involvement. A diagnosis of bilateral anterior uveitis secondary to brimonidine was made and she was treated with 2 hourly topical dexamethasone 0.1% and cyclopentolate 1% thrice daily to both eyes. Both timolol and brimonidine were discontinued and the intraocular pressure initially managed with oral acetazolamide. The inflammation settled within 3 weeks and timolol was restarted without any side effect. Systemic evaluation comprising full blood count, urea and electrolytes, liver function tests, serum angiotensin converting enzyme, ESR, CRP, syphilis serology, and chest X-ray were all normal.

The patient was understandably reluctant to consider a subsequent rechallenge test with topical brimonidine, feeling that it posed a threat to her already limited vision. After 1 month, she restarted brimonidine to the right eye but presented after only three instillations with florid conjunctival injection and an anterior chamber flare in the right eye. There were no cells and the intraocular pressure was normal. The left eye remained quiet. The challenge was discontinued and the conjunctival reaction settled quickly on topical steroids.

Comment

Goyal and Ram[2] reported granulomatous uveitis occurring in one patient in association with topical brimonidine therapy in this journal. Byles *et al*[1] also reported four cases of granulomatous anterior uveitis occurring after 12 months of brimonidine use. In all cases, the uveitis settled rapidly after withdrawal of the drug and reoccurred on rechallenge testing, in the absence of other causes for uveitis. The authors observed that brimonidine, nevertheless, fails to meet the suggested criteria for establishing causality of adverse events by drugs.[3] These include the reaction being frequently described and well documented. Our case lends additional weight to the evidence for brimonidine causing granulomatous anterior uveitis. We feel it is likely that the rechallenge test was negative in our patient because brimonidine was only instilled on three occasions before being discontinued.

Allergic reactions to brimonidine are well recognised, usually consisting of allergic contact dermato-conjunctivitis or follicular conjunctivitis, with reported rates of up to 25%.[4,5] Cessation of treatment at this stage, usually after 6–9 months of therapy, may mean that many patients stop using topical brimonidine before anterior uveitis arises at a later stage. Byles *et al* proposed that anterior uveitis may be more likely to occur in patients who continue to instil brimonidine after an allergic reaction has developed. This was almost certainly the case in our patient in whom the new observation of iris nodules may reflect the chronicity of uveitis arising from continued administration of brimonidine.

We reported our case of granulomatous uveitis as a suspected adverse reaction to topical brimonidine to the Committee on Safety in Medicines via the UK adverse drug reactions reporting scheme. They had no record of any previous reports despite there being published cases. We feel it is important to bring any further cases to the attention of the Committee on Safety in Medicines as well as the ophthalmic literature in order to establish the incidence of this potentially sight-threatening side effect.

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Sir,

Bitot's spots following hemicolectomy

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Vitamin A deficiency, a known complication after small bowel bypass surgery, is rare after large bowel bypass surgery. We report a case of Bitot spots on the conjunctiva

secondary to vitamin A deficiency following right hemicolectomy.

Case report

An 81-year-old man, a known case of bilateral lower lid ectropions and bilateral age-related macular degeneration (ARMD), was referred to the eye clinic complaining of bilateral sore eyes for the last 4 months. He had undergone right hemicolectomy for Dukes B adenocarcinoma of ascending colon approximately 2.5 years ago, and since then was experiencing sporadic diarrhoea. His current medical prescription included oral loperamide and topical viscotear gel.

On ophthalmic examination, his best-corrected visual acuity was 6/18 in the right eye and 6/36 in the left eye. An anterior segment examination revealed foamy plaques on the bulbar conjunctiva temporal to limbus (Figure 1). The Schirmer test was within normal limits. There was diffuse punctate Rose Bengal staining of the conjunctiva. The rest of the anterior segment was normal. Dilated fundus examination showed bilateral dry ARMD. Anterior segment findings were consistent with vitamin A deficiency. When questioned, the patient did not complain of any night blindness. Dark adaptometry was however not performed. Serum levels of fat-soluble vitamins were performed. The serum vitamin A levels were 0.1 μmol/l (range 1.5–2.5) and vitamin E levels were 4.3 μmol/l (range 12.0–28.0). Other investigations, including FBC, U&E, LFTs, serum lipase, lipid profile, clotting time, vitamin B_{12}, folic acid, and ultrasound of the abdomen, were all normal. He was commenced on oral vitamin A supplements 5000 IU three times a day. On examination, the Bitot's spots were still present at a follow-up appointment after a month when conjunctival biopsy was performed. This showed hyperkeratosis, parakeratosis, and chronic inflammatory infiltrates in conjunctival epithelium consistent with those seen in Bitot's spots. At 3 months following the commencement of the oral vitamin A supplements, the serum vitamin A levels became normal and the Bitot's spots regressed completely in 6 months time.

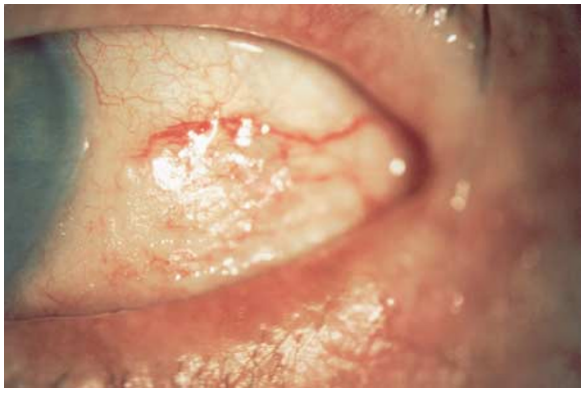

Figure 1 Anterior segment photographs showing Bitot's spots.

Comment

Vitamin A is a fat-soluble vitamin that is absorbed in the terminal ileum. Bile and lipase play an important role in the absorption of vitamin A across the intestinal wall. Once absorbed, it is stored mainly in the liver, where it provides a ready supply to serum. The liver store in a well-nourished healthy adult may last up till 3 years.[1] A true vitamin A deficiency state results only after the vitamin stores in the liver are depleted completely. Although the main cause of vitamin A deficiency worldwide is malnutrition, in developed countries the cause is either malabsorption or inadequate storage.

Vitamin A deficiency has been reported in various gastrointestinal disorders, including Crohn's disease,[2] cystic fibrosis,[3] primary biliary cirrhosis,[4] Jejuno ileal bypass surgery,[5] and hemicolectomy.[6] The clinical manifestations of vitamin A deficiency include night blindness, xerophthalmia, and follicular hyperkeratosis of the skin. Xerophthalmia ranges from mild conjunctival dryness to keratomalacia.

Impaired dark adaptation or night blindness is the earliest manifestation of the disease, but it is seldom noticed until it has become very pronounced.[7] Conjunctival changes occur long before those in the cornea and clinically appear as lustreless conjunctiva. Occasionally, the conjunctiva in addition to being xerotic exhibit foamy plaque on the bulbar conjunctiva known as Bitot spots. Histologically, Bitot's spots show hyperkeratosis, parakeratosis, and loss of goblet cells.

There are only two previously reported cases of vitamin A deficiency after hemicolectomy.[6,8] Both cases had night blindness as the only ophthalmic manifestation. One of the cases[8] had radiation enteritis as the main cause of vitamin A deficiency. The case here is unusual because he presented with Bitot's spots as the sole clinical manifestation of vitamin A deficiency. The patient might have poor dark adaptation, but when questioned he denied any night blindness. This may, of course, be due to his already poor vision because of ARMD. As a result of his old age and poor vision, dark adaptometery was not performed.

This patient developed vitamin deficiency 2.5 years after right hemicolectomy, which indicates that his presumed diet was healthy and his liver functions were normal.[1] There was no clinical or biochemical evidence of generalized malabsorption or liver and pancreatic disease. In the case where a short bowel resection has been performed, the cause of vitamin deficiency is well

understood because of the removal of the absorptive surface. The cause of vitamin deficiency in hemicolectomy is uncertain, but it may be related to decreased transit time through the small intestine owing to gut hypermotility. Chronic diarrhoea and the continued use of laxatives have been implicated as the cause of vitamin deficiency.[9]

Our patient did have chronic diarrhoea since the operation, and we feel that gut hypermotility was the cause of his vitamin A deficiency. The reason why the patient only had a fat-soluble vitamin deficiency is that, in mild intestinal absorption disorders, it is the fat-soluble vitamins that are least well absorbed.[10]

In summary, this is the first reported case of Bitot's spots following hemicolectomy.

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Sir,

Bilateral transient amaurosis following *Mycoplasma pneumoniae* infection: a manifestation of acute disseminated encephalomyelitis

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Mycoplasma pneumoniae is an atypical bacterium that can cause a great variety of respiratory infections and can be responsible for ocular involvement such as conjunctivitis, anterior uveitis, and very rarely optic neuropathy. We report a case of bilateral vision loss with optic disc swelling that developed 4 days after starting treatment for atypical pneumonia and was accompanied by multiple demyelinating brain lesions.

Case report

A 24-year-old male presented as an outpatient with a 2-day history of deterioration of vision in both eyes, headache, stiff neck, and general malaise. Owing to febrile illness accompanied by herpes labialis, he had been taking amoxicillin for a week without improvement. An infectologist diagnosed atypical pneumonia 4 days before ophthalmological examination and administered clarithromycin 500 mg b.i.d. The patient had been without fever for the preceding 3 days.

On examination, his unaided visual acuities (VA) were counting fingers (CF)/2 m right, 0.25 left. Pupillary reactions were diminished and relative afferent pupillary defect (RAPD) was absent. Fundoscopy revealed swelling of both optic discs with few splinter haemorrhages peripapillary and mildly dilated veins. Optic papillitis was diagnosed, however, because of the history of fever and meninigeal signs present, he was referred to an infectologist. Laboratory tests and a lumbar puncture were carried out. Serology was positive for *M. pneumoniae* (IgG >200 IU/ml; range 20–30 IU/ml, IgM 149 IU/ml, range 13–17 IU/ml), negative for *Chlamydia pneumoniae*, *Chlamydia psittaci*, *Chlamydia trachomatis* and *Legionella pneumophila*, Herpes simplex, Varicella Zoster and Epstein–Barr virus. A lumbar