Case report: acute kidney injury secondary to myxedema accompaning rhabdomyolysis

N.Eftimovska – Otovikj, E. Poposka, B. Popovska, E.Nikolova, I. Mickovski, R. Miloshevska

General City Hospital "8mi Septemvri", Department of nephrology, Skopje, R. Macedonia

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Introduction

- Acute kidney injury (AKI) occurring in hypothyroidism is uncommon, because association between hypothyroidism and AKI is rare.
- The exact mechanism for kidney failure still remains unknown.
- It has been postulated that absence of thyroid hormone alters mitochondrial oxidation and reduces glycogenolysis resulting in muscular atrophy
- Increased deposition of glycosaminoglycan and hypertrophy of slow muscle fiber occurs causing myopathy and increased fatigue.
- T4 deficiency alters muscle permeability resulting in release of muscle enzymes, known as creatnine kinase.
- Its accumulation, known as rhabomyolysis is a well-known attribute of AKI.

Desciption of case

- A 64-year-old man presented with 2 weeks history of weakness, malaise, lost appetite and oedema came in EC.
- Medical history: hypertension and coronary stenting (2023).
- Physical examination:grey-brown dry skin, peripheral oedema, slow thought and speaking.
- Laboratory examinations on day of admission show:
  - CK =4158 U/l, CK-MB = 683 U/l, creatinine = 675 μmol/L, urea = 35 mmol/L, AST =1671U/L, ALT = 968 U/l and LDH = 1477 U/ml.

  Laboratory analysis on 1 day show (hipothyroidism):
  - Free T4:0.08 ng/dL and TSH:150 μIU/mL.

Working diagnosis and treatment

- Working diagnosis: hypothyroidism-induced rhabdomyolysis causing AKI.
- Treatment: Intravenous fluids, urinary alkalization and substitution with L-thyroxine initiation dose of 100 ug/d and corticosteroids (urbason 2x40 mg/d) were started.
- All hepatotoxic agents were removed due to hepatic lesion.
- Ultrasound of urinary tract: normal dimension of both kidneys with hyperechoic parenchyma
- Forced diuresis with furosemide led to a progressive improvement in symptoms, but renal function worsened:
  - urea 35….39.6 mmol/L
  - creatinine 675…841 µmol/L
  - K 4.5 mmol/L

  } requiring hemodialysis

- Four hemodialysis and thyroxine substitution led to partial recovery of renal function: creatinine 267 µmol/L, urea 21.9 mmol/L.
- Hepatic enzymes and CK fell to normal values and thyroid hormones increased
  - ALT: 69
  - AST: 28
  - CK: 377; CK-MB 38
  - T3:1,16 ng/d
  - TSH:50.2 µIU/mL
  - anti TPO:1300 U/ml).

Patient follow up

- 2 weeks after discharge, patient came on regular nephrological control.
- Lab analysis confirmed persistence of elevated uremic toxins: creatinine: 199µmol/L, urea 10mmol/L, K 5.1mmol/L
- Dose od L thyroxine was increased to 125 mcg every day resulting to TSH 5.98µIU/mL, FT4 1.61ng/dL, T3 3.7ng/dL

Conclusion

- Hypothyroidism related AKI due to rhabdomyolysis is reversible, if it's correctly diagnosed.
- We describe a case of severe autoimmune hypothyroidism and AKI requiring hemodialysis, where thyroid hormone replacement resulted in partial restoration of the renal function.
- This case emphasizes the necessity for evaluation of thyroid functions in patients with unexplained AKI because it's a reversible cause and normal renal function can be attained with initiation of levothyroxine therapy.

Thank you for your attention