SCIENTIFIC REPORT

Vitamin A deficiency in patients with a remote history of intestinal surgery

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Background/aims: Vitamin A deficiency, often presenting with nyctalopia, has been described in a number of patients with malabsorption as a result of intestinal bypass surgery and, more recently, bariatric surgery. In these reports vitamin A deficiency developed within several years of gastric or intestinal surgery. Three patients who developed decreased vision from vitamin A deficiency more than 18 years after their intestinal surgery are reported.

Methods: A retrospective review of the clinical findings of all patients diagnosed with vitamin A deficiency, as confirmed by serological testing, over the past year in a single neuro-ophthalmic practice.

Results: Four patients with vitamin A deficiency were seen, three of whom had intestinal surgery more than 18 years before the development of visual symptoms.

Conclusion: The authors suggest that vitamin A deficiency should be suspected in patients with unexplained decreased vision and a history of intestinal surgery, regardless of the timing of the surgical procedure.

Vitamin A deficiency is the leading cause of childhood blindness in the developing world and is caused by malnutrition.¹ In contrast, vitamin A deficiency has been relatively rare in developed countries where it is largely the result of malabsorption or abnormal metabolism.² Vitamin A is a fat soluble vitamin absorbed through the small intestine either as retinol or carotene, and then converted to retinyl palmitate which is stored in the liver. Normally the liver contains a 2 year store of vitamin A in the form of retinyl palmitate which can be hydrolysed to retinol, bound to retinol binding protein, and transported to target tissues. In the retina, retinol serves as the backbone of photosensitive pigment in both rods and cones, and on the ocular surface it is necessary for epithelial cell RNA and glycoprotein synthesis. As a result, vitamin A deficiency has a wide range of ocular manifestations including conjunctival and corneal xerosis, keratomalacia, retinopathy, visual loss, and nyctalopia, or night blindness, which is the earliest and most common symptom.^{2–3}

Vitamin A deficiency, often presenting with nyctalopia, has been described in a number of cases of malabsorption as a result of intestinal bypass surgery.^{4–8} With the growing popularity of bariatric surgery for morbid obesity, vitamin A deficiency is becoming increasingly common and increasingly recognised in clinical practice.^{6–9} In these reports vitamin A deficiency developed within months to a few years after gastric and intestinal surgery. We report three patients who developed decreased vision from vitamin A deficiency more than 18 years after their intestinal surgery. We suggest that vitamin A deficiency should be suspected in patients with unexplained decreased vision and a history of intestinal surgery, regardless of the timing of the surgical procedure.

MATERIALS AND METHODS

We reviewed the records of all patients diagnosed with vitamin A deficiency in a single neuro-ophthalmic practice over a 1 year period from January through December 2005. Four patients were noted with decreased vision from vitamin A deficiency, all confirmed with blood testing. One woman had gastric bypass surgery several months before the onset of symptoms and was excluded from this report. Details of the three other patients are given below.

CASE REPORTS

Patient 1

A 69 year old man presented with 4 months of night blindness. Medical history included intestinal bypass surgery 20 years earlier, childhood hepatitis, and possible cirrhosis. Ocular history included childhood strabismus treated with patching for 1 year. He reported no family history of night blindness or retinal disorders. Best corrected visual acuity was 20/60 right eye and 20/50 left eye. He identified eight of 11 Ishihara pseudoisochromatic colour plates with the right eye and 10 of 11 with the left eye. Humphrey 24-2 automated perimetry showed decreased sensitivity in a superior and inferior arcuate pattern bilaterally. Pupils were briskly reactive without an afferent pupillary defect. Slit lamp examination was remarkable for a Bitot's spot in the left eye (fig 1), and funduscopy was normal, without any evidence of drusen or white dots. Laboratory studies revealed vitamin A deficiency with a retinol level of <0.06 mg/l (normal 0.30–1.20 mg/l), retinyl palmitate level of 0.00 mg/dl (normal 0.00–0.10 mg/dl), and a retinol binding protein level of 1.0 mg/dl (3.0–6.0 mg/dl). He was treated with intramuscular vitamin A supplementation. He reported an improvement of vision in both eyes within the first week of treatment but did not return for follow up assessment. He was seen 1 week after treatment by another ophthalmologist who noted that the Bitot's spot had resolved.

Patient 2

An 80 year old man presented with 4 months of decreased vision in the right eye that was worse in dim illumination. He had undergone cataract surgery in the right eye that improved his visual acuity but he continued to report decreased vision in dim illumination. Medical history included Crohn's disease with partial small and large bowel resection for ruptured ileum at 44 years of age and end stage renal disease. His father had congenital colour deficiency but there was no other family history of visual loss. Best corrected visual acuity was 20/25 right eye and 20/40 left eye. He was only able to see the test plate of the Ishihara pseudoisochromatic colour plates with each eye. Automated perimetry showed bilateral superior and inferior arcuate defects. He had a small right afferent pupillary defect. Slit lamp examination showed a posterior chamber intraocular lens on the right and nuclear sclerosis on the left. Funduscopy revealed bilateral optic disc drusen greater on the right, without evidence of

Figure 1 External photograph of the left eye of patient 1 illustrating a Bitot's spot.

drusen or white dots in the retina. Laboratory testing was consistent with vitamin A deficiency with a retinol level of 0.09 mg/l (normal 0.30–1.20 mg/l), retinyl palmitate level of 0.00 mg/l (0.00–0.10 mg/l), and retinol binding protein level of 3.9 mg/dl (normal 3.0–6.0 mg/dl). He was treated for vitamin A deficiency with 9 days of intramuscular injections and then 24 000 IU per day orally until follow up 6 weeks later. He noted visual improvement within days of initiating treatment. Visual acuity remained stable at 20/25 right eye and improved slightly to 20/30 left eye. The afferent pupillary defect in the right eye was no longer seen. Colour vision improved to seven of nine plates right eye and eight of nine plates left eye. Visual field testing, slit lamp examination, and funduscopy remained unchanged.

Patient 3

A 79 year old woman noted several months of decreased vision in each eye that was worse at night. Medical history was significant for stroke 3 years earlier and cholecystectomy, with complications resulting in multiple abdominal surgeries, 18–20 years before. She had undergone cataract surgery in the left eye 1 year before presentation with visual acuity of 20/25 postoperatively. At the time of presentation best corrected visual acuity was counting fingers at 1 foot right eye and 20/50 left eye. She identified only the test plate of the Ishihara pseudoisochromatic colour plates with each eye. Confrontation visual fields suggested a right homonymous hemianopia as had been noted at the time of her stroke. Her pupils were brisk without a relative afferent pupillary defect. She had a dense cataract on the right and a posterior chamber intraocular lens on the left. Funduscopy revealed myelinated nerve fibres on the right, a small chorioretinal scar adjacent to the fovea on the right, and bilateral soft drusen throughout the fundus. Laboratory testing revealed vitamin A deficiency with a retinol level of <0.06 mg/l (normal 0.30–1.20 mg/l), retinyl palmitate level of 0.00 mg/l (0.00–0.10 mg/l), and retinol binding protein of 1.7 mg/dl (normal 3.0–6.0 mg/dl). The patient refused vitamin A replacement therapy and did not return for follow up assessment.

DISCUSSION

Vitamin A deficiency is a relatively rare disorder in the United States.² However, our report illustrates that without a high degree of suspicion vitamin A deficiency from chronic malabsorption may go unrecognised. There have been several case reports of nyctalopia and vitamin A deficiency in patients with a history of gastric or intestinal bypass 3–12 years before presentation.^{4–8}

The three patients described in our report had a more remote history of intestinal surgery, 18–36 years before presentation, and developed vitamin A deficiency despite taking oral vitamin supplementation, although none was being specifically treated for vitamin A deficiency. None of our patients had other clinical evidence of systemic vitamin A deficiency such as pneumonia. In patients 1 and 2, vitamin A deficiency from chronic malabsorption may have been compounded by co-morbidities such as inflammatory bowel disease and cirrhosis but patient 3 had no other known aetiology of vitamin A deficiency. Though Crohn's disease could in theory contribute to malabsorption, in a study of 137 patients with Crohn's disease retinol levels were found to be normal.¹⁰ In patient 2, cirrhosis may have exacerbated the underlying deficiency by reducing hepatic stores and the production of retinol binding protein. A study of fat soluble vitamin deficiency in bariatric surgery patients demonstrated that vitamin A deficiency progressively worsens as long as 4 years postoperatively despite vitamin supplementation.⁹ Although the World Health Organization recommends oral treatment with 200 000 IU per day for vitamin A deficiency, we chose to give intramuscular injections of vitamin A in patients 1 and 2 because we were concerned that the oral form would not be absorbed as well and that potentially the visual symptoms might respond more quickly. It is possible that the visual recovery that occurred in these two patients may also have occurred with the oral formulation of vitamin A.

Our study emphasises that even a remote history of intestinal bypass surgery, especially in the setting of co-morbidities such as liver disease, should raise the suspicion of vitamin A deficiency.

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