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Research Article

HYPERPARATHYROIDISM PRESENTING AS NEPHROCALCINOSIS-A RARE PRESENTATION

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A R T I C L E I N F O

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A B S T R A C T

Primary hyperparathyroidism can be associated with renal impairment such as Nephrolithiasis , Nephrocalcinosis. Hypercalciuria is one of the reason involved in the formation of renal stones.PHPT is associated with decline in renal function however the presentation is asymptomatic. The prevalence of renal manifestations in PHPT is rare, the most commonest being Nephrolithiasis. Other manifestations include Nephrocalcinosis, chronic renal insufficiency, renal tubular dysfunction. Here we report a rare case of PHPT which presented as Nephrocalcinosis with decline in renal function.

INTRODUCTION

PHPT is a common endocrine disorder due to inappropriate PTH release from tumour of parathyroid glands .Prevalence of renal disease in PHPT has changed significantly over years, presenting asymptomatically, nephrolithiasis being the most commonest presentation (25-50%), Nephrocalcinosis (2-3%). With the advent of routine calcium screening and imaging modalities, early diagnosis of renal stones in PHPT has been increased in the last two decades. The presence of renal stones (symptomatic or asymptomatic) categorises PHPT as an indication for parathyroid adenomectomy. Renal calcifications (kidney stones ,nephrocalcinosis were associated with higher calcium/creatinine excretion rate .The risk of progression of nephrocalcinosis is reversible after successful surgery, but the residual risk persists. However Annual renal function is advised for all patients with mild asymptomatic PHPT.

CASE REPORT

A 45 year old female patient presented to the causality at Sree Balaji medical college and hospital, general medicine department with complaints of reduced urine output for the past one week, burning micturition present, associated with lower abdominal pain, dragging type of pain on and off , no aggravating on passing urine with no relieving factors.The patient also complaints generalised body pains, nausea for the past 6 months, history of bilateral knee, elbow, ankle pain for the past 1 month , headache for 1week increased in frequency and intensity, history of early morning periorbital puffiness present .There is no history of fever, cough, sore throat. The patient gives history of significant weight loss 5 kgs in the past two months. No history of vomiting, loose stools. The patient is known case of hypothyroidism for the last 10 years on regular medications, no other known co-morbidities. On examination the patient is conscious, oriented, afebrile. Vitals are stable. On general physical examination, patient is dull looking, pallor is present, bilateral pitting pedal edema present. Systemic examination-normal.

The patient was admitted with the above mentioned complaints all routine baseline investigations done. In view of reduced urine output with abdominal pain, urine routine and renal function tests done. Reports showed plenty of pus cells with no proteinuria in urine routine and deranged RFT, mentioned in table below. In view of suspected chronic kidney disease, ultrasonography of abdomen was done to look for kidney size and corticomedullary junction, along with serum iPTH, iron studies to rule out metabolic bone disease and anaemia of chronic disease in CKD. Reports mentioned in table below. Serum iPTH was markedly elevated value of 2292pg/ml. USG abdomen was done reports which showed right renal exophytic cyst, increased echogenicity of bilateral renal pyramids-features suggestive of bilateral nephrocalcinosis (image1) and kidney size appears to be normal in size. Serum calcium levels were done I/v/o suspected hyperparathyroidism reports showed presence of hypercalcemia 15.8mg/dl, serum phosphorus 2.0mg/dl. Based on the above mentioned investigations and imaging the diagnosis is primary hyperparathyroidism. CT abdomen was done reports showed bilateral symmetrical nehrocalcinosis, diffuse osteopenic and osteomalacia changes in vertebral and bilateral hip bones, rugger jersey appearance of dorsolumbar

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vertebra, expansile lytic lesion in D8 vertebral body. Endocrinologist opinion was obtained for PHPT and advised hypocalcemia treatment with IV- fluids and inj. calcitonin 200mg TDS and additional imaging test sestamibi scan to localise the adenoma and X-ray spine to rule out brown's tumour (for treatment and regular follow up)

All the above mentioned investigations and treatment was given and followed up for the patient. Sestambi scan revealed right superior parathyroid adenoma and the presence of brown's tumour on X-ray spine. Hypocalcemia correction was done and the patient was taken up for right superior parathyroidectomy and was sent for Histopathological examination. Biopsy reports showed parathyroid carcinoma.

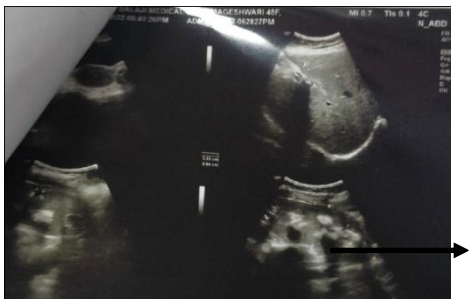

Image1 Bilateral renal nephrocalcinosis

This case report of a 45year female, who came with the above mentioned complaints initially was thought to have CKD , but diagnosed with primary hyperparathyroidism presenting as nephrocalcinosis with cause being parathyroid carcinoma .which is a rare presentation.

RFT / S.electrolytes	Urine routine	Additional tests
Urea -31	Pus cells-18-20	iPTH-2292
Creatinine-1.4	RBC 2-3	(normal-15-65pg/ml)
Uric acid-7.1	Urine sugar - nil	
		Phosphorus - 2.0(normal value 2.8-4.5mg/ml)
		Serum calcium-15.8mg/dl(normal value 8.5-10.2mg/dl)
S.sodium-136.9		24H Urine Protein-1395
S.potassium-3.32		Total volume - 4360
S.Chloride-103		
		TSH-5.93
		ANA- weakly positive

DISCUSSION

Nephrocalcinosis and nephrolithiasis are the most common kidney manifestation of PHPT. The reported incidence of nephrocalcinosis among patients with PHPT is between (16 and 22%). Nephrocalcinosis is subclinical kidney disease in PHPT other diseases include chronic kidney insufficiency, with asymptomatic PHPT have an estimated eGFR below 60ml/min/1.73m2. a contributing factor for stone formation and nephrocalcinosis is hypercalciuria. Although PTH directly stimulates distal tubular reabsorption of calcium this effect is overshadowed by the increase in filtered calcium due to hypercalcemia leading to increased urine calcium excretion in 30-40% patient with PHPT. Some of the common renal manifestation in PHPT are decreased GFR, impaired urinary concentrating ability sometimes leading to polyuria, reduced fractional phosphate reabsorption leading to hypophosphatemia, increased urinary excretion of magnesium.

CONCLUSION

Nephrocalcinosis is characterised by the generalised deposition of calcium, phosphate or calcium oxalate in the kidney (medulla or cortex). Patients who have neohrocalcinosis may have acute or chronic kidney injury or may sometime have normal kidney function. Nephrocalcinosis is almost always incidentally detected by imaging studies that are obtained for reasons unrelated to kidney. The prognosis of nephrocalcinosis depends on the underlying cause, while most patients do not progress to end stage renal disease. Nephrocalcinosis can be detected by plain film, USG and CT imaging. The underlying cause of nephrocalcinosis should be determined and treated if possible since the kidney prognosis is determined by the underlying cause. No specific treatment is available to stop the progression of nephrocalcinosis.

References

1. Lila AR, Sarathi V, Jagtap V, Bandgar T, Menon PS, Shah NS. Renal manifestations of primary hyperparathyroidism. Indian J Endocrinol Metab. 2012 Mar; 16(2):258-62. doi: 10.4103/2230-8210.93745. PMID: 22470864; PMCID: PMC3313745.
2. Anurag Ranjan; Sarathi, Vijaya; Jagtap, Varsha; Bandgar, Tushar; Menon, Padma S.; Shah, Nalini Samir. Renal manifestations of primary hyperparathyroidism. Indian Journal of Endocrinology and Metabolism 16(2):p 258-262, Mar–Apr 2012. | DOI: 10.4103/2230-8210.93745
3. 3.Lars Rejnmark, Peter Vestergaard, Leif Mosekilde, Nephrolithiasis and Renal Calcifications in Primary Hyperparathyroidism, The Journal of Clinical Endocrinology & Metabolism, Volume 96, Issue 8, 1 August 2011, Pages 2377–2385,
4. Lo TE, Tan IT. Distal renal tubular acidosis in primary hyperparathyroidism. BMJ Case Rep. 2015 Jan 29; 2015:bcr2014208120. doi: 10.1136/bcr-2014-208120. PMID: 25634859; PMCID: PMC4322272.
5. Vazquez AM. Nephrocalcinosis and Hypertension in Juvenile Primary Hyperparathyroidism. Am J Dis Child. 1973; 125(1):104–106.doi:10.1001/archpedi.1973.04160010 070016

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