Table 1 Results of newspaper survey

Total newspapers, $n = 57$
No mention of Transit of Venus, $n = 34$
Mentioned Transit of Venus, $n = 23$
No warning against looking directly at sun, $n = 15$
Warning against looking directly at sun, $n = 8$
No safe viewing method suggested, $n = 3$
Safe viewing method suggested, $n = 5$
<i>Suggested safe viewing method</i>
Pinhole projection, $n = 5$
Other Image projection, $n = 2$
'Eclipse glasses', $n = 4$
Solar Filter, $n = 2$
Website referral, $n = 1$

the event.² Safe methods for observing solar events include a pinhole or other image projection system, 'eclipse glasses', or other appropriate solar filter.

Acknowledgements

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Sir,

Acute irreversible diabetic cataract in adolescence: a case report

Acute metabolic cataract in patients with newly diagnosed Type 1 diabetes mellitus is a rare complication, which may develop within a few weeks or months after starting treatment.^{1,2} It can affect visual acuity from slight visual impairment to complete blindness.¹ We report a case of acute bilateral irreversible metabolic cataract in a patient with recently diagnosed Type 1 diabetes mellitus.

Case report

An 18-year-old Caucasian female presented with a 4-week history of feeling unwell; a 2-month history of thirst and polyuria; and a 1-year history of weight loss. There was no significant past medical history. Her body mass index was 24.3. Urinalysis revealed moderate ketones and glucose but she was not acidotic. Her fasting plasma glucose level was 22.3 mmol/l (normal range: 3.3–5.5 mmol/l) and HbA1C was 10.5% (normal range: 4–6%). Urea, electrolytes and arterial blood gases were within normal limits. She was diagnosed as having Type 1 diabetes mellitus, and started on Novomix™ 30–26 U in the morning and 28 U in the evening.

After 1 week of treatment, she noticed blurring of vision in both eyes, which gradually worsened. She was referred to the Ophthalmology Department. There was no history of previous eye problems and visual acuity was 6/6 in both eyes at the start of treatment. On presentation visual acuity was only perception of light in both eyes. Slit-lamp biomicroscopy revealed dense intumescent cortical cataracts bilaterally (Figures 1 and 2). No fundal details were visible in either eye. She was diagnosed as having bilateral acute irreversible metabolic cataract and cataract surgery was planned. She underwent right phacoemulsification with an acrylic posterior chamber intraocular lens (PCIOL) implantation (Sensar® OptiEdge) under general anaesthesia (GA) and her visual acuity improved to 6/5 without glasses. After 2 months, left phacoemulsification and PCIOL implantation (Sensar® OptiEdge) under GA was performed and her visual acuity improved to 6/6 without glasses, although she required +2.5 for near vision. There were no changes of diabetic retinopathy seen.

After 3 months, left posterior capsular opacification developed and her visual acuity reduced to 6/9. Left YAG posterior capsulotomy was performed to improve visual acuity.

On her last visit the visual acuity in both eyes is 6/5 and there is no diabetic retinopathy.

Figure 1 Anterior segment photograph of right eye showing dense cortical cataract. There is no fundal glow visible due to density of cataract.

Figure 2 Anterior segment photograph of left eye showing dense cortical cataract and no view of fundus.

Comments

The exact mechanism of diabetic cataract is not known although it is thought to be related to poor glycaemic control and abnormalities in the polyol pathway.³ Reduction of glucose to sorbitol by aldose reductase (AR) leads to accumulation of sorbitol, which produces osmotic stress. It also produces oxidative stress by depleting cofactor NADPH that is an important cofactor for regeneration of reduced glutathione (GSH).³ Some authors also mention the probable importance of genetic factors in their case studies but the mechanism is still poorly understood.⁴

Our patient was started on insulin and just after a week, noticed blurring of vision. It deteriorated very quickly to perception of light within 4 weeks. Her blood glucose level was 22.3 mmol/l and it came down

rapidly to 10 mmol/l with insulin treatment. The rapid change in blood glucose following initiation of insulin may be responsible for the development of metabolic cataract.⁵ The cataract was of cortical snowflakes type and the whole lens was swollen in both eyes. Acute metabolic cataract can either be transient resolving spontaneously with improved glycaemic control or irreversible.

Phacoemulsification cataract surgery with intraocular lens implantation has a good visual outcome but is challenging in intumescent cataracts, especially there is a high risk of peripheral radiation of capsulorhexis or capsulotomy because of swollen lens. We used Sensar® OptiEdge IOL, which has sharp posterior edge and it is well known to reduce posterior capsular opacification.⁶ In our patient, left posterior capsular opacification developed earlier as early posterior capsular opacification is a well-known postoperative complication in diabetic patients.⁷

Young patients with diabetes mellitus can present with eye problems during the early course of the disease and treatment.⁸ These cataracts once developed can be irreversible in spite of good metabolic control and require surgical intervention.

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Sir,

Familial Arnold-Chiari Type I malformation

Arnold-Chiari Type I malformation (CMI) is a congenital malformation of unknown incidence. It occurs in a small posterior fossa and constitutes 3–5 mm herniation of the cerebellar tonsils into the spinal canal.¹ In all, 25% of patients have cerebrospinal fluid displacement into the spinal cord canal, causing syringomyelia.^{1,2}

Familial clustering of CMI with and without syringomyelia has been documented in American scientific literature but this case of siblings appears to be the first instance reported in the UK.

Case report

Case 1: A 49-year-old gentleman presented to ophthalmic outpatients in 1996 with an 8-month history of horizontal diplopia, vertigo, left arm and thigh pain, a change in gait, and occipital headaches. The patient smoked 20 cigarettes/day, but there was no past medical and, at that stage, no known family history.

Snellen acuity was 6/9 right (amblyopia), 6/6 left. Examination demonstrated a small right esotropia with limited abduction of the right eye. Horizontal nystagmus with a vertical component on right gaze, and reduced convergence were present. There was no sign of optic nerve dysfunction. Humphrey visual fields were full. Intraocular pressures were 15 bilaterally and fundoscopy was normal.

Neurological examination demonstrated broad-based gait and a positive Romberg's test. Tone and reflexes were normal.

Routine blood tests and vitamin B12 and folate levels were normal, and VDRL/TPHA screen was negative. Initial MRI scan was normal and differential diagnoses were scan-negative demyelinating disease, Arnold–Chiari malformation, basilar invagination, and paraneoplastic syndrome.

In late 1997, the patient developed oscillopsia and had increasing difficulty walking. Ophthalmic examination further revealed downbeat and rotatory nystagmus in all gaze positions.

Repeat MRI in 1997 demonstrated Chiari Type I malformation, the cerebellar tonsils having herniated to the posterior arch of C2 (Figure 1a and b).

The patient underwent foramen magnum decompression in early 1998 with marked symptom resolution. He presented to ophthalmology again in mid-1998 with recurrence of diplopia and had a small right VI nerve palsy on examination. Further MRI showed no recurrence of hindbrain disease. Following extraocular muscle surgery 1 year later, the patient has remained asymptomatic.

Case 2: Aged 52 years, the elder sister of the aforementioned patient presented to neurologists in 1995 with intermittent shaking and weakness of the left hand. She was an asthmatic on bronchodilators; nothing else was significant medically. Neurological examination and CT brain scan were normal, the history was felt to be suggestive of focal motor seizures and the patient was commenced on Phenytoin 100 mg tds.

In 1996, the patient developed left thenar eminence fasciculation and in 1998 described upper limb paraesthesiae. Examination revealed hypertonic lower limbs, hyper-reflexia, right extensor plantar response, and decreased pin prick sensation in the C2–C8 distribution. Power remained full throughout.

MRI scan showed extensive syringomyelia, from C1 to the mid-dorsal region with associated Chiari Type I malformation, the cerebellar tonsils having herniated to the arch of C1 (Figure 2a and b).

In early 1999, the patient underwent foramen magnum decompression with good symptom resolution. She later became ataxic and developed an upper limb tremor.

Repeat MRI scan in 2003 demonstrated that despite satisfactory decompression, the caudal end of the syrinx remained. The patient awaits further neurosurgical intervention.

Comment

The first reported case of familial CMI was in the United States in 1990, involving siblings aged 18 months and 2