Letters to the editor

Copper deficiency and myelopathy after bariatric surgery

The follow up of patients after bariatric surgery ought to include evaluation, not only of iron and cobalamin status,[1] but also copper status, given the fact that bariatric surgery was implicated in 7 out of 55 cases of hypocupraemic myelopathy in a literature review published in 2010.[2]

Hypocupraemic myelopathy typically occurs months to years after bariatric surgery[3] and can be clinically indistinguishable from cobalamin deficiency myelopathy.[2,3] Given that the haematological profile of hypocupraemia is sometimes characterised by macrocytosis,[4] there is a potential for mistaken diagnosis of cobalamin deficiency myelopathy to be made when hypocupraemic myelopathy is accompanied by macrocytosis attributable to hypocupraemia.

In 44–47% of cases of hypocupraemic myelopathy, MRI shows a segment of high T2 signal in the dorsal midline of the cervical and thoracic cord.(2) By contrast, in cobalamin deficiency myelopathy, T2 weighted MRI shows hyperintense signals in the thoracic spinal cord forming an inverted 'V' sign.[5]

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Authors' reply

We appreciate the comment that Dr Jolobe makes and agree that copper status should of course be included in the screening of the metabolic patient pre and post-surgery.

The aim of our paper was not, however, to delve into follow-up post bariatric surgery of the patient with the metabolic syndrome. Indeed, we think this may be an interesting review of itself for future issue of this journal. In our review, we referenced the importance of mineral follow up. As such, we gave some examples and highlighted the importance of follow up of macronutrient and micronutrient levels: 'patients should be followed up by an expert nutritionist to establish the caloric, protein, fat, carbohydrate and micronutrients quality and quantity in their diets and the need to reinforce specific dietary intakes…'. We furthermore supported this section with appropriate references including Thibault and Pichard.[1] We are therefore grateful to Dr Jolobe for his comment and its further highlight of the importance of monitoring minerals and micronutrients including, of course, copper post-bariatric surgery.

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Sanatoria revisited: sunlight and health

The intriguing proposal by Greenhalgh and Butler[1] to restore sunlight in treatment of tuberculosis deserves serious consideration and might appeal to the people of the relatively sun deprived nations of Great Britain and Ireland. However, one doubts if it would have similar appeal to patients and doctors of some sun satiated nations like Australia, where, for instance, in the state of Queensland, invasive melanoma is 3–5 times higher than in Europe and the USA in the 15–24 age group.[2]

Greenhalgh and Butler rightly refer to the disruptive downside of the sanatorium regime. This included social isolation, which was frequently referred to in conversation with this correspondent some three decades ago by long term survivors of radical surgery for tuberculosis, a practice which preceded and overlapped with the introduction of chemotherapy.[3] The hazards of sun exposure are widely appreciated and Auguste Rollier, the Swiss pioneer of sanatorium treatment, introduced tuberculosis patients to sunlight in a gradual way, as noted by Greenhalgh and Butler. Sometimes forgotten results of sun exposure in patients with sarcoidosis (a disease which can be difficult to distinguish from tuberculosis) are hypercalcaemia , acute pancreatitis ,and soft tissue calcification, and physicians advise patients with sarcoidosis to avoid sunlight or excessive exposure to sunlight.[4,5,6]

The sanatorium regime per se does seem a thing of the past from the point of view of disruption of patients' personal, working or student lives, as well as the issue of building and staffing such units, which would be best sited as part of multidisciplinary hospitals. However the use of sunbeds as adjunctive therapy to the chemotherapy regime may be a more convenient and much less expensive option. One also has the impression that in this less authoritarian era, and with the growth of patient advocacy groups, some

tuberculosis sufferers would not agree to prolonged hospital confinement even with the rise of extensively resistant forms of this disease. Ironically, a month after publication of Greenhalgh and Butler's paper, in its budget the government of Ireland, concerned about the health hazards of sunlight referred to above, increased the VAT on sunbed services from 13.5% to 23%.

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In response to: 'Thyroid hormone replacement – a counterblast to guidelines'

Dr Toft's paper[1] describes our paper from 2010[2] as being flawed; we think results may be over-interpreted but not flawed. In our paper, people on thyroid replacement with a TSH of less than 0.1 mU/L had increased risk of cardiovascular disease and fractures after 4.5 years follow-up. Most of the 17,684 patients only took L-thyroxine, but patients on liothyronine were included. Additionally, our paper showed the risks of having a TSH between 0.1–0.4 were low,[2] allowing clinicians to make a judgement on the risks of prescribing thyroid replacement with a serum TSH in this range, even though some people may describe this as 'over-treatment'. We never claimed to distinguish between those on thyroxine and liothyronine, or to use serum T4 or T3 measurements as predictors, or to address symptoms. These are valid research questions requiring further studies, but do not invalidate the association of adverse outcomes with a TSH < 0.01 mU/L.[2]

More recently we looked at the safety of patients taking liothyronine alone or in combination (n = 400), compared to patients only taking L-thyroxine (33,955), followed up for 9 years.[3] There was no additional risk of atrial fibrillation, cardiovascular disease or fractures, although there was an increased incident use of antipsychotic medication during follow up. Patients had serum TSH titrated according to current guidelines,[4] although again data on serum T3 were not available.

Furthermore, we believe that Dr Toft should be proud of his and the College's pioneering role in the use of evidence-based guidelines. However, guidelines need to be used fairly and wisely. Evidence-based guidelines describe a foundation of knowledge, but should not be the ceiling of clinical practice. They allow confident clinical practice where there is high quality evidence, but make clinicians and patients aware of uncertainty when evidence is lacking. For example, further clinical trials for thyroid replacement are required, which address appropriate clinical issues. Far from restricting the development of evidence-based guidelines, we need to be educated to use them constructively and to ensure they are not misinterpreted or misused by people who do not have a patient-focused agenda.

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