CASE REPORT

Dietary vitamin B12 deficiency in an adolescent white boy

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Dietary deficiency of cobalamin resulting in tissue deficiency in white individuals is unusual. However, several patients with dietary deficiency who were neither vegan nor Hindu have been described. This report describes the case of a 14 year old boy who was a white non-Hindu with a very low intake of cobalamin, which was not apparent until a detailed dietary assessment was performed. The patient responded rapidly to a combination of oral and parenteral B12. This case illustrates the fact that severe dietary vitamin B12 deficiency can occur in non-Hindu white individuals. Inadequate dietary content of B12 may not be apparent until a detailed dietary assessment is performed. This patient is likely to have had subclinical vitamin B12 deficiency for several years. Increased vitamin B12 requirements associated with the adolescent growth spurt may have provoked overt tissue deficiency.

Dietary deficiency of B12 is thought to be rare in the UK and is most often found in strict vegetarians, particularly religious Hindus.^{1,2} We report the case of a previously fit and well 14 year old white boy who presented with a two week history of jaundice, lethargy, anorexia, nausea, and weight loss. There had been no change in colour of stools or urine, no history of bruising, abdominal, or bone pain. There was no history of recent travel abroad and no infectious contact. The patient's mother remarked that he had always been a "picky eater". His father was a butcher. No family history of autoimmune disease including pernicious anaemia or coeliac disease was noted. On examination he was pale and icteric but was apparently well nourished and was on the 25th centile for height and weight. Cardiovascular and respiratory examinations were normal. No lymphadenopathy or organomegaly was palpable. A thorough neurological assessment demonstrated no abnormalities.

Investigation revealed a haemoglobin of 7.90 g/litre, a mean cell volume (MCV) of 117 fL, and an absolute reticulocyte count of 20×10^9 /litre. The platelet count was reduced at 119×10^9 /litre, but the total white cell count was normal at 5×10^9 /litre, with a normal differential—neutrophils being 3.4×10^9 /litre. The blood film showed oval macrocytosis, anisocytosis, poikilocytosis, and hypersegmented neutrophils. Examination of the bone marrow aspirate confirmed megaloblastic haemopoiesis and the presence of giant metamyelocytes.

Serum vitamin B12 was undetectable at < 60 ng/litre (normal range, 180–1132); red cell folate was moderately reduced at 136 mg/litre; serum folate was 3.6 mg/litre (normal range, 3.1–12.4); serum ferritin was 244 mg/litre (normal range, 30–275). An autoantibody screen including anti-intrinsic factor, antigliadin, anti-endomysial, and antiparietal antibodies was negative. A Schilling test part 1 was normal; that is, 33.9%

of a test dose of vitamin B12 was excreted in the absence of added intrinsic factor (normal $> 10\%$).

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There was no evidence of nutritional deficiencies. Coagulation screening was normal, as were serum albumin, alkaline phosphatase, calcium, phosphate, and zinc. Both wrists were x rayed and showed no evidence of rickets.

DIETARY ASSESSMENT

In the initial history, the patient's mother described him as a "picky eater" but there was no suggestion that he was a vegan. On closer questioning it emerged that his diet consisted of white bread, margarine, crisps, chips, jelly, sweets, coca cola, and chocolate milk. A formal dietary assessment was performed. A prospective seven day record of all meals eaten at home was compiled. The amounts of each dietary constituent consumed weekly were calculated and compared with standardised population reference nutrient intake values (RNI) using the Microdiet for Windows computer program. Analysis revealed that the patient's diet contained adequate calories but a very limited intake of foods of animal origin, with no regular vitamin B12 intake and a folate intake of 66% of RNI (fig 1).

The diagnosis was confirmed as megaloblastic anaemia secondary to an isolated dietary deficiency of vitamin B12.

MANAGEMENT

Following confirmation of the diagnosis of dietary vitamin B12 deficiency, he was started on a course of six weekly injections of 1000 µg of vitamin B12. Oral vitamin B12 containing supplements were prescribed later. He and his parents were advised of the adjustments necessary to his diet to include vitamin B12 containing foods and that lifelong adherence to this normal diet was necessary. Four days after the initial injection the reticulocyte count rose to 349×10^9 /litre. His haemoglobin increased at a rate of 10 g/litre/week and the MCV normalised within four weeks. The platelet count returned to the normal range within 21 days.

DISCUSSION

Dietary vitamin B12 deficiency most often occurs in vegans who abstain from meat for religious reasons.^{1–3} Of the 13 cases of dietary B12 deficiency reported by Stewart *et al*, only one patient was neither a vegan nor a Hindu.⁴ However, in a prospective study of 106 patients with a low serum cobalamin value from the north of England, 10 patients had a normal

Abbreviations: MCV, mean cell volume; RNI, reference nutrient intake

Figure 1 Graph of nutrient values as per cent of reference nutrient intake (RNI). Dietary assessment revealed adequate caloric intake but negligible vitamin B12 content in the patient's diet. Folate intake was 66% of RNI.

Schilling test and a low dietary intake of vitamin B12, none of whom were Hindu or vegan.⁵ The inadequate dietary intake was ascribed to alcohol abuse in three and poverty in two. One patient lived with a vegan. Nine of the 10 patients were women and all were adults. None of the patients was a Hindu or vegan. In all cases the cause of the low serum cobalamin was unexplained until a detailed dietary assessment was carried out.

"There has been a dramatic reduction in the consumption of red meat since 1987 and a concomitant increase in the consumption of chicken and pork, which contain less cobalamin"

We have found no reports of a patient as young as 14 years old with such a severe deficiency. Ideally, we would have confirmed the diagnosis by observing the response to oral vitamin B12 alone. However, patients with severe dietary B12 deficiency do not respond to oral B12 alone, presumably because of megaloblastosis of the intestinal mucosa. Therefore, the patient was treated initially with parenteral vitamin B12. However, the normal Schilling test rules out the other likely causes of deficiency, such as pernicious anaemia and isolated intrinsic factor deficiency. The detailed dietary assessment confirmed the diagnosis.

The main dietary sources of vitamin B12 are meat, liver, fish, cheese, and eggs. Recent trends in food consumption in the UK are likely to make dietary B12 deficiency more common. There has been a dramatic reduction in the consumption of red meat since 1987 and a concomitant increase in the consumption of chicken and pork, which contain less cobalamin.⁶ Against this background and during periods when parental supervision is more difficult, as it is during adolescence, we speculate that dietary deficiency of vitamin B12 may become more common in white children and young adults. The inadequate dietary intake of vitamin B12 may not be apparent until a detailed dietary assessment is performed. Overt tissue deficiency may present during periods of increased requirement for vitamin B12 such as pregnancy or, as in this case, the adolescent growth spurt.

Take home messages

- Severe dietary vitamin B12 deficiency usually occurs in Hindus who have a vegan diet
- However, this case shows that it can occur in non-Hindu white individuals, and may not be apparent until a detailed dietary assessment is performed
- With the recent changes in diet in the UK, vitamin B12 deficiency is likely to become more common in white children and young adults, especially during periods of increased requirement for vitamin B12

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