullary nephrocalcinosis when it involves the medulla and is considered the mixed form when it involves both the renal cortex and medulla. The main etiological agents of this condition are primary hyperparathyroidism, renal tubular acidosis, medullary sponge kidney, hyperoxaluria and a few drugs. These are the factors that lead to hypercalcemia and/or hypercalciuria, which can further give rise to nephrocalcinosis. A 71 year-old woman, who was a known case of pulmonary tuberculosis (completed anti tuberculosis treatment), presented with urge incompetence, constipation and bilateral pedal edema for past three months On routine radiological examination, she was found to have bilateral cortical nephrocalcinosis. Nephrocalcinosis may be detected incidentally through diagnostic imaging studies without any presenting symptoms. Although there are various causes for nephrocalcinosis, post tuberculous sequelae, although rare should also be considered.

Keywords: Chronic kidney disease, Kidney, nephrocalcinosis

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INTRODUCTION

Nephrocalcinosis is a condition where there is increase in the concentration of calcium and the deposition of calcium salts in the renal parenchyma [1]. The main etiological agents of this condition are primary hyperparathyroidism, renal tubular acidosis, medullary sponge kidney, idiopathic hypercalciuria, hyperoxaluria (primary, enteric or toxic), secondary hypercalcemia (sarcoidosis, neoplasm and osteo- porosis) and drugs (including furosemide, acetazolamide and amphotericin B) [2].

In the early phase, this condition has no clinical symptoms. Only in the background of nephrolithiasis (renal stone) and ureteral colic, patient presents with symptoms of loin pain and hematuria (microscopic / macroscopic). The first symptom is increased thirst or polyuria which is due to the enhanced concentrating defect in the renal tubules. The complication of this condition if untreated is kidney failure, and the prognosis depends on the causative cause. Laboratory examinations may reveal erythrocytosis, and microscopic pyuria in the urinary sediment is nearly a constant, even in the absence of urinary infection, causing a chronic inflammatory response stemming from calcification [2,3].

Nephrocalcinosis can be detected by diagnostic imaging examinations (radiography, ultrasound and computed tomography) [1,4] which reveal calcium deposits in the renal parenchyma suggesting irreversible lesions accompanied by a variable degree of compromised kidney function [5]. The literature and documented case reports on familial mixed nephrocalcinosis is limited.

In this report we describe a case of cortical nephrocalcinosis in a post menopausal woman, without any significant history of kidney dysfunction.

CASE PRESENTATION

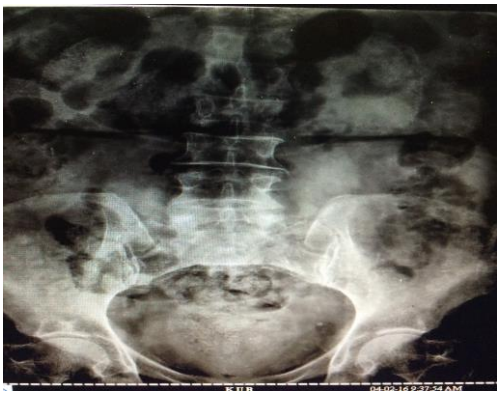
A 71 year old woman of Asian origin presented to the general medicine department with history of urge incompetence, constipation and bilateral pedal edema for past three months. She was not a known case of hypertension, diabetes, bronchial asthma. Her blood pressure was 110/80 mmHg, random blood glucose was 98 gm/dl and oxygen saturation was 100%. She gave a previous history of pulmonary tuberculosis 20 years ago and had completed anti tuberculous therapy. Her ESR and CRP levels, liver function tests and renal parameters were within normal limits (Urea - 35 mg/dl, creatinine 0.9 mg/dl). Her serum calcium level was 8.1 mg/dl and urinary oxalate excretion was 34 mg/day. Her physical examination was uneventful. She was then referred to the radiological department for abdominal radiograph.

RADIOLOGICAL FINDINGS

Radiograph abdomen

Renal outline of both the kidneys was well made out. Multiple peripheral egg shell type of calcification involving bilateral kidneys was seen.

Fig 1: Radiograph abdomen



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Ultrasound abdomen

Both the kidneys appeared normal in size with the right kidney measuring 9.5 x 4.2cm. and the left kidney measuring 9.9 x 4.6 cm. Both the kidneys showed a significant increase in cortical echogenicity, including both septal and basal cortex. Medulla appeared relatively unaffected. Both the pelvicalyceal systems appeared normal. Urinary bladder appeared normal.

Fig 2: Ultrasound abdomen showing right kidney

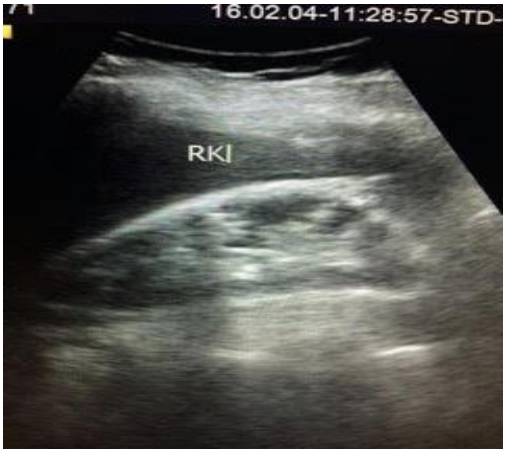
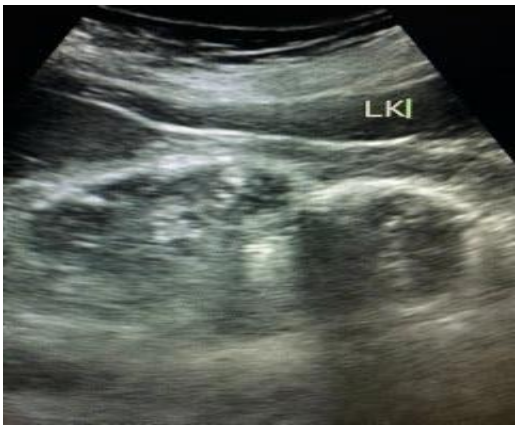


Fig 3: Ultrasound abdomen showing left kidney



CT abdomen

Fig 4: CT abdomen coronal view

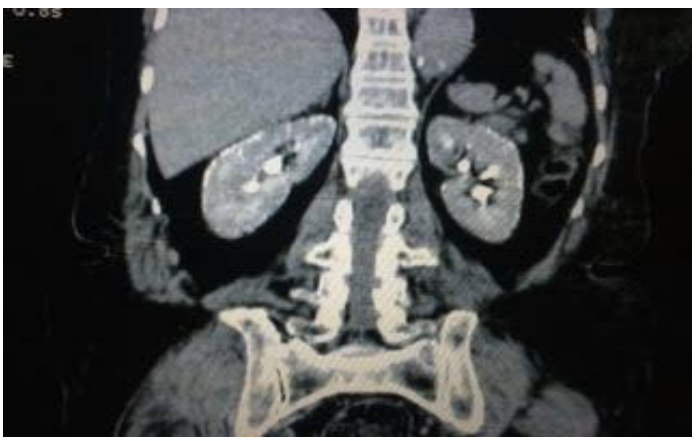


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Fig 5: CT abdomen axial view

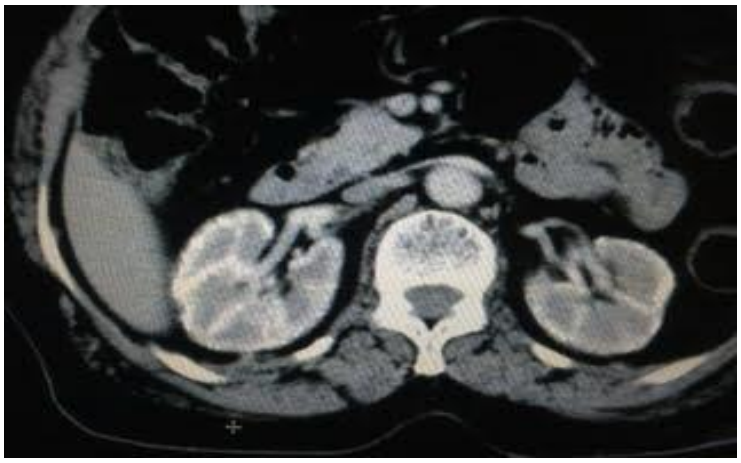
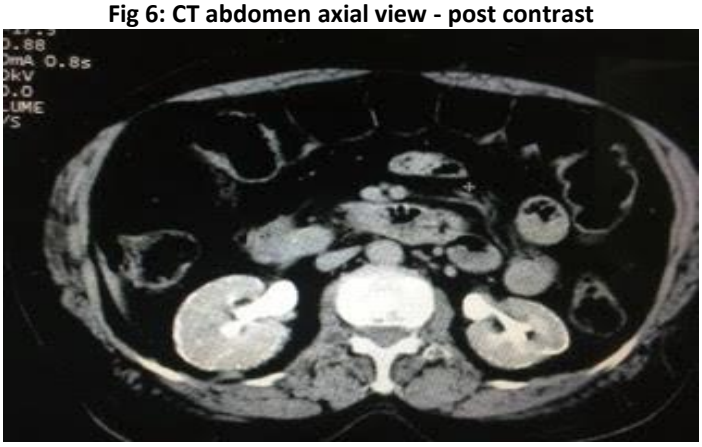


Fig 6: CT abdomen axial view - post contrast



CT abdomen revealed calcification noted along bilateral renal cortex. Renal pyramids were spared. There was no dilatation of pelvicalyceal system or proximal ureters. Post contrast examination revealed normal opacification of the renal pyramids and ureters, with contact filling into the urinary bladder in the delayed phase.

Chest Radiograph

Chest radiograph appeared normal for age with mildly prominent descending thoracic aorta and aortic knuckle calcification. Bilateral lung fields appeared normal.

DISCUSSION

Nephrocalcinosis is a condition that consists of deposition of calcium salts within the renal parenchyma. It is most commonly misdiagnosed as renal calculi but differs from the latterwhere there is formation of calculi in the excretory tract. Progression of kidney damage depends on the three phases of sequential increase in calcium content in the kidneys, namely chemical, microscopic and macroscopic.

Macroscopic nephrocalcinosis is classified as cortical (3% of cases), medullary (97% to 98% of cases) or mixed (involving the renal cortex and medulla) [2]. Among these, cortical nephrocalcinosis (as is our case) remains relatively rare, with only a few documented cases in literature. The mixed variant is an even rarer condition and may be seen in primary oxalosis or atypical infection by Mycobacterium avium-intracellulare in patients with AIDS [2].

Childhood nephrocalcinosis occurs in the medullary form in most cases and can progress to chronic kidney failure with a need for dialysis during childhood or early adulthood [6].

Nephrocalcinosis can be detected using imaging examinations such as radiography, ultrasound or computed tomography [2,3]. However computed tomography is ideally more superior to the other two modalitiesin differentiating cortical and medullary nephrocalcinosis. Contrast is generally not required. Computed tomography also demonstrates the relationship between calcifications in the parenchyma and the calyceal system, allowing the precise localization of the calcifications [5]. In our presently reported case, imaging examinations including plain radiograph, trans abdominal ultrasound and computed radiography confirmed that the patient had an advanced stage of bilateral cortical nephrocalcinosis, and owing to the previous history of pulmonary tuberculosis, cortical nephrocalcinosis due to post tuberculous sequelae was considered as the final diagnosis.

MANAGEMENT

For patients with nephrocalcinosis presenting with kidney stones, the conservative management is ingestion of plenty of fluids to increase the urine output. Patients are also advised to reduce sodium intake in food and limit it to <100 mEq/day. Measures are also taken to reduce calciuria and increase inhibitors of urinary crystallization [3-5]. However, the above measures were not possible in this case, as there were no demonstrable renal calculi. In general, the management of choice for nephrocalcinosis should be to treat the underlying causative factor.

CONCLUSION

In our presently reported case, imaging examinations including plain radiograph, trans abdominal ultrasound and computed radiography confirmed that the patient had an advanced stage of bilateral renal cortical nephrocalcinosis and owing to the previous history of pulmonary tuberculosis, cortical nephrocalcinosis due to post tuberculous sequelae was considered as the final diagnosis.

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