Review J Clin Endocrinol Metab. 2006 Jul;91(7):2490-5. doi: 10.1210/jc.2006-0356.

Epub 2006 Apr 11.

Clinical review: Thyrotoxic periodic paralysis: a diagnostic challenge

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PMID: 16608889 DOI: 10.1210/jc.2006-0356

Abstract

Context: The aim of this article was to review the clinical presentation, pathogenesis, and management of thyrotoxic periodic paralysis (TPP).

Evidence acquisition: A MEDLINE search was conducted for articles published during the last 40 yr based on the key words thyrotoxic periodic paralysis and hypokalemic periodic paralysis. A total of 281 primary articles and 168 references of the retrieved articles were also reviewed.

Evidence synthesis: TPP is a common complication of hyperthyroidism in Asian men but is increasingly seen in Western countries. Hypokalemia and muscle paralysis results from a sudden intracellular shift of potassium and is not due to potassium deficiency. Clinical features of hyperthyroidism in patients with TPP may be subtle. Immediate potassium supplementation prevents serious cardiopulmonary complications and may hasten the recovery of muscle weakness. Nonselective beta-adrenergic blockers can ameliorate and prevent recurrence of the paralytic attacks. This episodic paralysis will remit with definitive control of hyperthyroidism. Increased sodium-potassium ATPase pump activity and enhanced insulin response in patients with TPP is postulated to contribute to the hypokalemia. The genetic predisposition for TPP is not entirely clear. Association of polymorphisms of the calcium channel alpha1-subunit gene with TPP has been noted.

Conclusions: Due to population mobility, TPP is increasingly common in Western countries. Early diagnosis and prompt treatment prevent life-threatening complications associated with hypokalemia and muscle weakness. Assaying of thyroid function in patients with hypokalemic paralysis distinguishes TPP from other forms of hypokalemic periodic paralysis.

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