Thyrotoxic hypokalemic paralysis: a case report

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Transient periodical muscle paralysis is an important neurological presentation. Thyrotoxic hypokalemic paralysis is an uncommon type of thyrotoxic paralysis. Except for the findings specifically caused by the subtle hyperthyroid state the clinical and biochemical features of thyrotoxic hypokalemic paralysis are identical to those of familial periodic paralysis. In this article, a case of a 26 year old male patient, previously diagnosed with toxic hyperthyroidism for 3 weeks without treatment presented to the physician at the emergency room with onset of tetraparesis. After administration of potassium phosphate, the patient began to improve, and one day after the onset the was able to walk normally. The labatory change of this case was presented.

Key words: Thyrotoxic hypokalemic paralysis.

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ภาวะกล้ามเนื้ออย่างแรงเป็นช่วงช่วงบางครั้งบางคราวเป็นอาการที่มีความสำคัญทางด้าน
ประสาทวิทยา ภาวะกล้ามเนื้ออย่างแรงชนิดไปแสดงเชื่อมต่อในผู้ป่วยโรคยอดเป็นพิษจัดเป็นความผิด
ปกติที่หายจากภาวะกล้ามเนื้ออย่างแรงที่เกิดกับผู้ป่วยโรคยอดเป็นพิษ นอกจากอาการตรวจพบความ
ผิดปกติของสรีนิ STDOUT ซึ่งสูงขึ้นแลกว่าอาการทางคลินิก ตลอดจนอาการตรวจทางห้องปฏิบัติการชนิด
อื่น ๆ ให้ผลลัพธ์คล้ายกับโรคภาวะกล้ามเนื้ออย่างแรงที่พบกันในกระเพาะบิน ในบทความนี้ได้รายงาน
ผู้ป่วย 1 รายที่มีพบแพทย์ให้เจ็บปวดด้วยอาการแนวทางกล้ามเนื้ออย่างแรง โดยผู้ป่วยรายนี้เป็นผู้ป่วยที่เพิ่งจะ
ได้รับการวินิจฉัยว่าเป็นโรคโรคยอดเป็นพิษในช่วง 3 สัปดาห์ก่อนเกิดอาการผิดปกติในครั้งนี้และยังไม่
ได้รับการรักษาด้วยยาโรคยอดเป็นพิษ หลังจากให้ผลทดสอบและมีผลป่วยมีอาการครั้งนี้และยัง
ได้เป็นปกติใน 1 วัน ความเปลี่ยนแปลงของผลการตรวจทางห้องปฏิบัติการได้ถูกนำเสนอในรายงานนี้
Transient periodical muscle paralysis is an important neurological presentation. Unlike the myasthenias, paralyses in this group of diseases is transient and quickly reversible to a normal state.

Four common causes of transient periodical muscle paralyses have been identified: 1) familial periodical paralysis, 2) hyperthyroidism with periodical paralysis, 3) hyperkalemic paralysis of von Eulenberg, and 4) hereditary periodic adynamia or Gamstorp's disease. Differences among these recurrent muscle weakness disorders are small and require good history taking and investigation.

In this article, a case of thyrotoxic hypokalemic paralysis in a young male, focusing in laboratory change is presented and a literature review on thyrotoxic hypokalemic paralysis performed.

Case report

Previous history

A 26 year old made patient previously diagnosed with toxic hyperthyroidism for 3 weeks without treatment, presented to the physician at the emergency room with onset of tetraparesis. He revealed no history of concomitant drug usage. No familial history of muscle weakness disorder was revealed. In the emergency ward, the patient was agitated, with tachycardia (110/min). He was not able to flex the lower and upper extremities against a light resistance and was hypotonic, without tendon reflexes. The cranial nerve and sensory was intact. No pathologic reflex was detected.

Present laboratory history

Biochemical analyses revealed severe hypopotassemia ($K^+ = 2.5\ \text{mEq/l}$) while the other biochemical analytes were within normal limit. The complete blood count, urinalysis muscle enzyme results were within normal limit. After administration of potassium phosphate, the patient began to improve ($K^+ = 3.0\ \text{mEq/l}$), and one day after the onset he was able to walk normally ($K^+ = 3.2\ \text{mEq/l}$). One month later, this patient was underwent subtotal thyroidectomy ($K^+ = 3.8\ \text{mEq/l}$).

Discussion

The periodic paralyses are a rare group of disorders which may be familial, sporadic, occur in association with hyperthyroidism or as a result of potassium loss. In this case the periodic myopathy is a hypokalemic paralysis, Generally, hypokalemic paralysis is a relatively uncommon but can be a potentially life-threatening clinical syndrome. The syndrome of hypokalaemic paralysis represents a heterogeneous group of disorders characterized clinically by hypokalaemia and acute systemic weakness.

Most cases are due to familial or primary hypokalaemic periodic paralysis; sporadic cases are associated with numerous other conditions including barium poisoning, hyperthyroidism, renal disorders, and gastrointestinal potassium losses. Thyrotoxic periodic paralysis is a self-limiting disorder that is cured by the treatment of the underlying hyperthyroidism. It is the most common acquired form of periodic paralysis. Generally thyrotoxic periodic paralysis presented with normal blood potassium level.

As the pathogenesis of hypokalemic periodic paralysis, depolarization block induced by membrane permeability change in the face of hypokalemia triggered by excess insulin was strongly suggested.
and supported experimentally in part. Muscle fibers chronically depolarized by 10 – 15 mV, but membrane conductance normal between attacks. Recent linkage analysis on familial hypokalemic periodic paralysis revealed that the abnormality is linked to a mutation in voltage-gated Ca channel. The difficulty remains how to explain the cause of hypokalemia which is almost always preceding the attack of periodic paralysis of this type.

Thyrotoxic hypokalemic paralysis is an uncommon type of thyrotoxic paralysis. It has been observed predominantly in Asians, however, it is known as a sporadic cause of muscular paralysis in Caucasians also. The demographic data of our case can match with the previous description of patients affected with this disease. As previous mentioned, this disease is predominantly a disease of males, (male: female ratio = 20 : 1). The usual age of onset of the disorder is similar to that of thyrotoxicosis, with the second to fourth decades being the most common. Rarely, there is family history of this disorder.

Except for the finding specifically caused by the subtle hyperthyroid state the clinical and biochemical features of thyrotoxic hypokalemic paralysis are identical to those of familial periodic paralysis. Successive investigations usually documented an undiagnosed case of Graves' disease. As detected in our case, Graves' disease is the most common cause of hyperthyroidism in affected patients, but any cause of thyrotoxicosis, including administration of excessive amounts of exogenous thyroid hormone, can trigger attacks of the disorder in susceptible patients.

In spite of the striking clinical similarities, familial periodic paralysis can be differentiated on the basis of the relationship of the paralytic attacks to thyroid status.

Patients will have normal thyroid function, and challenge with exogenous thyroid hormone does not aggravate or worsen their symptoms. On the other hand, patients with thyrotoxic hypokalemic paralysis have attacks only when they are hyperthyroid.

Once a hypokalaemic attack has started, administration of potassium is standard therapy. Treatment of underlying thyrotoxicosis abolish further attack. This case also presented the dramatically improvement of the symptoms accompanied with the improvement of serum potassium level. Correction of the underlying disease by subtotal thyroidectomy should also be considered. However, in our case, the final decision in this case was subtotal thyroidectomy and the follow up result was satisfied.

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References


