Although rare in patients with diabetes, acute-onset cataract is not uncommon in type 1 diabetes, especially in children, and gives a typical ‘snowstorm’ appearance. Although usually associated with chronic hyperglycaemia, it may also occur on rapid restoration of euglycaemia. Cataract may also occur with exposure to medications such as steroids, barbiturates, phenothiazines and diuretics. It may also occur in Cushing’s syndrome. Early detection of diabetes and adequate glycaemic control, particularly in female adolescents, may prevent this debilitating complication of diabetes.

Case histories

Case 1

A 56-year-old woman with well-controlled hypertension on hydrochlorothiazide 12.5 mg daily was referred to an ophthalmologist by her general practitioner because of fluctuating visual acuity of 2 months’ duration, which led to frequent changes in spectacle prescriptions. An ophthalmological examination demonstrated bilateral early nuclear sclerosis with minimal cortical lens changes. Visual acuity was 6/120 in the right eye and 6/36 in the left eye. In the right eye, uncorrected visual acuity (UCVA) was 3/60 (or 6/120), and best corrected visual acuity (BCVA) 6/9. Lens-induced myopia was present with a refraction of -3.50/-0.75x19. In the left eye, UCVA was 6/36, BCVA 6/9 and lens-induced myopia -2.75. At this stage diabetes was diagnosed on the basis of a fasting plasma glucose (FPG) level of 26.7 mmol/l. Ketonuria was absent. Despite the introduction of gliclazide 160 mg daily and metformin 1 g daily in addition to diet modification, she had persistent polydipsia, polyuria, significant weight loss and paraesthesiae in her hands and feet, prompting her referral to the diabetes service at the University of Cape Town. On examination she was found to be overweight (body mass index (BMI) 29.5 kg/m²), without features of Cushing’s syndrome, but with evidence of peripheral sensory neuropathy. The glycated haemoglobin (HbA1c) level was 19.2% (target range < 7%), FPG 13.4 mmol/l (target range < 6 mmol/l), total serum cholesterol 6.3 mmol/l (normal reference < 5 mmol/l) with a dyslipidaemic profile, triglycerides 1.98 mmol/l (normal reference < 1.5 mmol/l), low-density lipoprotein (LDL) cholesterol 4.8 mmol/l (normal reference ≤ 2.6 mmol/l) and high-density lipoprotein (HDL) cholesterol 1.4 mmol/l (normal reference > 1.2 mmol/l). The oral hypoglycaemic agents were substituted with intermediate premixed insulin (Actraphane) 18 units daily, 12 units before breakfast and 6 units before supper, and blood glucose levels normalised within 3 days. There was a dramatic decrease in visual acuity within 24 hours of commencing insulin therapy. Urgent ophthalmological review revealed a visual acuity of less than 6/120 in both eyes, such that the patient could only count fingers. She had bilateral dense cataracts with cortical and post-subcapsular opacities. Normoglycaemia was maintained despite

Two patients, an adult female with newly diagnosed diabetes and an adolescent female with type 1 diabetes of 3 years’ duration, presented with acute-onset bilateral cataracts. Normal vision was restored to both eyes after the cataracts had been extracted successfully.

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the insulin doses being reduced to 12 units daily (8 units before breakfast and 4 units before supper). Bilateral cataract extraction with insertion of intraocular lenses was performed a month later as there was no resolution of the cataracts or improvement in visual acuity. Postoperative visual acuity was as follows: right eye – UAVA 6/6, refraction -0.25; left eye – 6/6, refraction plano. The patient remains well with normoglycaemia on 8 units of insulin daily.

Case 2
A 17-year-old girl with diabetes of 3 years’ duration presented with bilateral, progressive and complete loss of vision over a period of 1 month. She admitted to poor compliance with diet and insulin therapy (Actraphane 17 units before breakfast and 16 units before supper). She had had no antecedent trauma, life-threatening diarrhoea, or renal failure, and no exposure to drugs known to be associated with cataracts, such as steroids, barbiturates, phenothiazines and diuretics. There were no features to suggest chronic hypocalcaemia, or a family history of cataracts. The HbA1c level was 15.7% and FPG 26.1 mmol/l. About 9 months earlier the HbA1c and FPG levels had been 17.3% and 12.4 mmol/l, respectively, indicating prolonged poor control. The patient was underweight, with a body weight of 38 kg and a BMI of 14.48 kg/m². Ophthalmological assessment revealed dense bilateral mature cataracts. The cataracts did not resolve, nor did the vision improve, despite a reduction in glycaemic levels, with blood glucose profiles in the range of 6.2 - 13.3 mmol/l. One month after presentation she had successful bilateral cataract extractions and intraocular lens implant insertions with subsequent restoration of vision. She was discharged on actraphane 64 units daily, 34 units and 20 units before breakfast and supper respectively, with improvement in HbA1C.

Discussion
These cases demonstrate that acute-onset cataracts can occur in adults and adolescents with diabetes. The condition may antedate the diagnosis of diabetes, as in the first case, or it may develop after the diagnosis of diabetes as illustrated by the 17-year-old patient. The condition is usually bilateral, although a unilateral presentation has been reported. Cataracts or lens opacifications may be transient and reversible with good glycaemic control or progressive and irreversible despite achieving euglycaemia. The course of transient and reversible cataracts has been noted to range from noticeable improvement in vision to complete resolution of cataracts within 5 weeks, as described in a 62-year-old man with acute-onset bilateral cataracts at the diagnosis of diabetes. With effective glycaemic control and a stable serum glucose level, visual acuity improved from finger counting at 2 feet with the right eye and 20/63 vision in the left eye, to 20/20 in both eyes. In the case of a 52-year-old man...
who developed cataracts within 5 days of initiating insulin therapy for severe hyperglycaemia, cataracts resolved completely within 7 weeks.

Chronic hyperglycaemia may induce cataracts and this is consistent with the finding that 2 of our patients had high HbA1c levels. While long-term use of diuretics for blood pressure control in the first diabetic patient may have contributed to cataract formation, it is noteworthy that there was no exposure to corticosteroids. There were no features suggestive of Cushing’s syndrome in either patient, as this can lead to the development of diabetes and cataract formation. As reported, rapid normalisation of glycaemia may result in cataract formation; our type 2 patient illustrated this in that there was worsening of bilateral cataracts with improved glycaemic control on initiation of insulin therapy.

Acute cataracts are a rare complication of childhood-onset diabetes. Montgomery and Batch reported only 9 subjects with cataracts in their cohort of 1 319 type 1 diabetes subjects. Not all were of acute onset. Two cases had cataracts on diagnosis of diabetes, one 3 weeks after the diagnosis of diabetes, and the remainder (N = 6) presented 1.7 - 13 years after diabetes was diagnosed. Subjects with congenital causes of cataracts such as rubella syndrome and trisomy 21 were excluded from their analysis.

The factors associated with cataracts in young persons with diabetes include high HbA1c levels, adolescence and female gender. A female preponderance has been suggested in various case series on acute cataracts and newly diagnosed cases of type 1 diabetes. Both patients reported in our case report were female, but the incidence in our hands is too low to make generalisations about gender.

The postulated mechanisms for the development of cataracts include non-enzymatic glycation of lens proteins, oxidative stress and hyperosmotic stress due to excessive sorbitol formation and accumulation in the lens from increased polyol pathway activity. Sorbitol also produces oxidative stress by depleting co-factor NADPH (reduced nicotinamide adenine dinucleotide phosphate), which is an important co-factor for regeneration of reduced glutathione. It is important to note that glucose entry into the lens is independent of insulin action, therefore hyperglycaemia may potentiate the aforementioned mechanisms of cataract formation. A role for genetic predisposition has been suggested by the finding of cataracts in identical twins at diagnosis of diabetes.

Cataracts in the young person with diabetes may be ascribed to mitochondrial disease, as described in a 12-year-old Japanese girl. Mitochondrial disease may result in MELAS (mitochondrial myopathy, encephalopathy, lactic acidosis and stroke-like episodes) or mitochondrial diabetes, and cataracts. The second patient in our series was not obviously deaf. Additionally, no genetic studies were undertaken to exclude mitochondrial disease. Acute-onset bilateral cataracts may also be associated with neuropathic cachexia. Although the second patient in our series was cachectic, she had no symptoms or signs to suggest peripheral sensory or motor neuropathy, a prerequisite for the diagnosis of diabetic cachexia. While the first patient had peripheral neuropathy she was not cachectic.

We were unable to definitively classify the type of diabetes in our patients as auto-antibodies were not measured. While the second patient was lean, she did not demonstrate ketosis as she had received sufficient insulin to prevent a ketotic state, as is frequently seen with type 1 diabetics. Although the diagnosis of type 2 diabetes is suggested by the features of the metabolic syndrome in patient 1, latent autoimmune diabetes of adults (LADA) may be considered as euglycaemia was rapidly achieved and maintained, using small doses of insulin.

In conclusion, acute-onset visual loss from cataracts is an unusual and occasionally reversible manifestation of diabetes. The temporal association of cataract formation and rapid introduction of tight glycaemic control in some patients, a phenomenon previously reported, is perhaps a reason for advocating gentle reduction in levels of hyperglycaemia.