A case of Graves' disease presenting after commencing alemtuzumab treatment for multiple sclerosis

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Case:

We present the case of a 44 year old gentleman, who presented with symptoms of palpitations, weight loss and loose stools since September 2009. He had no preceding history of fever, neck pain or swelling. He had no family history of thyroid illness. Interestingly he had been commenced on alemtuzumab treatment for relapsing multiple sclerosis 12 months ago. (Thyroid function tests prior to treatment with alemtuzumab were normal).

Examination and Results:

On examination he had a tachycardia, fine tremor but no evidence of eye signs or goitre. Blood tests showed Ft4 of 27.0, TSH<0.03. He had positive TPO antibodies (1:6400) and positive TSH receptor antibodies. Thyroid isotope scan showed diffuse increased uptake (9%) consistent with Graves' disease. He was started on carbimazole and has since responded well to treatment.

Discussion:

Alemtuzumab, a monoclonal antibody which targets the surface protein CD52 found on lymphocytes and monocytes was granted fast track FDA status in June 2010 for the treatment of multiple sclerosis. It causes rapid and prolonged lymphopenia particularly targeting CD4+ T cells. Novel autoimmunity is the principal adverse effect arising months to years after commencing treatment. Phase II trials show that 20-30% patients develop a form of thyroid autoimmunity, typically Graves' disease. The causal factors are unknown. Graves' disease has also been associated with the use of beta interferon for the treatment of multiple sclerosis. An increase in the prevalence of Graves' disease in family members of patients with multiple sclerosis was noted during the trials, proposing the theory of common autoimmune genetic susceptibility factors with the specific autoimmune phenotype being determined by additional genetic or environmental factors.