

Right extended hemicolectomy, falls and low B12

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Right extended hemicolectomy, falls and low B₁₂

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Abstract

Background: Falls are common and can be multifactorial.**Settings:** An older man presented with a fall and an unremarkable past medical history including a right extended hemicolectomy for a caecal carcinoma and a left hip replacement.**Results:** Clinically he had a marked peripheral neuropathy and low serum cobalamin.**Conclusion:** The removal of the terminal ileum may remove the site of uptake of vitamin B₁₂ even in the presence of intrinsic factor. Therefore, older patients in whom curative surgery for colonic carcinoma has been undertaken should be monitored for B₁₂ deficiency five years following surgery and until death. B₁₂ may be replaced in the usual fashion in such individuals

A 83 - year - old man presented following a fall which resulted in lacerations to the face requiring plastic surgery input. He was otherwise well with an extended hemicolectomy for a caecal carcinoma 18 years earlier and a left elective hip replacement for osteoarthritis, seven years previously. He was a non-drinker on no medication. Clinical examination showed a marked peripheral neuropathy in both hands and feet with signs of diminished pressure, vibration and light touch sensation particularly in the lower limbs bilaterally.

His MCV was 104 fl with a haemoglobin of 102 g/dl. His serum cobalamin was < 20 pg/ml. Folic acid and iron studies were normal, anti-IF antibody test was negative as was his anti-gastric parietal cell antibody test (GPC).

The neurological signs were likely to be secondary to B₁₂ deficiency in the absence of any other abnormal finding. His vitamin B₁₂ dietary income was adequate and his liver function tests were normal. In the absence of any gastric pathology or the use of PPIs or antacids it seemed likely that the vitamin B₁₂ deficiency was not related to intrinsic factor but more likely to be due to removal of the terminal ileum during the extended hemicolectomy. The terminal ileum is the site of uptake of vitamin B₁₂ in the presence of intrinsic factor. Having crossed the brush border, the vitamin B₁₂ dissociates from the intrinsic factor and enters the circulation where it binds to transcobalamin II and haptocorrin which are then responsible for the delivery of cobalamin to peripheral tissues and the liver respectively [1-3].

It must be remembered when taking a history from an older patient that surgery even twenty years previously may have unexpected late complications such as almost unrecordable B₁₂ levels resulting in a marked peripheral neuropathy and falls [4]. Treatment was commenced with hydroxycobalamin daily for five days and folic acid. Six months after commencing therapy his peripheral neuropathy was reduced but not fully resolved. He had however had no further falls. Although bacterial overgrowth can also cause low B₁₂ post hemicolectomy, the

patient denied any diarrhoea and there was no biochemical evidence of malabsorption [5]. The delay in manifestation of B₁₂ deficiency is due to the large amounts of B₁₂ stored in the liver and therefore any reduction in vitamin B₁₂ for whatever mechanism may take five to ten years to manifest clinically.

Learning point for clinicians

- Falls in the presence of neurological signs may reflect B₁₂ deficiency.
- Previous colonic surgery may remove the terminal ileum - the site of B₁₂ absorption.
- Treatment of B₁₂ deficiency may improve but not ameliorate symptoms and this may be dependent on how long the deficiency has remained undetected.

Conflicts of interest

None declared.

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