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Nephrology Dialysis Transplantation

Nephroquiz
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'Stone'-blindness

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Case

A 15-year-old boy with juvenile rheumatoid arthritis (JRA) and end-stage CKD was referred to us for renal replacement therapy. He was previously under follow-up with a paediatric unit. JRA was diagnosed at the age of 3½ years when he developed polyarthritis and uveitis. He received steroids, sulphasalazine, non-steroidal anti-inflammatory drugs, calcium supplements (calcium phosphate) and vitamin D.

Corneal opacities and redness of eye developed 3 years ago and he underwent many sessions of corneal scrapings in a private ophthalmological unit, but was not evaluated further. On evaluation for growth retardation 2 years ago, he was found to have renal dysfunction. A renal biopsy revealed chronic interstitial nephritis. He was initiated on conservative management for CKD.

He was continuing treatment at the private hospital for the eye problem without any significant improvement. He had progressive deterioration in vision, redness and worsening of the opacities which remained refractory to surgical debridement.

On clinical examination in our unit, he had greyish white haziness extending across the cornea (Figure 1A). Biochemical values showed an elevated calcium-phosphorous product (110.16 mg^2/dl^2), renal dysfunction (estimated GFR-6 ml/min/1.73 m^2, CKD stage 5) and suppressed serum parathyroid hormone (23.8 pg/ml).

Questions

(a) What is the diagnosis?
(b) What are the other aetiologies?
(c) What may be the precipitating factor in this patient?
(d) How can this be treated?

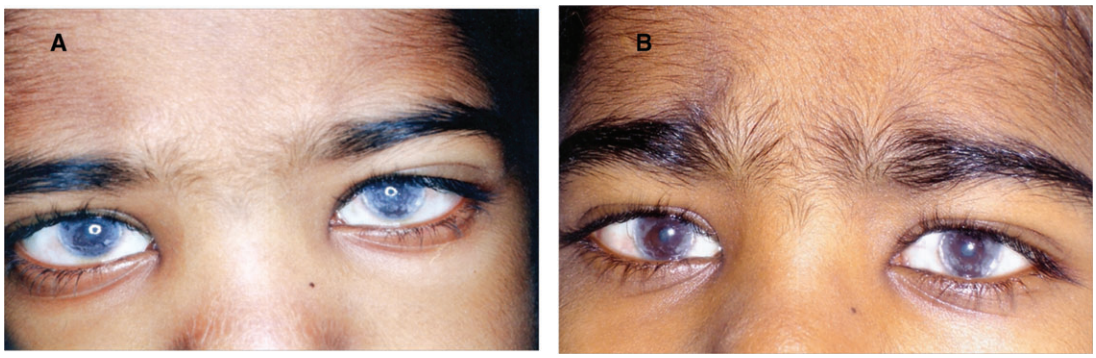

Fig. 1. (**A**) Dense Corneal opacity at first visit. (**B**) Improvement in corneal opacity after renal transplantation.

Answers

(a) *Band keratopathy*: Greyish white haziness across the cornea in a patient with CKD having high Ca-P product should make one suspect band keratopathy. Band keratopathy is a clinical problem usually encountered by ophthalmologists [1]. It results from precipitation of calcium and phosphate in the interpalpebral region of the cornea. Elevated levels of calcium or phosphorous along with the local pH changes predispose to calcification in the cornea [1].

(b) Vitamin D overdose, hyperparathyroidism and granulomatous conditions such as sarcoidosis are associated with this condition. Other local conditions such as juvenile rheumatoid arthritis with uveitis can also contribute to this problem [2].

(c) Our patient has two reasons to develop the problem-uveitis and the high calcium–phosphorous product. The former would have predisposed to the development of this problem and the latter should have aggravated the condition. Continuation of the calcium phosphate preparation along with vitamin D after the onset of renal disease would have worsened the scenario. He received ophthalmological care at a different hospital where he did not disclose the details of his treatment and continued taking calcium salts. Calcium salts chelate dietary phosphate and hence many calcium salts such as calcium acetate and carbonate are commonly used for phosphate binding [3]. Calcium phosphate and citrate, however, should be avoided for this purpose. Calcium–phosphorous product when it exceeds 55 mg^2/dl^2 may result in ectopic calcification.

(d) Dialysis was initiated. Calcium and vitamin D tablets were stopped. Non-calcium phosphate binder was introduced. After restoring normal calcium phosphate product, corneal debridement with ethylenediaminetetraacetic acid (EDTA) was continued which resulted in partial recovery of vision [4]. Renal transplantation was performed subsequently which hastened the recovery (Figure 1B).

Conflict of interest statement. None declared.

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