

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 laboratory change of this case was presented.

Key words : *Thyrotoxic hypokalemic paralysis.*

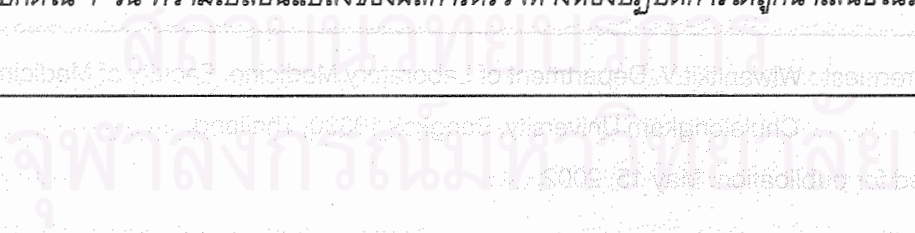
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ภาวะกล้ามเนื้ออ่อนแรงชนิดโบทูลินัมในผู้ป่วยไตเรื้อรังเป็นพิษ

วารสาร วชิรเวช. ภาวะกล้ามเนื้ออ่อนแรงชนิดโบทูลินัมในผู้ป่วยไตเรื้อรังเป็นพิษ. จุฬาลงกรณ์เวชสาร 2545 ต.ค ; 46(10): 823 - 7

ภาวะกล้ามเนื้ออ่อนแรงเป็นช่วงช่วงบางครั้งบางคราวเป็นอาการที่มีความสำคัญทางด้านประสาทวิทยา ภาวะกล้ามเนื้ออ่อนแรงชนิดโบทูลินัมในผู้ป่วยไตเรื้อรังเป็นพิษจัดเป็นความผิดปกติที่หายากของภาวะกล้ามเนื้ออ่อนแรงที่เกิดในผู้ป่วยไตเรื้อรังเป็นพิษ นอกจากการตรวจพบความผิดปกติของฮอร์โมนไตเรื้อรังที่สูงขึ้นแล้วอาการทางคลินิก ตลอดจนการตรวจทางห้องปฏิบัติการชนิดอื่น ๆ ให้ผลคล้ายคลึงกับโรคภาวะกล้ามเนื้ออ่อนแรงที่พบร่วมกันในครอบครัว ในบทความนี้ได้รายงานผู้ป่วย 1 รายที่มาพบแพทย์ที่ห้องฉุกเฉินด้วยอาการแขนขาอ่อนแรง โดยผู้ป่วยรายนี้เป็นผู้ป่วยที่เพิ่งจะได้รับการวินิจฉัยว่าเป็นโรคไตเรื้อรังเป็นพิษในช่วง 3 สัปดาห์ก่อนเกิดอาการผิดปกติในครั้งนี้และยังไม่ได้รับการเริ่มต้นรักษาภาวะไตเรื้อรังเป็นพิษ หลังจากให้โบทูลินัมเสริมผู้ป่วยมีอาการดีขึ้นและเดินได้เป็นปกติใน 1 วัน ความเปลี่ยนแปลงของผลการตรวจทางห้องปฏิบัติการได้ถูกนำเสนอในรายงานนี้



Transient periodical muscle paralysis is an important neurological presentation. Unlike the myasthenias, paralyzes in this group of diseases is transient and quickly reversible to a normal state.

Four common causes of transient periodical muscle paralyzes have been identified: 1) familial periodical paralysis, 2) hyperthyroidism with periodical paralysis, 3) hyperkalemic paralysis of von Eulenberg, and 4) hereditary periodic adynamia or Gamstop's disease.⁽¹⁾ 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 male 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 ($K^+ = 2.5$ 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 ($K^+ = 3.0$ mEq/l), and one day after the onset he was able to walk normally ($K^+ = 3.2$ mEq/l). One month later, this patient was underwent subtotal thyroidectomy ($K^+ = 3.8$ mEq/l).

Discussion

The periodic paralyzes are a rare group of disorders which may be familial, sporadic, occur in association with hyperthyroidism or as a result of potassium loss.⁽¹⁾ In this case the periodic myopathy is a hypokalemic paralysis. Generally, hypokalemic paralysis⁽²⁾ 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⁽³⁾ is a self-limiting disorder that is cured by the treatment of the underlying hyperthyroidism. It is the most common acquitted 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).⁽³⁻⁴⁾ The usual age of onset of the disorder is similar to that of thyrotoxicosis, with the second to fourth decades being the most common.^(3,5) Rarely, there is family history of this disorder.^(3,6)

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.⁽⁷⁻⁹⁾ 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.⁽⁷⁻⁹⁾

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.^(3,5,7) 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.

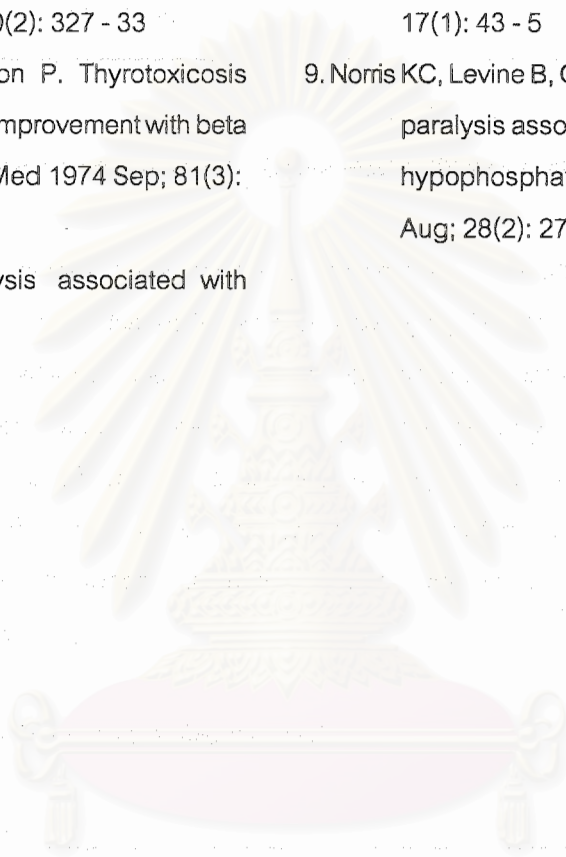
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References

1. Aminoff MJ. Nervous system. In: Tierney LM, McPhee SJ, Papadakis MA, eds. Medical Diagnosis & Treatment. 35th ed. Stamford: Prentice-Hall International, 1996: 858 - 914
2. Papakadis MA. In: Tierney LM, McPhee SJ, Papadakis MA, eds. Medical Diagnosis & Treatment. 35th ed Stamford; Prentice-Hallo International, 1996: 768 - 94
3. McFadzean AJ, Yeung R. Periodic paralysis complicating thyrotoxicosis in Chinese. Br

- Med J 1967 Feb 25; 1(538): 451 - 5
4. Ghose R, Quail G, King R, Moloney P. Hypokalaemic paralysis in remote aboriginal communities. Aust Fam Physician 1996 Jul; 25(7): 1172 - 3
5. Engel AG. Thyroid function and periodic paralysis. Am J Med 1961 Feb; 30(2): 327 - 33
6. Conway MJ, Seibal JA, Eaton P. Thyrotoxicosis and periodic paralysis: improvement with beta blockade. Ann Intern Med 1974 Sep; 81(3): 332 - 6
7. Linder MA. Periodic paralysis associated with hyperthyroidism: report three cases. Ann Intern Med 1955 Aug; 43(2): 241 - 54
8. Kodali VR, Jeffcote B, Clague RB. Thyrotoxic periodic paralysis : a case report and review of the literature. J Emerg Med 1999 Jan-Feb; 17(1): 43 - 5
9. Norris KC, Levine B, Ganesan K. Thyrotoxic periodic paralysis associated with hypokalemia and hypophosphatemia. Am J Kidney Dis 1996 Aug; 28(2): 270 - 3



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