Ward Round - Seizures, tremor and muscle weakness 20 years after thyroid surgery

Discussion

The persistent fever despite broad spectrum antibiotics, lack of positive identification of infection elsewhere and finding of pericardial effusion with strands on echocardiogram led to a diagnosis of presumed TB pericarditis[1]. He was treated empirically with anti-tuberculous drugs and prednisolone and made an excellent response. He continued with cotrimoxazole prophylaxis.

Thyroid function tests confirmed primary hypothyroidism with raised TSH and low fT3 and fT4. The low calcium, raised phosphorus and intracranial calcifications are all highly suggestive of hypoparathyroidism. We were unable to measure parathyroid hormone levels to confirm this. Both hypothyroidism and hypoparathyroidism are likely to have occurred secondary to the thyroidectomy he had 20 years ago. Intracranial calcification, particularly of the basal ganglia is a common occurrence in hypoparathyroidism. It increases with the duration of hypoparathyroidism and is rarely visible on plain X-rays but can be seen on a CT scan[2]. The most common site is the globus pallidus but it can also occur in the cerebellum, subcortical white matter, corona radiata and thalamus[3]. The mechanisms behind this paradoxical intracranial calcification are still unknown but some have speculated that it may be a function of hyperphosphataemia promoting ectopic calcification[4]. The cerebral calcification is associated with neurological damage, most commonly Parkinsonism, but other extra-pyramidal syndromes, cerebellar and pyramidal signs have been described[5]. The neurological problems are usually irreversible or only partially improve when hypoparathyroidism is treated.

The incidence of hypothyroidism following thyroidectomy varies depending on the underlying thyroid problem and the amount of thyroid removed but can be as high as 50%[6]. We assume our patient must have had an extensive thyroidectomy in order to have lost all 4 parathyroid glands. In such situations, thyroid function and calcium levels should be routinely monitored postoperatively.

The reported incidence of permanent hypoparathyroidism following thyroid surgery varies between 0.5 and 4% depending on the extent of operation, the surgical technique and the surgeon's experience. Symptoms of hypoparathyroidism post thyroid surgery generally begin 1 or 2 days after the procedure. In approximately 50% of cases this abnormality is transient and caused by oedema and glandular haemorrhage while the rest is permanent because of the removed parathyroid glands[7].

His problems seem to originate from his thyroidectomy 20 years ago, at the age of 12. The indication for this is not known. It is unlikely that he was thyrotoxic or had thyroid malignancy, as he was not aware of these diagnoses, so we speculate that it was done for treatment of goiter. There is no conclusive local data for the national prevalence of childhood and adult endemic or other causes of goiter, but iodine deficiency may cause childhood endemic goiter especially in people living in mountainous areas and not using iodised salt. This patient comes from a mountainous district, and although iodised salt use is widely used in Malawi these days we are not sure if it was the same 20 years ago.

His neuromuscular problems on presentation were complex. Hypocalcaemia can cause neuromuscular excitability, indicated by parasthesia, tetany, positive Chvostek and Trousseau's signs, and in the most extreme cases, seizures, as in this case. In addition he had muscular complications of hypothyroidism including weakness and poor muscle tone. We also suspect that, although he did not have typical Parkinsonism, he had involuntary movements due to the basal ganglia calcification.

The patient also had renal impairment which normalized on subsequent tests which may have exacerbated the high phosphorus and low calcium and may be the reason he developed symptomatic hypocalcaemia at this time, having been asymptomatic for 20 years. He was treated with calcium/ vitamin D supplements and thyroxine. Ideally activated Vitamin D (1,25-hydroxylated) should be used, because of its greater potency, however, it was not available so he was given the 25-hydroxylated form only.

One month after starting treatment, all the neurological and neuromuscular abnormalities had disappeared, his facial oedema and pallor had improved and his voice was normal. He continues to attend the medical department for review and repeat prescriptions- his thyroxine, calcium and vitamin D supplements will be life-long. He is yet to finish tuberculosis treatment and will start anti-retroviral therapy in March this year (2008).

Final diagnoses

- TB pericarditis (presumed)
- Hypothyroidism – post thyroidectomy
- Hypocalcaemia,hyperphosphataemia,and basal ganglia calcification- due to hypoparathyroidism
- Normochromic anaemia- multiple possible contributory factors
- Mild intercurrent renal impairment

- HIV infection: WHO clinical stage IV (Extra-pulmonary tuberculosis and oesophageal candidiasis), AIDS

Like many of the patients we see on the medical wards this patient presented with opportunistic infection (Tuberculosis) in the context of HIV. It was only after thorough clinical assessment he was found to have other serious and potentially life threatening diagnoses; hypothyroidism and hypoparathyroidism.

This case highlights the value of taking a detailed history for more accurate diagnosis at first patient's review. It also illustrates how treatment of one problem can lead to the development of another problem. Where it is difficult to follow patients up long term, or to monitor patients for the development of metabolic and endocrine problems, thyroidectomy should only be undertaken where there is a strong indication and care should be taken not to remove all of the thyroid gland or the parathyroid glands to minimize the risk of these postoperative complications.

References

1. Harries M.A., Maher D. Tuberculosis pericardial effusion: a prospective clinical study in a low-resource setting – Blantyre, Malawi. Tuberc lung dis 1997 1(4): 358-364
2. Forman, M.B., Sandler, M.P., Danziger, A., Kalk, W.J. Basal ganglia calcification in postoperative hypoparathyroidism. Clin endocrinol (Oxf) 1980 12 (4): 385-90
3. Litvin, Y., Roster, A., Bloom, R.A. Extensive cerebral calcification in hypoparathyroidism. Neuroradiology.1981 21(5):271-2.
4. Yuji M, Matsui K, et al. Cerebral subcortical calcification and Hypoparathyroidism. Jap J Med 1985 24(1),
5. Uncini, A., Tartaro, A., Di Stefano, E., Gambi, D. Parkinsonism, basal ganglia calcification and epilepsy as late complications of postoperative hypoparathyroidism. J Neurol 1985 27(1):32-7
6. Farkas, E.A., King, T.A, Bolton, J.S., Fuhrman, G.M., A comparison of total thyroidectomy and lobectomy in the treatment of dominant thyroid nodules. Am Surg. 2002 68(8):678-83
7. Arit W, Fremerey C, Callies F, Reincke M, Schneider P, Timmermann, and Allolio B. Wellbeing, mood and calcium homeostasis in patients with hypoparathyroidism receiving standard treatment with calcium and vitamin. Europian Journal of endocrinology,Feb 2002; 146: 215-222.