

# SEVERE CHEMOSIS IN SYSTEMIC LUPUS ERYTHEMATOSUS — A NEW SIGN?

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## SUMMARY

*A young Chinese female presented with severe bilateral chemosis without any other systemic evidence of oedema or systemic lupus erythematosus (SLE). Investigations confirmed the diagnosis of SLE with early diffuse proliferative glomerulonephritis. The condition improved with steroid therapy.*

## CASE REPORT

A 21 year old Chinese female presented with a rapidly progressive facial swelling of 2 weeks duration. This was not related to time of day or posture and not associated with any ankle swelling or abdominal distension. There was no preceding history of fever, sore throat or joint pains. She denied taking any form of oral medication prior to illness and did not apply any eye drops. There was no change in micturition habits.

Although she noticed the conjunctival oedema

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she was quite definite that there was no preceding redness, pain or itchiness in both eyes and her vision was never affected. However there was slight epihora. She denied having any hair loss or skin rash and there was no history of SLE in the family.

The patient was slightly obese, afebrile and not anaemic. There was no scalp, sacral or pedal oedema. The blood pressure was normal. The striking abnormalities were bilateral periorbital oedema with severe chemoses (Figs. 1, 2 and 3). There was no evidence of conjunctivitis. The visual acuity was normal and funduscopy revealed no abnormalities. There was no skin rash noted and all other systems were essentially normal.

Laboratory investigations on admission showed normal haemoglobin of 13.6 gm% and a total white cell count of 4400 with a normal differential. However the ESR was 109 mm/hr. The serum electrolytes were essentially normal, blood urea 20 mg% and serum creatinine of 0.8 mg% further confirming the normal renal function. The 24 hour urinary protein loss was only 0.4 gm and repeated urinalysis failed to show any significant cells or casts. The total serum protein was however, 5.7 gm% with the albumin lowish at 2.9 gm%. The fasting serum cholesterol was 205 mg% and triglyceride 308 mg%. Immunological investigations were very helpful in deciding the underlying etiology. The LE cells were positive on all three occasions it was done. The complements were extremely low, C<sub>3</sub> = 9, C<sub>4</sub> = 4, and the C-reactive protein was positive. The antinuclear factor titre was 1:80 and the immunofluorescence staining of



Fig. 1 Frontal view of the face showing the marked periorbital puffiness. The conjunctival oedema can just be seen.

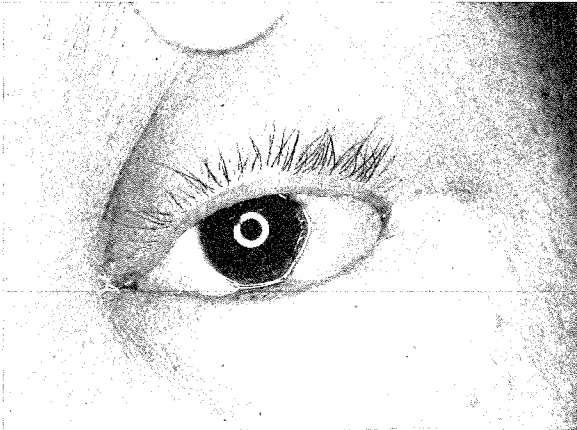


Fig. 2 Close up of the left eye. Notice the marked conjunctival swelling and the epiphora.



Fig. 3 Oblique view to show the anterior protrusion of the conjunctiva as a result of tense oedema.

the skin biopsy specimen showed a positive lupus band.

The intravenous urograph showed bilaterally swollen kidneys and the ensuing renal biopsy confirmed a grade II (Baldwin) diffuse proliferative glomerulonephritis.

The patient was initially given *Maxidex* eye drops but there was minimal improvement. She was then given prednisolone when the diagnosis of SLE was confirmed. Soon after initiation of steroid therapy the malar rash typical of SLE appeared and at the



Fig. 4 After treatment.

same time we noted the progressive disappearance of the chemoses (fig. 4).

Conjunctival involvement in SLE is rare and in most instances are non-specific conjunctivitis.<sup>1</sup> Chemosis alone without underlying inflammatory changes have not been reported. Periorbital swellings and conjunctival oedema can be seen in patient with gross Nephrotic Syndrome where there is generalised fluid retention. In this aspect this patient is odd because there were no gross proteinuria and biochemical evidence to support nephrotic syndrome was lacking. Except for the eye manifestation there was no pedal sacral or scalp oedema.

Clinical signs of systemic lupus erythematosus were not apparent until she was given prednisolone when the butterfly rash and alopecia started to appear. With such odd presentation one begins to wonder in what other manner SLE can manifest.

#### REFERENCE

<sup>1</sup> Watson P G and Hazleman B L (1976) The sclera and systemic disorders, Saunders pg. 240-241.