International Journal of Advances in Medicine
Sathyan S et al. Int J Adv Med. 2015 Nov;2(4):423-425
http://www.ijmedicine.com
pISSN 2349-3925 | eISSN 2349-3933
DOI: http://dx.doi.org/10.18203/2349-3933.ijam20151023

Case Report

Hypothyroidism: a reversible cause of 'acute kidney injury'- a series of 5 cases

Swaraj Sathyan[1], Sunil George[1], Poornima Vijayan[2]*

[1]Department of Nephrology, MES Medical College, Malaparamba, Palachode, Malappuram-679338, Kerala, India
[2]Department of Pathology, MES Medical College, Malaparamba, Palachode, Malappuram-679338, Kerala, India

Received: 02 September 2015
Accepted: 05 October 2015

***Correspondence:**
Dr. Poornima Vijayan,
E-mail: poornima_vij@yahoo.co.in

ABSTRACT

The effects of thyroid hormones on the kidneys are well known. Thyroid dysfunction causes remarkable changes in glomerular and tubular functions and electrolyte and water homeostasis. There have been reports of elevated serum creatinine associated with hypothyroidism, the pathogenesis of which is thought to be multi-factorial. We present five cases, all of who were referred to the nephrology department for evaluation of unexplained 'acute kidney injury'. Hypothyroidism was suspected based on history and clinical examination. In all cases, investigation revealed significantly elevated TSH, low T3, T4 levels, elevated CPK levels, normal urine analysis and normal sonological study of kidney. All the cases showed complete normalization of renal parameters after 4-8 weeks of treatment with levothyroxine. This case series depicts the need for high index of suspicion for diagnosis of hypothyroidism in the setting of unexplained 'acute kidney injury'.

Keywords: Serum creatinine, Hypothyroidism, Unexplained acute kidney injury

INTRODUCTION

Thyroid hormones (TH) are necessary for growth and development of the kidney and for the maintenance of water and electrolyte homeostasis. The kidney is not only an organ for metabolism and elimination of TH, but also a target organ of action of iodothyronines. Thyroid dysfunction causes remarkable changes in glomerular and tubular functions and electrolyte and water homeostasis. This decline of kidney function is in turn accompanied by changes in the synthesis, secretion, metabolism, and elimination of thyroid hormones.[1] Thus the interactions between thyroid and the kidneys have been well documented and understood.

Both hypothyroidism and hyperthyroidism affect renal blood flow, GFR (glomerular filtration rate), tubular function, electrolytes homeostasis, electrolyte pump functions, and kidney structure. These are the result of direct renal effects, as well as systemic hemodynamic, metabolic, and cardiovascular effects. And fortunately, most of these renal manifestations of thyroid disorders, especially those associated with hypothyroidism, are readily reversible with treatment.[2] Awareness of this unusual presentation of hypothyroidism is of great significance because prompt diagnosis and timely correction of hypothyroidism can almost completely reverse the GFR and RFT (renal function tests). We present a series of five cases of acute kidney injury associated with hypothyroidism, all of whom showed marked improvement in serum creatinine and CK (creatine kinase) levels after 4-6 weeks of treatment with levothyroxine.

CASE REPORT

All five of our patients were referred to the nephrology OPD with provisional diagnoses of unexplained acute

kidney injury (AKI). On detailed history talking, all our cases gave history of symptoms suggestive of moderate to severe hypothyroidism, such as cold intolerance, constipation, muscle weakness, and lower extremity edema (Table 1). Initial serum creatinine levels ranged between 1.6 and 2.5 mg/dL, e-GFR ranging from 31 to 57 and blood urea ranging from 23 to 30. Urine routine and ultrasound abdomen did not reveal any significant abnormalities. TSH was markedly elevated in all patients ranging between 78 and 353 and T3 and T4 were decreased. CK levels were also markedly elevated ranging between 1200 and 9500 (Table 2). Urinary casts and urinary myoglobin were absent in all patients. Since there was no evidence of a primary renal disease, renal biopsy was deferred and all patients were started on appropriate doses of levothyroxine.

All patients were reviewed after 4weeks and 8 weeks. Serum creatinine, e-GFR, TSH and CK levels were monitored at each visit. At the two month follow up, all patients showed normalization of serum creatinine, blood urea, e-GFR, TSH and CK levels (Table 3).

Table 1: Clinical details of four cases of unexplained acute kidney injury.

Case No.	Age/sex	Signs & symptoms	Co-morbidities	Blood pressure (mmHg)	Ultrasound abdomen and pelvis
1.	45/m	Facial puffiness, fatiguability, constipation	Nil	120/80	Normal study
2.	45/m	Facial puffiness, cold intolerance, easy fatiguability, no urinary complaints	Nil	110/70	Normal study
3.	31/m	Facial puffiness, easy fatiguability, proximal muscle weakness	Nil	130/80	Normal study
4.	47/m	Facial puffiness, easy fatiguability, cold intolerance, proximal muscle weakness	Nil	130/80	Normal study
5	31/m	Facial puffiness, hoarseness of voice, goiter, proximal muscle weakness	Nil	120/80	Normal study

Table 2: Investigations done at first OPD visit.

Case No.	Urine routine	B. Urea (mg/dl)	S. creatinine (mg/dl)	eGFR (ml/min/1.73m^2)	TSH (0.3-5.5)	T3 (60-200)	T4 (4.5-12)	CK (55-170)
1.	Trace proteins absent sediment / casts	30	2.4	31	128	29.8	0.523	2450
2.	Within normal limits	34	1.8	44	83.7	30.4	0.557	2707
3.	Within normal limits	23	1.7	53	126	27.6	0.867	9592
4.	Within normal limits	26	2.5	29	353	32.8	1.14	6890
5.	Within normal limits	29	1.6	57	78	45.6	1.09	1203

B. Urea-blood urea, eGFR-estimated glomerular filtration rate, TSH-Thyroid stimulating hormone, CK-Creatine kinase

Table 3: Investigations done at 4 weeks and 8 week follow up.

Case No.	B. Urea (mg/dl)		S. creat (mg/dl)		eGFR (ml/min/1.73m^2)		TSH (0.3-5.5)		CK (55-170)	
	4 weeks	8 weeks	4 weeks	8 weeks	4 weeks	8 weeks	4 weeks	8 weeks	4 weeks	8 weeks
1.	28	24	1.2	1	73	90	29	0.628	528	180
2.	28	23	1.2	0.9	73	103	57.3	1.04	468	154
3.	22	22	1.3	1	74	100	33.4	1.32	1249	202
4.	27	24	1.7	1.1	47	80	44.1	1.97	854	130
5.	27	27	1.1	0.8	89	119	35.3	1.54	359	125

S. creat-Serum creatinine, eGFR-estimated glomerular filtration rate, TSH-Thyroid stimulating hormone, CK-Creatine kinase

DISCUSSION

The effects of thyroid hormone on kidneys are well known. There are various reports of acute kidney associated with hypothyroidism. The exact mechanism of acute kidney injury due to hypothyroidism is still unclear, but many explanations have been put forth. Hypodynamic circulation causing a reduction in renal plasma flow and GFR is thought to be the predominant mechanism causing acute kidney injury. Reduced cardiac output and low plasma volume contributes to the pre-renal status of hypothyroidism. Rhabdomyolysis is another extremely rare but well known complication of hypothyroidism which may cause acute kidney injury, but it is usually precipitated by trauma or certain drugs.[3] Elevation of serum creatinine levels can also occur without a decrease in actual GFR. In hypothyroidism, myopathy can result in release of creatinine from the injured muscles, which should be suspected especially in the background of disproportionate elevation of serum creatinine without significant elevation of blood urea and high CPK levels.[4] Elevation of serum creatinine can occur as early as 2 weeks of onset of significant hypothyroidism and usually recovers fully with thyroxine replacement, but may be slower and incomplete if prolonged and severe. Elevated serum creatinine due to hypothyroidism is usually in the range of 1.5-2.5 mg/dl, though serum creatinine above 6 mg/dl has been documented.[2] Increase in serum creatinine was observed in more than half of adults with hypothyroidism in some studies,[1,5] with some reporting association with even subclinical hypothyroidism.[6]

In our case series, all the patients presented with varying degrees of elevated serum creatinine, with blood urea in the normal range and high CPK levels with normal urinalysis and ultrasonogram of kidneys. Even though multiple factors would have contributed, most probable cause would have been release of creatinine from injured muscles in the background of pre-renal state. Absence of urinary casts, urine myoglobin and no other features of intrinsic renal involvement makes rhabdomyolysis unlikely.

CONCLUSION

In conclusion, this case series highlights the necessity for a high index of clinical suspicion in patients with unexplained isolated elevation of serum creatinine especially in the absence of abnormal urinalysis or ultrasonography of kidneys to diagnose hypothyroidism, because its prompt correction by thyroxine supplementation can lead to complete normalization of serum creatinine and CK levels.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Sathyan S, George S, Vijayan P. Hypothyroidism: a reversible cause of 'acute kidney injury'- a series of 5 cases. Int J Adv Med 2015;2:423-5.