Vitamin A

Intestinal surgery a villain?

W B Lee, I R Schwab

You need A vitamin

Vitamin A is an essential fat soluble vitamin that must be obtained solely from diet or vitamin supplementation. Normal vitamin A levels are required for appropriate functioning of our immune system, skin, retina, and ocular surface. Without adequate levels of vitamin A in the body (hypovitaminosis A), serious systemic consequences can occur, including significant ophthalmic complications leading to potential vision loss and ultimate blindness if the deficiency is not recognised and corrected. Hypovitaminosis A remains a major health problem of epidemic proportions worldwide with over 127 million people and 4.4 million preschool children affected. Vitamin A deficiency remains the leading cause of preventable blindness in children with an estimated half a million children rendered blind each year; yet even more alarming is many of those children die of the same disease.[1] While the major cause of hypovitaminosis A across the world remains malnutrition, Chae and Foroozan in this issue of *BJO* (p 955) have astutely reminded us that iatrogenic vitamin A deficiency from surgical alteration of the small intestine, the site where vitamin A absorption occurs, can create any number of ophthalmic findings as seen with a malnourished vitamin A deficient patient.[2] This problem has global implications as developed countries, which have typically been spared malnutrition and vitamin A deficiency, are now at risk for the same co-morbidities affecting less developed countries. Yet the difficulties in the developed world are different and are not a result of inadequate food supplies. Because hypovitaminosis A remains a relatively rare condition in the developed world, it is often either misdiagnosed or not suspected by physicians, particularly ophthalmologists.[3]

Chae and Foroozan performed a retrospective review of the ophthalmological findings in three patients diagnosed with hypovitaminosis A over the course of 1 year in a neuro-ophthalmic practice. They describe three patients presenting with decreased vision, which after history, examination, and laboratory testing, was attributed to complications from vitamin A deficiency. Each of the three patients had a remote history of previous intestinal surgery spanning from 3 years to 12 years before presentation including one patient with gastric bypass surgery, one patient with intestinal bypass surgery, and one patient with multiple unspecified abdominal surgeries. All cases were attributed to malabsorption of vitamin A, which typically occurs in the duodenum of the small intestines. Postsurgical anatomical changes created a disturbance in the normal physiological absorption of vitamin A, a finding suspected by the authors to potentiate the late ocular manifestations in each case. The ophthalmic findings of the three patients included nyctalopia and abnormal colour vision testing in all patients and a Bitot spot in one patient. While either an alternative or even a combination of deficient vitamins could have possibly led to the development of vision loss in these cases, hypovitaminosis A was confirmed by serology in each case and treated in two patients with intramuscular vitamin A supplementation. The authors conclude that vitamin A deficiency should be suspected in any patients presenting with nyctalopia and/or generalised decreased vision if the medical history includes previous intestinal or gastric bypass surgery, regardless of whether the surgery was performed in the remote past.

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The first case described by the authors in which hypovitaminosis A occurred following gastric bypass surgery deserves special discussion. The authors also briefly mention a fourth patient who developed decreased vision from hypovitaminosis A following gastric bypass surgery. While this patient was excluded from the study because of a shorter time between onset of vision changes and gastric bypass surgery, the finding of two patients with visual complications following gastric bypass surgery raises concern for a procedure that is gaining wide popularity across the globe.

Developed countries across the world have seen a steep rise in obesity prevalence resulting from the increased availability of food, the cultural emphasis on fatty foods, insufficient nutritional education, and the persistent failure of medical therapy for obesity including weight loss programmes of diet and exercise modification. With the increased failed attempts of weight loss regimes, many countries have seen astounding increases in obesity including countries like the United Kingdom, where obesity prevalence nearly tripled between 1980 and 2002, and the United States, where obesity prevalence in adults aged 20 years or older doubled and the number of overweight children and adolescents tripled from 1980 to 2002.[4 5] In fact, in just five short years from 1999 to 2004, prevalence estimates in the United States ranged from 16.5% to 17.1% for obese adolescents and children and 30.4% to 32.2% for obese adults. An additional obesity prevalence report from China saw an increase in obese preschool children from 1.5% in 1989 to 12.6% in 1997.[5]

As self restraint disappears, fast food restaurants proliferate, and nutritional education fails to alter the inexorable march towards obesity, patients have increasingly turned to operative solutions such as bariatric surgery in an attempt to achieve a ''quick fix.'' Bariatric surgery has seen an astounding increase in popularity, perhaps best seen in the United States, where 16 200 gastric bypass procedures were performed in 1992 compared to nearly 170 000 cases performed in 2005.[6] As with any major surgical procedure, complications must always remain a concern. A particularly new concern with gastric bypass surgery is the risk of hypovitaminosis A from malabsorption resulting in ophthalmic complications, as described by Chae and Foroozan. In fact, several reports have recently described severe visual complications from this new syndrome including blindness.[3 7] A recent study looking at vitamin deficiencies after bariatric surgery found deficiencies in all fat soluble vitamins, including an alarming rate of vitamin A deficiency seen in 52% of patients 1 year after surgery and 69% of patients 4 years after surgery despite

oral vitamin supplementation.[8] When the remarkable rise in gastric bypass surgical procedures is considered across the world, the realisation of a potential epidemic syndrome of iatrogenic vitamin A deficiency becomes an alarming reality. It is essential that eye care professionals not only recognise and appropriately treat this new syndrome with vitamin A supplements, either the oral (preferably liquid) or intramuscular routes, but also provide education to our colleagues in other fields of medicine to ensure appropriate counselling and early recognition in the hope of preventing this potential epidemic syndrome of gastric bypass surgery and iatrogenic blindness.

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Brain damage in children

Visual problems as a result of brain damage in children

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Affected children include those with cerebral palsy, who may or may not have learning difficulties, and those with profound brain damage causing cerebral blindness

Damage to the brain is the commonest cause of visual impairment in children in developed countries.[1] Improved survival of premature infants has increased the prevalence of periventricular leucomalacia, and greater success in managing profoundly ill children has led to increased survival of children with meningitis, encephalitis, and hypoxic ischaemic encephalopathy. Hydrocephalus is now also successfully treated, while congenital disorders of the brain add to the prevalence of brain dysfunction.[2] On the other hand, early treatment of cataract, the successful management of glaucoma, screening for retinoblastoma, and effective immunisation for rubella have all decreased the prevalence of blindness in children as a result of eye disorders.

A large proportion of the brain serves visual function. The cerebral cortex, underlying white matter, and (during early visual development) the basal ganglia[3] all play a major part. The classic view is that the visual system comprises the anterior visual pathways, the lateral geniculate bodies, the optic radiations, and the occipital cortices wherein the process of ''vision'' takes place. Disruption of these pathways may result in restricted visual fields and impairment in visual acuity. The article by Lowery *et al* in this issue (p 960)[4] highlights the importance of having a high index of suspicion when a child presents with undiagnosed poor visual function. Cerebral (or cortical) visual impairment may well be the cause.

In addition to unexplained impairment of visual acuity in a child, what features can lead the clinician to suspect that there is brain damage affecting the visual system? In our experience there is a range of features (table 1) that may accompany impaired acuities and restricted visual fields, or that may even occur in isolation, in a child with brain damage affecting vision.[5]

Primary visual processing takes place in the striate cortex. The analysis of motion takes place more anteriorly. Impaired ability to discriminate the movement characteristics of different animals has recently been shown to be a sensitive marker for periventricular leucomalacia affecting vision.[6]

The fusiform gyri of the inferior temporal lobes, on both sides, ostensibly act as an image store for the panoply of imagery encountered. If the incoming data from the occipital lobes match what is already known, recognition takes place; if not, the new information is learned from. This visual pathway is known as the ventral stream. A child with cerebral visual impairment and good acuities may mistake a stranger for a parent, if the ventral stream is dysfunctional. Another function of the ventral stream is orientation and navigation. Children with ventral stream dysfunction can easily become lost.[7]

The posterior parietal lobes and the accompanying dorsal stream pathways from the occipital lobes serve a number of fundamental visual functions. The *British Journal of Ophthalmology* of 1918 contains two seminal articles by Holmes.[8 9] The first delineates the structure of the visual pathways by relating the location of discreet shrapnel wounds to the resultant visual field loss. The second highlights the functions of the posterior parietal lobes by giving clear descriptions of the visual features resulting from bilateral damage. The affected soldiers had lower visual field loss. They could only attend to (and therefore see) one or two items in their visual scene, and despite having intact stereopsis in a number of cases, they were all unable to use vision to guide movement.

The functions of the dorsal stream pathways comprise the analysis of the complexity of the visual scene, the ability to accord selective visual attention to specific elements, the ability to suppress other elements so that they do not distract, and the handling of other incoming data such as hearing and touch. The dorsal stream also serves the function of determining the visual coordinates of the elements within the visual scene. This informs the motor cortex to facilitate visually guided movement of the body, and the frontal eye fields to bring about rapid eye movement to view the object of interest. Disorder of this complex process is commonly seen, in