

# Acute kidney injury secondary to hypothyroidism induced rhabdomyolysis: a case report

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### ABSTRACT

*Renal impairment is occasionally reported in association with hypothyroidism. We report a case of a middle aged man, who presented with features of rhabdomyolysis and acute kidney injury which was ascribed to underlying undiagnosed hypothyroidism. The etiology is presumed to be multifactorial; hemodynamic effects and a direct effect of thyroid hormone on the kidney play important role. This case enlivens the necessity of assessing thyroid function in cases of unexplained renal failure. Awareness about this rare presentation of a common disease would alert the physician to effectively treat this dreaded yet reversible complication.*

**Key words:** Acute kidney injury, hypothyroidism, rhabdomyolysis.

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### INTRODUCTION

Acute kidney injury (AKI) is common in clinical practice with diverse etiology. Severe hypothyroidism has been reported as being associated with renal dysfunction and thyroid hormone replacement may lead to an improvement in renal function.<sup>1</sup> In hypothyroidism, muscle involvement is common. However, rhabdomyolysis and consequent AKI due to

overt, untreated hypothyroidism, especially in the absence of other identifiable causes are rare and only a few cases have been reported.<sup>2</sup> This atypical presentation assumes great clinical significance, as prompt identification and correction of underlying hypothyroidism almost completely reverse renal injury. If untreated, AKI may lead to potential adverse consequences.<sup>3</sup> We report a case of severe hypothyroidism leading to rhabdomyolysis and AKI without any additional precipitating factor.

### CASE REPORT

A 52-year-old male patient, presented with a one-month history of worsening edema, accompanied by breathlessness for 7 days and oliguria for 5 days. His medical history included hypertension and diabetes mellitus. His medication consisted of short-acting insulin and losartan potassium. The man was obese, edematous and had a puffy face and anemia. His pulse was 52 beats/min and blood pressure was 140/90 mm Hg. Precordium examination findings were unremarkable. He had features of ascites and bilateral pleural effusion. There was delayed relaxation of ankle jerks and bilateral non-proliferative diabetic retinopathy (NPDR). Bed side urine revealed proteinuria (++).

Laboratory evaluation showed low hemoglobin (8.3 gm/dl), deranged renal function (blood urea 90 mg/dl, serum creatinine 2.2 mg/dl), low serum sodium (125 mmol/L)

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and albumin (29.8 gm/L), markedly raised serum creatine phosphokinase (CPK) (1128 U/L, ref. 24-195) and slight elevation of aspartate aminotransferase (131 IU/L). Thyroid function tests confirmed the diagnosis of hypothyroidism [freeT4 6.77 pmol/l, ref. 9.14-23.18 and thyroid-stimulating hormone (TSH) 150 mIU/ml, ref.0.47-5.01, anti-thyroglobulin antibody 34.9 IU/ml, anti-thyroid peroxidase antibody 1300 IU/ml]. Urine analysis showed proteinuria and pyuria. The urinary total protein was 1.18 g/24-h. Ultrasonography showed bilateral moderate pleural effusion and moderate ascites. Electrocardiogram (ECG) showed sinus bradycardia. A chest x-ray postero-anterior view revealed mild cardiomegaly and bilateral pleural effusion. A diagnosis of AKI secondary to hypothyroidism induced rhabdomyolysis was made.

After admission, losartan potassium was stopped. After having the diagnosis of hypothyroidism, levothyroxine was started. After 10 days of hospital stay, his renal function started to improve and his urine output also increased. The patient received one unit of red cell concentrate transfusion during the hospital stay. His medication at discharge included split mix regimen of insulin, levothyroxine 150 µgm daily and prazosin 1 mg once daily.

The patient came for follow up after 3 months; he had no edema, his serum creatinine was 2.4 mg/dl (was 4.5 mg/dl during discharge), TSH level was 5.2 mIU/ml.

## DISCUSSION

The present case describes a patient with hypothyroidism and AKI due to rhabdomyolysis with no additional precipitating factor. Hypothyroidism in older patients is most often primary and caused by an autoimmune disorder.<sup>4</sup> Hypothyroidism is often associated with renal involvement. These renal abnormalities occur because the deficiency of thyroid hormones causes a reduction in plasma flow and glomerular filtration rate due to the hypodynamic circulation. The hypodynamic circulatory state results in a pre-renal renal insufficiency and this may be aggravated by other multi-system effects of hypothyroidism such as reduced cardiac output, low volume status, hyponatremia with associated hemodynamic changes and increased peripheral resistance due to arterial wall stiffness.<sup>5</sup> Two more mechanisms that are thought to contribute to renal

dysfunction include tubular secretion of creatinine and creatinine release from muscle.<sup>6</sup> This results in an elevation in the serum levels of creatinine and a reduction in its clearance.<sup>7</sup> Primary hypothyroidism which is associated with a reduction of glomerular filtration and renal blood flow, that are normalized following levothyroxine administration.<sup>6,8</sup>

Myopathy is a common presentation of hypothyroidism and may be accompanied by mild to moderate CPK elevation, usually less than 10 times the upper limit of normal.<sup>9</sup> Although hypothyroid myopathy is usually limited to myalgias, very rarely which can complicate to rhabdomyolysis.<sup>10</sup> Rhabdomyolysis is characterized by the leakage of muscle cell contents, including electrolytes, myoglobin and other sarcoplasmic proteins such as CPK, aldolase, lactate dehydrogenase, transaminases into the circulation. Rhabdomyolysis can be diagnosed by greatly elevated CPK levels, muscle pain and weakness and/or detection of myoglobinuria. Myoglobin appears in the urine only when the renal threshold of 0.5 – 1.5 mg of myoglobin per decilitre is exceeded.<sup>11</sup> Rhabdomyolysis leading to AKI is a rare complication of hypothyroidism.<sup>5,9,11</sup>

The exact cause of rhabdomyolysis in hypothyroidism still remains unclear. A possible explanation may be a reversible defect in glycogenolysis and impaired mitochondrial oxidative metabolism due to low mitochondrial enzyme activity and an autoimmune mechanism could be possible.<sup>9</sup> AKI is a dreaded complication of rhabdomyolysis, if not promptly managed and can be seen in one-third of cases.<sup>11</sup> Dehydration and signs of volume depletion are commonly seen in rhabdomyolysis due to the sequestration of water in injured muscles and interstitium.<sup>11</sup> However, if dehydration is present, it can enhance progression to AKI in rhabdomyolysis.<sup>12</sup> Therefore, aggressive fluid replenishment forms the cornerstone in the management of rhabdomyolysis.<sup>11</sup> We suggest that patients with renal impairment of unknown causes have thyroid function tests undertaken as part of routine investigation.

**Authors' contribution:** MAIKU did literature search and drafted the manuscript. RKG managed the case. FK did literature search and helped drafting manuscript. IJ managed the case. TAC supervised managing the case. MAR supervised management, did literature search, revised the manuscript. MAA and SI supervised

managing the case. All authors read and approved the final manuscript for submission.

**Conflicts of interest:** Nothing to declare.

## REFERENCES

1. Rao NS, Chandra A, Malhotra KP. Severe hypothyroidism-associated acute renal failure—A case series from North India and review of literature. *Thyroid Research and Practice* 2019 Sep 1; 16(3): 121.
2. de Menezes NPD, Bridi RA, Balbi AL, Ponce D. Hypothyroidism and acute kidney injury: an unusual association. *Case Reports* 2013 Aug 9; 2013:bcr2013200585.
3. Mangaraj S, Sethy G, Sen RK, Rout RN. Hypothyroidism presenting as acute kidney injury. *J Health Specialties* 2014 Jan 1; 2(1): 28.
4. Habra M, Sarlis NJ. Thyroid and aging. *Rev Endocr Metab Disord* 2005; 6(2): 145-54.
5. Liakopoulos V, Dovas S, Simopoulou T, Zarogiannis S, Giannopoulou M, Kourti P, et al. Acute renal failure: A rare presentation of hypothyroidism. *Ren Fail* 2009; 31: 323-6.
6. den Hollander JG, Wulkan RW, Mantel MJ, Berghout A. Correlation between severity of thyroid dysfunction and renal function. *Clin Endocrinol (Oxf)* 2005; 62: 423-7.
7. Mahjoub S, Ben Dhia N, Achour A. Hypothyroïdie primitive et atteinte glomérulaire. [Primary hypothyroidism and glomerular involvement]. *Ann Endocrinol (Paris)* 1991; 52(4): 289-92.
8. Montenegro J, González O, Saracho R, Aguirre R, González O, Martínez I. Changes in renal function in primary hypothyroidism. *Am J Kidney Dis* 1996; 27(2):195-8.
9. Barahona MJ, Mauri A, Sucunza N, Paredes R, Wägner AM. Hypothyroidism as a cause of rhabdomyolysis. *Endocr J* 2002; 49: 621-3.
10. Altay M, Duranay M, Ceri M. Rhabdomyolysis due to hypothyroidism. *Nephrol Dial Transplant* 2005; 20: 847-8.
11. Bosch X, Poch E, Grau JM. Rhabdomyolysis and acute kidney injury. *N Engl J Med* 2009; 361: 62-72.
12. Vanholder R, Sever MS, Ereke E, Lameire N. Rhabdomyolysis. *J Am Soc Nephrol* 2000; 11: 1553-61.