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Case Report

Acute onset myopia – A rare presentation of systemic lupus erythematosus

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Introduction

Sudden onset myopia is seen in several local or systemic conditions.¹ Local conditions include blunt ocular trauma and ocular inflammation. Systemic conditions include diabetes, pregnancy and certain drugs like hydrochlorothiazide and topiramate.¹

However Systemic Lupus Erythematosus (SLE) as a cause of transient myopia is so rare that it has not been reported in these articles. Neither has sudden onset transient myopia been included in the ocular manifestations of SLE.^{2,3} A case of SLE which presented with acute myopia is presented herewith.

Case report

An 18-year-old female patient, presented with complaints of sudden diminution of vision of both eyes accompanied by swelling of both the eyelids of 16–18 h. She had no history of previous ocular disease or trauma. The patient also gave a history of multiple joint pains involving the knees, ankle, shoulder and knuckles, history of irregular menses on and off over the past few months along with intermittent fever.

On ocular examination, the visual acuity was 5/60 in the right eye and 6/60 in the left eye with her current spectacles of –2.25 DS in both eyes. She had been wearing corrective glasses since the past five years. Dry refraction as well as automated refraction revealed a refractive error of –6.50 DS in both eyes. The patient improved to 6/12 in the right eye and 6/6 in the left eye with a correction of –6.0 DS. Upper and lower eyelids of both eyes had mild edema (Fig. 1). There was ciliary congestion in both eyes. Anterior chamber appeared shallow with Van Herrick's grade II in both eyes. Direct ophthalmoscopy as well as 90D slit lamp biomicroscopy revealed multiple retinal folds around the macula. Intraocular pressure was 20 mm Hg with applanation in both the eyes. Gonioscopy confirmed that the anterior chamber was shallow (Shaffer's grade I-II in both eyes in all quadrants) (Fig. 4). Ophthalmic A/B Scan ultrasound showed choroidal effusion and a “T”-sign positive. Maximum choroidal thickness was 2.1 mm in the right eye and 2.4 mm in the fellow eye (Fig. 2). Retinal periphery showed no treatable lesion in either eye by indirect ophthalmoscopy.

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Fig. 1 – Edema of upper and lower lid. Bilateral swelling of hands and feet.

The anterior chamber depth (measured by IOL Master) was 3.17 mm and 3.06 mm respectively in right and left eye and corneal thickness was 519 and 507 microns respectively.

General examination revealed a temperature of 99.2 F with a regular pulse rate of 104/min, blood pressure of 134/88 mm Hg right arm supine with pitting pedal edema in both legs. All the joints of both hands and both legs showed symmetrical swelling (Fig. 1). Rest of the systemic examination was unremarkable.

Laboratory examination revealed Hb – 8.7 gm%, TLC – 3200/mm³, Platelets – 1, 50,000/ μ l, Serum Urea/Creatinine – 56 mg/dl/1.2 mg/dl, Serum uric acid – 6.3 mg/dl, Serum Na⁺/K⁺ – 125/4.6 mEq/l. Urine routine and microscopy showed proteins 3+, 2–3 RBCs, numerous WBCs with granular casts with 24 h urine protein being 350 mg/dl. Chest radiograph PA view and abdominal USG were normal. Serum examination for Systemic Lupus Erythematosus was positive for Antinuclear Antibodies (ANA) and ds DNA levels. Renal biopsy showed class IV A lupus nephritis.

For the ophthalmic manifestations the patient was started on 1% Prednisolone and 2% Homatropine eye drops. In view of rapid progression of disease and features of acute renal failure, she was started on intravenous pulse methylprednisolone (55 mg in 200 ml NS over 60 min OD $\times$ 3 days) followed by tablet Prednisolone 50 mg OD (1 mg/kg/day). The patient was managed appropriately by the nephrologist and the rheumatologist for the underlying conditions.

The patient's refractive error worsened in the next two days with the refraction being –7.50 DS both eyes on the second day of treatment. She started showing improvement from the fifth day onwards. The refraction was –5 DS in RE and –4 DS in LE by the 7th day of presentation with an anterior chamber depth (measured by IOL Master) of 3.35 mm and 3.31 mm respectively in right and left eye and corneal thickness was 512 and 498 microns respectively. She was also started on tab Hydroxychloroquine 200 mg BD thereafter.

By the 17th day, the unaided vision improved to 6/24 in both eyes and the refraction was –2 DS in BE. The patient

Fig. 2 – Ophthalmic A/B Scan ultrasound showing choroidal effusion and a positive “T”-sign. Maximum choroidal thickness was 2.1 mm in the right eye and 2.4 mm in the left eye.

Fig. 3 – Periorbital swelling, edema of feet and hands completely resolved by 17th day.

Fig. 4 – Slit lamp photograph on presentation.

improved to 6/6 BE with -2.25 DS correction in BE. Anterior chamber depth increased to 3.58 mm and 3.54 mm in right and left eye respectively and the pachymetry readings were 481 and 484 microns respectively. Intraocular pressure was 10 mm Hg in BE. The corneal curvature remained unchanged throughout the episode. The anterior chamber had deepened to VH grade IV and gonioscopically was Shaffer's grade IV in all quadrants in BE. Periorbital swelling, edema of feet and hands completely resolved (Fig. 3). Ophthalmic A/B Scan ultrasound revealed resolution of the choroidal effusion and funduscopy revealed reduced retinal folds.

Discussion

Keratoconjunctivitis sicca, posterior scleritis with retinal involvement, lupus retinopathy and choroidopathy and optic neuritis are all known causes of diminution of vision in SLE.^{2,3} Acute myopia as the presenting sign in SLE is a rare entity. Only a handful of cases, about 06 so far have been reported where SLE has presented as acute myopia.^{1,4–7} The proposed causes in each were: anterior chamber shallowing and

macular folding, extensive choroidal detachment, anterior displacement of crystalline lenses in two of the cases and forward rotation of the ciliary body.

In this case the mechanism seems to be the forward displacement of the iris lens diaphragm due to choroidal effusion,³ as confirmed the changes in anterior chamber depth. Ciliochoroidal effusion and retinal folds were confirmed on ophthalmic ultrasonography and fundus photograph respectively. Choroidal effusion and retinal folds showed improvement on treatment, which correlated with improving vision. There was also a slight change in the corneal thickness but the cornea was clear and corneal curvature remained the same, which would indicate that the cornea was not a major causative factor. The absence of ocular trauma or a positive drug history further supports this by ruling out other known causes of transient acute myopia.¹

This rare presentation of SLE highlights the importance of early ophthalmic evaluation in a case of SLE. Thus acute transient myopia may be presenting symptom or an early predictor for SLE/lupus nephritis.

Conflicts of interest

All authors have none to declare.

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