Hypocalcaemic cataract as a presenting symptom of renal insufficiency

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Summary

A young man of 17 years presented with bilateral reduced vision because of cataracts. Investigations showed a low level of serum calcium along with other evidence of renal failure. The cataracts were mainly in the posterior pole (cupuliform); faint peripheral cortical opacities were also present—in the form of about a dozen half-loops straddling the equator and extending about half way towards the axial centre of the lens.

KEY WORDS: renal hypoplasia.

Introduction

This patient is reported because visual loss due to hypocalcaemic cataracts was the presenting symptom of renal failure. Although hypocalcaemia is common in renal failure, only three cataracts secondary to hypocalcaemia have been described to our knowledge (Berlyne, Danovitch and Ben-Ari, 1972; Stein and Godel, 1980). This may be partly because the hypocalcaemia is expected and treated, and partly because a duration of hypocalcaemia of around 6-12 months seems to be required to produce these cataracts (Berlyne *et al.*, 1972). It is rather surprising that cataracts are not more common in renal failure since uraemia, hypertension and other cataract-inducing conditions co-exist (Clayton *et al.*, 1980), albeit for a relatively short time. These potential adjuvants may explain the different morphology of this patient's cataracts from others.

Case report

A young white male aged 17 years attended with a complaint of poor vision in both eyes for about 9

months. He had had no serious illnesses, exposure to drugs, injury or radiation and there was no family history of poor vision. His parents were not consanguineous.

Examination suggested abnormal skin pallor but his mother said this was normal for him and that his general health was always excellent. Visual acuity was 6/18 (right) and 6/24 (left) because of bilateral posterior cortical, cupuliform-pattern cataracts. There were also about a dozen half-loops of faint peripheral cortical opacities straddling the equator and extending half way towards the centre of the lenses. These characteristics are morphologically unlike those of congenital cataracts, although, in any case, symptoms of visual impairment had started only 9 months previously, and the opacification was subsequently progressive.

At first, diabetes mellitus was suspected but urine was free from sugar on several occasions. Following the suspicion of hypocalcaemia, the following abnormal results were obtained: plasma calcium 1.25 mmol/litre (normal 2.12-2.62); phosphate 3.5 mmol/litre (normal 0.8-1.4); creatinine 1,000 µmol/litre (normal 55-150); blood urea 44.5 mmol/litre (normal 2.5-6.6); creatinine clearance 6 ml/min (normal 80 ml/min); alkaline phosphatase 13 KA units (normal 10 KA units); haemoglobin 6 g/100 ml (normal 13-18 g/100 ml).

A diagnosis of chronic renal failure, possibly due to hypoplastic kidneys, was made. There was no evidence of soft tissue calcification on X-ray in spite of the severe hyperphosphataemia.

Nine months after presentation, he required haemodialysis and 4 months later he had a cadaver renal transplant, which was lost following rejection after 6 months.

About 6 months after presentation, his visual acuity had deteriorated considerably (6/36 in the right eye and 6/24 in the left) so that a right

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extracapsular cataract extraction was done with 6/4 result. Operation on the left eye was postponed until 4 years later when a similar operation achieved a 6/6 result in the left eye. He is currently well on haemodialysis.

Discussion

It is very rare for an older child or young adult to present with recently developed progressive cataracts; diabetes mellitus would be the usual explanation. Hypocalcaemia due to renal failure is probably less rare than the paucity of previous reports in the literature suggest (Berlyne *et al.*, 1972; Stein and Godel, 1980).

Although iridescent particles occur in hypocalcaemic cataracts (Duke-Elder, 1965; Stein and Godel, 1980), they were not observed in our patient. 'Punctate stippling' in the lenses was observed in about half of a series of 23 patients with renal failure of whom five had serum calcium of less than 6 mg per 100 ml (Berlyne *et al.*, 1972), and in a patient with hypocalcaemic cataract (Stein and Godel, 1980). The

involvement of the posterior pole was prominent in our patient and in the other most recently described patient (Stein and Godel, 1980). The latter may also have had cortical opacities similar to the ones we describe as straddling the equator.

The possible contribution of relative hypocalcaemia to the aetiology of 'senile' cataract (Clayton *et al.*, 1980) will remain an open question until further evidence is accumulated.

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