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ACUTE RENAL FAILURE IN HYPOTHYROID PATIENT, CAUSE HADDOMYOLYSIS**N. S. SENGAR, NUTAN AGARWAL*, SAURABH GUPTA, KSHITIZ NATH
AND ASHISH SHARMA**Department of Medicine
M.L.B., Medical College
JHANSI 284128 (INDIA)
*Corresponding Author**Received : 12.2.15; Accepted : 23.3.15****ABSTRACT**

In our study, we are discussing about acute renal failure in five male patients of age group about 26-38 yrs. Patients presented with myalgia and signs and symptoms of acute renal failure. On further evaluation, all patients have higher value of serum TSH and elevated muscle enzyme CPK and increased Sr. creatinine. Immediate thyroid replacement therapy was given following which myalgia and Sr. creatinine improved. Hypothyroidism although rare, but has been reported as a cause of rhabdomyolysis. So, hypothyroidism must be considered in patient presenting with elevated muscle enzyme and acute renal failure.

Figure : 00

References : 14

Table : 01

KEY WORDS : Acute Renal Failure, CPK, Hypothyroidism, Rhabdomyolysis, TSH.

Introduction

Hypothyroidism is a systemic disorder with myopathic features including myalgia, muscle stiffness and cramp, and occasional moderately elevated levels of muscle enzymes¹. Overt rhabdomyolysis and renal failure have been reported in only few cases^{1, 7}. Rhabdomyolysis is an injury of skeletal muscle resulting in leakage of cell content into bloodstream. Although muscular disorders are usual in hypothyroidism, rhabdomyolysis due to hypothyroidism is very rare and only a few cases have been reported^{1,8,11}. Non-exertional and non-traumatic causes of rhabdomyolysis include drugs, toxins, infections, electrolyte abnormalities, endocrinopathies, inflammatory myopathies³. In literature, most of the cases of rhabdomyolysis due to hypothyroidism and acute renal failure (ARF) had additional precipitating factor⁷. We present five cases with ARF due to hypothyroidism associated rhabdomyolysis with no additional precipitating factor.

Case series

Case 1 : A 26 yrs old male presented with chief complaints of generalized body swelling and increased stiffness of body with severe myalgia and

both upper limb and lower limb weakness for 2-4 weeks. On further questioning he gave history of several months of vague constitutional symptoms included tiredness, lethargy, low mood and leg muscle cramping. There was no history of strenuous exercise, exposure to extreme heat, any drugs or toxins. There was no personal and family history suggestive of myopathies, diabetes or any other endocrinal disorder. On examination, he had periorbital and facial oedema and generalized non pitting oedema, stable vitals with normal general systemic examination, no sign suggesting systemic inflammatory disease; investigations are mentioned in Table 1. Patient was given oral levothyroxin and conservative supportive treatment for ARF. He showed marked recovery over 2-4 days and was discharged.

Case 2 : A 32 yrs male presented to us with generalized body swelling, dry skin, myalgia, muscle stiffness, decreased urine output for 15 days. His BP was 130/80 mmHg, pulse rate was 80/min, afebrile. CVS, CNS, Respiratory and abdominal examination were within normal limits. Patients TSH was 200µIU/ml. other investigation are in Table 1. Patient was started oral levothyroxine 100µg/day with other symptomatic treatment for

ARF. He showed marked improvement within two days. His urine output was increased and serum creatinine fell to normal limits. He was discharged after 4 days.

Case 3 : A 38 yrs old male was admitted to us with myalgia, increased body stiffness, lower limb weakness and decreased urine output for 7-8 days. He had no prior history of any medical illness. On examination his blood pressure was 132/84 mmHg and pulse rate was 76/min, he had periorbital puffiness and generalised nonpitting oedema. Systemic examination was within normal limits. Investigations are listed in Table 1. Patient was given intravenous fluids along with levothyroxine in incremental doses up to 100µg daily. Within few days the patient improved and creatinine returned to normal limits.

Case 4 : A 38 yrs old male presented to us with periorbital, facial and generalized leg swelling since three weeks. This was preceded by several months of vague constitutional symptoms including tiredness, lethargy, low mood and leg muscle cramping. He had no significant past medical or surgical history. There was no family history of renal or endocrine disorders. On clinical examination he was overweight (BMI 41) with marked peri-orbital edema and facial edema. He was a febrile, pulse of 74 bpm, blood pressure of 136/84mmHg, respiratory rate of 18 breaths/min and oxygen saturations of 100% on room air. Cardiovascular, respiratory, abdominal and neurological examinations were all unremarkable. On investigation TSH levels were elevated and creatinine was raised. Oral levothyroxin was started in gradually increasing doses upto 100µg. Patient recovered within 4-5 days.

Case 5 : A 36yrs old male presented to us with generalised body swelling, myalgia, generalised body weakness, dark brown urine and decreased urine output for 7-10 days. He was a known case of hypothyroidism but not taking any medication from 2 months. On examination his blood pressure was 134/82 mmHg, pulse rate 79/min, afebrile, had generalised nonpitting edema, periorbital puffiness, no pallor, no dyspnoea, JVP not raised, rest other examination was within normal limits. He was a chronic smoker, no any other addiction was found. On investigation, serum TSH and creatinine were raised and ABG showed mild metabolic acidosis. He was started oral levothyroxine and given intravenous fluids with other

supportive therapy. Patient got relieved in 4-5 days and was discharged with normal parameters.

Discussion

Although the exact cause of renal impairment in hypothyroidism is unclear but two mechanisms are hypothesised: 1). Thyroid hormone influence renal development, structure, hemodynamics, GFR, the function of transport system along the nephron and sodium and water homeostasis. Patient with hypothyroidism can have reduction in GFR due to decreased sensitivity to α -adrenergic stimulus and decreased rennin release alongwith decreased angiotensin II and impaired RAAS activity resulting in reversible renal failure in hypothyroid patient resulting in increased Sr. creatinine level. 2). Another cause is thyroid disorder with systemic feature such as myopathy, elevated levels of muscle enzymes causing rhabdomyolysis resulting in ARF.

Rhabdomyolysis was defined as creatine kinase levels above five times the upper limit of normal and renal findings in both cases. Although the main features of rhabdomyolysis are muscular symptoms and increased creatine kinase concentrations, it can become a life-threatening disorder when complicated by ARF. The exact cause of rhabdomyolysis (abnormal glycogenolysis, mitochondrial oxidative metabolism, and triglyceride turnover, impair muscle function) in hypothyroidism remains unclear⁶. As a cause of rhabdomyolysis, disorders such as collagen disease (e.g., polymyositis), ingestion of massive alcohol, other agents, infection and trauma were excluded in our cases from medical history. Although hypothyroidism is an uncommon cause of rhabdomyolysis causing acute renal failure, it is reversible to levothyroxin therapy. In our all five patients diagnosis of hypothyroidism with rhabdomyolysis with ARF was made on history, clinical examination and lab investigation and confirmed on response to levothyroxin therapy.

There is a wide variation in the clinical presentation of rhabdomyolysis. The classical triad of symptoms is muscle pain, weakness and reddish-brown urine. However, these classical features are seen in fewer than 10% of the patients².

Histological changes have been demonstrated in both rats and humans.¹⁴ Physiological effects more commonly include water and electrolyte abnormalities especially hyponatremia and associated with this altered

TABLE- 1 : Investigation for different parameters

	1	2	3	4	5
Hb (g %)	10	13	14	12.9	13.2
TLC, DLC, Platelet	WNL	WNL	WNL	WNL	WNL
Sr. Na	130	140	134	134	132
K ⁺	4.0	4.1	4.2	3.9	4.1
Sr. creatinine	2.6	2.4	2.2	2.5	4.8
SGPT (U/l)	103	106	176	89	106
CPK total	2500	1900	3900	3600	3700
Sr. TSH (mU/l)	150	200	250	302	212
Free T ₃ (2.3- 4.2pg/ml)	1.0	<0.89	<0.49	<0.36	<0.69
Free T ₄ (0.7- 1.7ng/dl)	0.3	<0.12	<0.2	<0.1	<0.2
CXR PA view	WNL	WNL	WNL	WNL	WNL
ECG	WNL	WNL	WNL	WNL	WNL
Urine R/M	Protein +	Protein ++	Protein ++	Protein +++	Protein ++
Urine - pigmented granular cast	Present	Present	Present	Present	Present
Urine for myoglobin	Positive	Positive	Positive	Positive	Positive

intrarenal hemodynamic, including decreases in both renal blood flow and glomerular filtration rate (GFR). This is believed to be principally due to the hypothyroidism induced hypodynamic state.¹³ In rare cases, renal failure can be secondary to rhabdomyolysis.^{10,12}

Current evidence is unclear of the timescale required to develop renal impairment and there is also considerable debate as to the degree of reversibility following adequate hormone

replacement. In one study, the renal failure did not recover fully to previous levels following the initiation of thyroid replacement therapy and correction of the hypothyroidism.⁴ However, a more recent study demonstrated abnormalities after two weeks of hypothyroidism, and fully reversed on appropriate thyroid replacement.⁹ Whether long-term untreated hypothyroidism will result in permanent renal impairment is not known. In present study, in all five patients, renal failure was reversed completely within a week.

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