Thyrotoxic periodic paralysis amongst the ethnic Asians living in the west—an important entity to consider in the hospital setting

Sir,

I read with interest the article ‘Newly diagnosed thyrotoxicosis in hospitalized patients: clinical characteristics’ by Rotnam-Pikielny et al.1 In their study, the team found that weakness, weight loss and palpitations were the leading symptoms in patients newly diagnosed with thyrotoxicosis during hospitalization. I totally agree with their view that thyrotoxicosis should be included in the differential diagnosis of patients presenting with those symptoms in the hospital setting.

In this retrospective study, in the authors’ own admission, there was an uncertainty regarding the presence of non-specific symptoms like weakness and weight loss in other acutely ill patients. The cohort (58 in total) had a mixed ethnicity of Jews, Arabs and interestingly 3% of Philipinos. There were obviously more women patients than males (36/22) as expected in this group. It was also interesting to see that none of the cohort patients had any electrolytic abnormalities (serum potassium being normal = 4.2 ± 0.4), albeit the presence of laboratory confirmed thyrotoxicosis.

In an Asian population, thyrotoxic periodic paralysis (TPP) is a well-recognized entity with hypokalaemia and thyrotoxicosis and this is the most common cause of periodic weakness of muscles in the hospital setting.2–6, 9 It has a male dominance (20:1),5 compared with the women dominance found by the authors in their study. Some 2% of all thyrotoxic patients in Japan were reported to have this complication.5 Consequently, 90% of case reports on TPP in the literature come from Japan and Southern China.5,6 It has also been recognized in Thai, Phillipino, Vietnamese, Korean and Malay populations.2 It is uncommon in Whites and Afro-Americans.4,8 There are reports of TPP amongst Hispanic individuals.9 Recently, a rare case of TPP with hypokalaemia was described in a white woman in the United Kingdom.10

As patients of Oriental ethnicity are now frequently encountered in the western world, I feel that TPP should also be included in the differential diagnosis of muscle weakness as a symptom in the hospital setting. In the western countries, autosomal dominant familial periodic paralysis (FPP), present similarly with weakness at the hospital doors, but always with normal thyroid function tests.

In the north of England, where there is a fair mix of diverse ethnicity in the medium to big cities, a case of Graves’ disease with hypokalaemic paralysis in a 30-year-old man of Vietnamese background was encountered recently.3 He presented to the emergency department with sudden generalized weakness which was subsequently diagnosed as TPP. He was clinically euthyroid, but had a palpable goiter, which prompted to check his thyroid status. He recovered promptly once the potassium level was restored and subsequently, he was given anti-thyroid treatment along with a Beta-blocker.3

TPP is characterized by sudden episodes of weakness usually occurring after heavy exertion or after having a high-carbohydrate meal, followed by a prolonged rest in the night.8 In the majority of reports, TPP begins between 20 years and 40 years of age and has a male dominance. The principal clinical difference between FPP and TPP is the presence of thyrotoxicosis in the latter. Recovery in TPP can be spontaneous after 3–36 h, and but may be hastened by IV K+ administration (beware of rebound rise). Beta-blockers are also very useful in TPP.

In TPP, the thyroid hormone roughly doubles the Na+-K+-ATPase-dependent K+ channel and increases the catecholamine-mediated intracellular potassium shift.11 Na+- K+-ATPase activity is also augmented by insulin12 (as a result of high-carbohydrate intake). Differences in HLA subtypes in different racial groups have been widely studied.8 Genetic mutations in the control of Na+-K+-ATPase activity within the same HLA subtype may explain the ethnic difference. A genetic mutation in TPP has recently been described in the K+ ionic channel gene KCNE3.13

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Microcytic anaemia can ‘mask’ co-existing cobalamin deficiency

Sir,

Given the fact that either autoimmune gastritis or *Helicobacter pylori* gastritis could be underlying causes of iron deficiency anaemia,¹,² or cobalamin deficiency,¹,³ no account of the haematological features of cobalamin deficiency⁴ would be complete without mentioning the fact that microcytosis may be its only manifestation when it co-exists with iron deficiency.¹ In one study comprising 160 subjects with autoimmune gastritis, the haematological profile was characterized by microcytosis (mean corpuscular volume <80 fl) in 51.9% of cases. In that study, 46% of the 83 iron deficient patients with microcytosis proved to have coexisting cobalamin deficiency.¹ As a corollary, *H. pylori* gastritis can generate an increase in intragastric pH, with the potential consequence of impairing gastrointestinal absorption of dietary iron,⁵ and can also be the underlying cause of cobalamin deficiency, the latter reversible on eradication of the offending pathogen, successful eradication being characterized by an increase in serum cobalamin levels.³ In this context, as well, there is a potential for combined cobalamin and iron deficiency to manifest itself as microcytosis. Accordingly, during upper gastrointestinal investigation for the underlying cause of iron deficiency, if no obvious cause is found, in addition to duodenal biopsy for suspected celiac disease, gastric biopsy should also be performed for evaluation of *H. pylori* status and for evaluation of autoimmune gastritis status. Confirmatory tests for the latter would also include presence of antiparietal antibodies and hypergastrinaemia.¹

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References


