Dear Editor,

Thyrotoxicosis is a common disorder and generally caused by Graves’ disease, thyroiditis, and toxic nodule goiter while hyperthyroidism especially refers to increased thyroid hormone synthesis and secretion (1). A number of diverse organ systems are affected by the excess of thyroid hormone. The basal metabolism acceleration can result in abnormalities of such biochemical parameters as glucose, lipid and protein. We report a rare case of severe hypoalbuminemia with thyroid storm. As far as we know, this is the first case of severe hypoalbuminemia due to thyrotoxicosis in the literature.

41 years old female patient with the diagnosis of Graves’ disease had been followed up for 16 years. However she didn’t have a regular control and drug use in the last one year. She referred to hospital with palpitation, dyspnea and complaints of discomfort. Her temperature was 38.3 °C, arterial blood pressure was 178/88mmHg, irregular heart rate was 140beats/min, respiratory rate was 22 breaths/min during the examination. Blood analysis revealed TSH of 0.005 IU/ml (normal ranges 0.2 to 4.4), FT3 of 10.9 pg/ml (normal ranges 2 to 4.4), FT4 of 3.5 pg/ml (normal range 0.9 to 1.7), A-TPO of 600 IU/ml, A-TG of 4000 IU/ml. The other routine examinations revealed postprandial blood glucose 158 mg/dl, ALT: 16 U/L, AST: 23 U/L, total-protein: 4.7 g/dl, albumin: 1.8 g/dl, urea: 49 mg /dl, creatinine: 0.6mg/dl. The patient was hospitalized with the diagnosis of thyrotoxic crisis. No proteinuria was detected in 24-hour urine analysis. Acute-phase reactant levels were normal. Anti-endomysium and antigladin antibodies were negative. Upper gastrointestinal endoscopy revealed normal mucosal findings. She was treated with large doses of propylthiouracil, propranolol, lithium, Lugol’s solution and dexamethasone. On the fourth day of treatment, TSH of 0.005 IU/ml, FT3 of 2.97 pg/ml, FT4 of 1.98 pg/ml and lithium of 1.02 mmol/L. After total thyroidectomy, L-thyroxine was started 10 days post-operatively. Levels of total protein and albumin returned to normal in the follow-up.

Hyperthyroidism is a condition where glucose turnover, energy expenditure, lipolysis, and protein turnover are all higher than normal (2). The more protein turnover goes up, the more protein breakdown in all parts of the body and muscle protein breakdown increase (2). Increased rates of proteolysis with stable protein synthesis rates have been reported in experimental hyperthyroidism (3).

Liver functions are affected in thyrotoxicosis and there are changes in liver function tests in serum. Liver failure in hyperthyroidism was first reported by Haberson in 1874 (4). Hyperthyroidism affects histology and metabolism of the liver (5). Elias RM et al.(6) reviewed 40 patients with acute thyrotoxicosis. Albumin and international normalized ratio (INR) values were examined in order to evaluate liver synthesis function. In the records of the patients with thyrotoxic crisis, decrease in albumin was detected in 11 out of 40 patients, the lowest value was 2.9 g/dl. Abnormal values of liver function tests returned to normal in the patients following treatment. As it comes to etiology of liver abnormalities in patients, it can be stated that patients with acute thyrotoxicosis suffer from direct thyroid hormone-mediated hepatocyte damage (6).

Severe hypoalbuminemia (albumin: 1.8 g/dl) was detected in our patient who has thyrotoxicosis in her history for a long time. No pathology was detected other than hepatocyte damage when we investigated the etiology of hypoalbuminemia. This was also supported by albumin values which returned normal range following treatment.

In conclusion, severe hypoalbuminemia can occur in many diseases. Long term progress of thyrotoxicosis is a rare cause of hypoalbuminemia. So we suggested...
that patients with acute thyrotoxicosis should be evaluated for direct thyroid hormone-mediated hepatocyte damage which can result with severe hypoalbuminemia.

References


