

中文題目：原發性甲狀腺功能低下合併黏液水腫昏迷：一種診斷不足的急症
英文題目：Hypothyroidism presented with myxedema coma: an underdiagnosed life threatening epidemic

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Introduction:

Myxedema coma, the extreme manifestation of hypothyroidism, is an endocrine emergency associated with a high mortality rate. It can present clinicians with a diagnostic dilemma because patients present with altered consciousness rather than coma. [1] Patients usually present with confusion, lethargy, and possibly obtundation. [2] A high index of suspicions is needed among clinicians in order to rapidly recognize the condition and make an early diagnosis. We describe the case of a typical presentation of hypothyroidism with impending myxedema coma, where prompt recognition and treatment led to quick recovery.

Case report:

A 65-year-old man developed progressive shortness of breath and generalized edema in recent two months. His past medical history were significant for coronary artery disease status post percutaneous transluminal coronary angioplasty (2 years ago), diabetes mellitus and hypertension. His symptoms were initially attributed to acute decompensated heart failure.

Concerning his further history, a high serum creatine kinase of 5393 U/l (normal: < 80 U/l) and creatine kinase-MB of 115 U/l on admission were noteworthy. His symptoms did not show any significant improvement despite use of diuretics. Repeated ambulatory investigations by his cardiologist did not reveal any sufficient explanation for his symptoms. He was not taking lipid-lowering drugs or anticonvulsants. He had no history of arthropathy or renal dysfunction.

Rheumatologist was consulted for progressive elevation of creatine kinase level to 7521 U/l and creatine kinase-MB of 148 U/L, suggesting that the source of CK elevations is skeletal muscle; suspecting myositis as the possible cause. After questioning, increased fatigability, cold intolerance, slowing in his intellectual abilities, and a notable weight gain of 16 kg in recent 2 months became evident. Physical examination revealed puffiness of the face, pitting edema, and somnolence. No proximal muscle weakness were found.

Under high suspicion of hypothyroidism with impending myxedema coma, thyroxine replacement therapy was initiated prior to obtaining formal laboratory results. A thyroid function tests revealed elevated thyroid-stimulating hormone (209 uIU/ml) and low free T4 (<0.038 ng/dl). Sonography of the thyroid gland showed essentially normal results. Primary hypothyroidism was highly suspected.

Treatment and follow-up: Thyroid hormone replacement therapy resulted in resolution of clinical symptoms.

Discussion:

Hypothyroidism is a common disorder, yet often go undetected [3]. Symptoms develop so insidiously and are so nonspecific [4], that identifying thyroid disease clinically can be challenging and individuals may not seek medical attention. However, individual symptoms associated with hypothyroidism are often unrecognized, and are frequently confused with other health problems, particularly in individuals with multiple comorbid conditions [5]. Diagnosis of hypothyroidism can be achieved by a combination of in-depth history, thorough physical examination and judicious use of laboratory investigations [6].

It is well known that muscle disease is a frequent complication of hypothyroidism, ranging from asymptomatic elevation of serum creatine kinase (CK) to disabling muscle weakness [7]. Such a high serum creatine kinase level in a patient with hypothyroidism underscores the importance of assessing thyroid function in patients with weakness and high serum muscle enzyme concentrations.

As hypothyroid myopathy is a treatable condition with typical resolution of symptoms after thyroid replacement therapy [8]; therefore, thyroid function tests should always be performed in all patients with unexplained myopathy.

References:

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