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Case Report

# Peripheral ulcerative keratitis Our challenging experience

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## Abstract

A 52 year old male presented with peripheral ulcerative keratitis in the right eye. Patient's history included retinitis pigmentosa, pseudophakia (right eye), cataract (left eye), bilateral partial deafness, ischemic heart disease, hypertension, type 1 diabetes mellitus, depression, hyperparathyroidism, hypertriglycemia and renal failure. The patient was on weekly hemodialysis. The peripheral corneal ulceration remained stable until he developed sudden and rapid thinning after eight months of regular follow up and management. Laboratory investigations including immunological studies were negative and we had to rely on treatment based on clinical signs, including the visual acuity, size, depth and staining of the ulcer and perilimbal, episcleral, scleral, corneal and anterior chamber reactions. The patient was treated with medical and conservative approaches and the eye was protected with a plastic shield to avoid injury. Despite our efforts, the patient perforated his eye due to a trivial trauma during sleep. He was managed successfully with cyanoacrylate glue and a bandage contact lens. The anterior chamber reformed after the perforation was sealed and the patient is on a regular follow up with a multidisciplinary approach.

**Keywords**: Peripheral ulcerative keratitis, Immune complex deposition, Peripheral corneal thinning, Impending perforation, Topical cyclosporine eye drops, Connective tissue diseases, Amniotic membrane transplantation


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## Introduction

Peripheral ulcerative keratitis is a rare condition with the potential for significant visual morbidity and potential loss of the eye. The presentation and management can be complicated by underlying diseases such rheumatoid arthritis (RA), wegener' granulomatosis (WG), polyarteritis nodosa (PAN), systemic lupus erythematosus (SLE), sarcoidosis, hepatitis B and C may also be present [1–3]. Often, management of peripheral ulcerative keratitis involves a multidisciplinary approach due to the existing co-morbidities. Initial treatment is usually medical followed by surgical intervention, including amniotic membrane transplantation[10] or tectonic keratoplasty.[11,14]

In this case report we present a challenging management of a patient with peripheral ulcerative keratitis, highlighting our management strategies. This case report presents the treatment in the scenario of multi-systemic disorders and several resource constraints.

### *Case presentation*

A 52 year old Saudi male, with a known history of retinitis pigmentosa and partial deafness since childhood, presented

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to our department last year with complaints of worsening vision in the right eye. He gave a vague history of trauma in the right eye during childhood which was found to be insignificant. He had undergone cataract surgery in the right eye, with an IOL implant, 8 yr ago, at another health facility in Jeddah, Saudi Arabia. He was on insulin for type 1 diabetes mellitus for the last 13 years. His hypertension was controlled with medications for the last 7 years. He also had ischemic heart disease and had undergone coronary artery bypass surgery (CABG) 2 years ago. He was on weekly hemodialysis for chronic renal failure for the last 4 years. He was also known to have hyperparathyrodism and hypertriglycemia and was being managed by a medical specialist. He was using topical preparations for pruritus prescribed by a dermatologist. The patient seemed to be mildly depressed and was referred to the psychiatrist for management.

He was married and had five healthy children. There was no family history of night blindness or deafness in the rest of his family. He was a non-smoker and had no known allergies. For his systemic ailments, he was using caps alfacalcidol 1 OD, tab carvedol 25 mg BD, tab isosorbide dinitrate 10 mg BD, tab asprin 81 mg OD, tab folic acid 10 mg OD, tab sevelamer hydrochloride 800 mg BD, tab simvastatin 20 mg OD, tab ranatidine 150 mg BD, tab gemfibrozol 600 mg BD, tab Ca-Co3 600 mg TDS, tab *methyldopa* 250 mg TDS, tab irbesartan 300 mg OD, tab resperidone 2 mg OD, and injection epoetin alfa 4000 IV once weekly.

On the first ocular examination, last year, the visual acuity was 20/150 SC (not improving with pinhole) and 20/100 SC (20/50 CC). The cornea showed a crescent shaped area of $10 \times 2$ mm thinning with a constant estimated depth of 20%. There was no fluorescein staining. The ulcer was located between 5 and 11 o'clock in the right eye (Fig. 1). The overlying epithelial edge was sloping and not overhanging. No vessels were bridging the defect. There was no corneal infiltrate and the anterior chamber was quiet. The sclera was not involved. The examination of the left eye showed early cataract. Fundus examination revealed advanced retinitis pigmentosa in both eyes. The visual fields were severely constricted (Fig. 2) consistent with retinal findings. Routine phacoemulsification surgery with foldable IOL was planned for the left eye, but was postponed due to delayed fitness by the cardiologist who wanted to see his latest echocardiogram. At 5 month follow up, the right eye and the vision remained relatively stable, with no change in the size and the depth of the initial lesion. Several systemic laboratory tests (basic and immunological) were performed and were all negative.

After nine months from initial presentation, with the patient being asymptomatic on a routine follow up visit, mild episcleritis was noticed at the 9 o'clock position in the right eye. Prednisolone Acetate Ophthalmic Suspension (1%) Q 4 h was prescribed and inflammation was controlled within 10 days. The patient was maintained on topical 0.1% fluorometholone eye drops Q 4 h for another 10 days, then the drops were reduced to Q 12 h for another fortnight. Episcleritis resolved completely and was not present at the subsequent, regular 3 weekly visits.

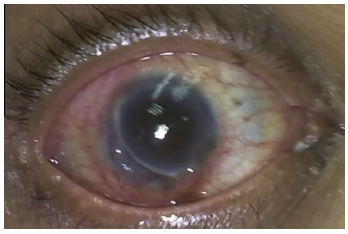

**Figure 1.** Peripheral Ulcerative Keratitis – (Rt) eye.

4 months later, his wife noticed an unusual redness in his right eye. The patient noted some reduction in vision, without any pain, so he chose to ignore it. When his symptoms did not improve over the next three days he presented to our ophthalmic department. On examination, the vision in the right eye was reduced to counting fingers at 1 m. There was an exacerbation in the perilimbal inflammation, associated with further corneal thinning and mild scleritis. There was no infiltrate and the anterior and posterior chambers were quiet, and there was no RAPD. An area of thinning of $0.5 \times 0.5$ mm along the limbus at 7 o'clock involving roughly 70% of depth of the stroma developed suddenly within the next 3 days (Fig. 3). There was no descemetocele and no endothelial folds. Corneal sensations were intact and equivocal in both eyes. The sudden thinning prompted us to postpone the surgery in the left eye until the right eye could be managed and stabilized.

Again, several laboratory investigations were urgently performed and most were negative. The results of the laboratory investigations were: CBC: Hgb: 13.9 g/dl, WBC: $7.89 \times 10^3/\mu l$ (normal), ESR: 34 mm/hr at the end of 1 h, HCT: 39.30% (Normal), RBC: $4.22 \times 10^6/\mu L$ (decreased), MCH: 30.30 pg (normal), MCHC: 32.6 g/dl (normal), MCV: 93.1 fl (normal), PLT: $260 \times 10^3/\mu l$, RDW-CV: 15.8% (11.6–14.4), **Immunological Testing:** Serum R.A. factor: negative (<8 iu/m), serum C-reactive protein (CRP): negative (<0.8 mg/dl), ANA by ELISA: 6.8 units which means negative (range < 20 negative, 20–60 moderately positive, >60 strong positive), serum ANCA: C-ANCA: 4.1 units, P-ANCA: 1.9 units, which means both were negative (range < 20 negative, 21–30 moderately positive, >30 strong positive), serum ACE levels were within normal limits, serum anti-CCP antibody requested by the rheumatologist (normal). **Blood Chemistry:** serum AST: 14 μ/l, serum BUN: 97.5 mg/dl, serum Creatinine: 9.39 mg/dl (↑↑↑), serum glucose (fasting): 119.3 mg/dl, serum K+: 4.97 mmol/L, serum Na+: 139.9, serum VDRL (syphilis serology): negative, Hepatitis B and C Serology: negative; **Chest X-Ray PA view**, no lung lesions were present.

Therefore, our earlier diagnosis of ''idiopathic peripheral ulcerative keratitis (PUK) – (Right Eye)'' was revised to ''idiopathic PUK with impending perforation OD with pseudophakia and cataract OS with advanced RP OU''.

## Management

Initially, we prescribed fluorometholone eye drops (0.1%) Q 12 h or Q 6 h (modified based on the waxing and waning of the injection around the lesion) and lubricants Q 4 h. A close follow-up was kept and the ulcer remained stable. Sizes, depth of thinning and staining and various ocular inflammatory reactions were noted overtime. There was no evidence of uveitis, throughout. Corneal infection/abscess was ruled out repeatedly. Dry eye and blepharitis were not present.

At the time of sudden exacerbation which set in, as mentioned above, a careful history was re-taken and a repeated systemic examination was carried out just in case some signs