

CASE REPORT IN EMERGENCY MEDICINE

A CASE OF THYROTOXIC PARALYSIS CAUSED BY CONSUMPTION OF IODOCASEINE

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Abstract

Acute hypokalaemic paralysis is a rare but treatable cause of acute limb weakness. Thyrotoxic paralysis is an uncommon, potentially life-threatening endocrine emergency and it is a rare complication of hyperthyroidism. The most common causes of hyperthyroidism include Graves' disease, multinodular goiters or solitary thyroid nodule, and iodine-induced thyrotoxicosis (Jod-Basedow syndrome). Thyrotoxicosis factitia, which is caused by the excessive ingestion of exogenous thyroid hormone or iodine derivatives administration, has been rarely reported as a cause of thyrotoxic paralysis. We describe the case of a young Caucasian male with flaccid paralysis of all four limbs and severe hypokalaemia after inappropriate iodine derivatives (iodocasein) intake to show, in conclusion, how critical care physicians need to be aware of this rare but curable condition.

KEY WORDS: Iodocaseine; hypokalaemia; thyrotoxic paralysis.

Riassunto

La paralisi acuta ipokaliemica è una causa rara ma curabile di astenia acuta. La paralisi tireotossica è un'emergenza endocrina non comune e potenzialmente pericolosa per la vita ed è una rara complicanza dell'ipertiroidismo. L'ipertiroidismo è causato prevalentemente dalla malattia di Graves, da gozzi singoli o multinodulari, e dalla malattia indotta da iodio (Jod-Basedow). La tireotossicosi factitia, che è causata dall'eccesso di assunzione esogena di ormone tiroideo o di derivati dello iodio, è stata raramente segnalata come una causa di paralisi tireotossica. Descriviamo il caso di un giovane maschio con paralisi flaccida ai quattro arti e riscontro di severa ipopotassiemia in seguito ad assunzione inappropriata di derivati dello iodio (iodocaseina), per evidenziare, in conclusione, come i medici che lavorano in emergenza, debbano essere consapevoli di questa rara ma curabile condizione.

TAKE-HOME MESSAGE: Iodine derivatives intake can generate hyperthyroidism, which in some cases can lead to severe hypokalaemia and subsequent acute limb weakness and paralysis. Therefore, critical care physicians need to be aware of thyrotoxic paralysis, which is a rare but treatable condition.

Competing interests: none declared

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INTRODUCTION

Acute systemic weakness is a common complaint in the emergency department, which requires very broad differential diagnoses including neurologic, metabolic, and infectious aetiologies [1]. Acute hypokalaemic paralysis, which is a clinical syndrome characterised by acute systemic

weakness and low serum potassium, is a rare but treatable cause of acute limb weakness. Hypokalaemia may be induced by a wide variety of medical disorders. Thyrotoxic paralysis (TP) is an uncommon, potentially life-threatening endocrine emergency, which is a rare complication of hyperthyroidism. Despite the fact that the pathophysiologic features of this endocrinological disorder have not been entirely elucidated, it is most likely that TP involves a hyperthyroidism-related hypokalaemia with a subsequent muscle-weakening condition caused by a sudden shift of potassium into cells and progressive depolarisation of the resting membrane potential [2]. The most common causes of hyperthyroidism include Graves' disease, multinodular goiters or solitary thyroid nodule, iodine-induced thyrotoxicosis (Jod-Basedow syndrome) [3, 4]. Thyrotoxicosis factitia, which is caused by the excessive ingestion of exogenous thyroid hormone or iodine derivatives administration, has been rarely reported as a cause of TP [5].

We describe the case of a young Caucasian male with flaccid paralysis of all four limbs after inappropriate iodine derivatives intake.

CASE REPORT

A 31-year-old white male presented to the emergency department with myalgias, extreme acute weakness and parasthesia of his lower extremities and inability to walk or stand without support; superior extremities motor strain and tongue parasthesias. There was no history of fever, gastrointestinal illness, palpitations, tremor, heat intolerance or known ingestion of toxins.

On examination, his vital signs revealed blood pressure 110/70 mmHg, heart rate 120 beats/min, respiratory rate 14 breaths/min, SatO₂ 98%, and body temperature 36°C. There was no exophthalmos, and the thyroid gland was normal in size. The neurologic examination showed normal cognition and symmetrical flaccid paralysis with hyporeflexia in upper and lower extremities. No fasciculations, myoclonus, or muscular atrophy were observed. The remainder of the physical examination was unremarkable.

Laboratory tests on peripheral blood revealed the following values: Haemoglobin 14.3 g/dL, white cell count 4,900/ μ L, and platelet count 254,000/ μ L, sodium 143 mmol/L, potassium 1.21 mmol/L, chloride 110 mmol/L, calcium 9.44 mg/dL, magnesium 1.89 mg/dL, creatinine 0.61

mg/dL, pH 7.39, and bicarbonate 20.2 mmol/L. The electrocardiogram showed sinus tachycardia, I° atrio-ventricular block (PQ 0.22 seconds), and QT prolongation (QTc 0.668 seconds). The thyroid panel showed a depressed serum thyroid stimulating hormone (TSH) level (0.057 μ U/mL), elevated free T₃ (9.16 pg/mL) and free T₄ (2.42 ng/dL).

The above neurologic and electrocardiographic abnormalities resolved within 12 hours after infusion of 190 mmol of potassium supplements. After thorough investigation, he admitted the regular intake of an iodocasein/thiamine-containing medicinal product, composed by iodocasein 125 mg (7.6% of iodine) and thiamine nitrate (12.33 mg), 6-8 tablets/day in the previous four months.

He was discharged three days later in better general conditions with specific instruction not to consume anymore iodine drugs. Seven days after, he was in good health and serum potassium level returned to normal (4.6 mmol/L).

DISCUSSION AND CONCLUSION

TP is a rare but serious complication of hyperthyroidism. Orientals make up to 90% of all cases reported in the literature [1, 6]; this condition has also been reported in Caucasians, native American Indians, and Blacks [1].

Unlike hyperthyroidism, which has a strong female preponderance, TP occurs 22–27 fold more in men as compared to women [1, 7].

Hypokalaemia in the setting of hyperthyroidism is the hallmark feature of this condition. Hypokalaemia occurs as a result of transcellular shift of potassium into the intracellular fluid [2]. This transcellular shift is due to activation of Na-K-ATP pump in thyrotoxicosis [8]. Normalization of potassium resolves the associated paralysis.

The majority of TP is seen in hyperthyroidism due to Graves' disease, however toxic adenoma goiter, thyroiditis, toxic multinodular goiter, and administration of excessive amounts of exogenous thyroid hormone or iodine derivatives, can trigger attacks of TP in susceptible patients [1].

The iodine-induced hyperthyroidism represents the Jod-Basedow phenomenon that occurs in patients unable to induce the Wolff-Chaikoff block, a normal protective mechanism that, in the presence of excess iodine, inhibits organification and subsequent excessive thyroid hormone synthesis [9, 10]. Iodine-containing medications are known to cause hyperthyroidism, and they include amiodarone, radiographic contrast dyes, iodinate glycerol, kelp supplements, and topical disinfectants used in surgery [4, 11, 12]. Iodine-induced TP is a very rare disorder, with not many cases identified upon review of the literature [4, 5, 12–17].

A report described a young male who developed TP after exposure to saturated solution of potassium iodide [15]. Another young male presented to the emergency department with sudden lower extremity weakness, heat intolerance, tremors, weight loss and hypokalaemia during therapy with amiodarone [12]. Akar et al [16] reported a iodine-induced TP case in a male who received radioactive iodine and Kane and Busch [4] described a TP case whose onset of symptoms was one day after assumption of iodinated contrast (radiographic dye) for coronary catheterization.

Both excessive exogenous T_3 or T_4 administration can lead to thyrotoxicosis factitia. Thyrotoxicosis factitia induced by excess exogenous thyroid hormone administration has been rarely reported as a cause of TP. Indeed, only 2 cases of TP from thyroxine (T_4) administration have been reported [13, 14]. More recently, a case of TP induced by exogenous triiodothyronine (T_3) administration has been reported [5] and another case has been described as a TP secondary to consumption of nutraceutical containing T_3 [17].

In the present case report, the cause of thyrotoxicosis was an abusive iodocasein consumption. To our knowledge, only two iodocasein-induced hyperthyroidism cases have been reported [11], one of which was an unpublished case by Veneroni et al. [18], but no case of idocasein-induced TP has been ever reported. TP should be considered in the differential diagnosis of all cases of muscle weakness or paralysis. This becomes especially important given its reversible nature in case of appropriate treatment. Emergency employees should give importance to this condition, which is a diagnostic and therapeutic emergency that may lead to life-threatening complications.

Therefore, in acute care setting it is imperative to be aware of TP and its causes for reducing morbidity and mortality.

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