SUMMARY AND CONCLUSION

SLE is a chronic inflammatory disease of unknown cause, characterised by immune complex formation.

It is more prevalent in females, in urban areas and in black races.

Etiology is unknown it may be attributed to genetic factors, chronic viral infection, hormonal status or auto- immunity. An immunologic profile of a case of SLE includes multiple autoantibodies production, secondary to impaired B-lymphocyte regulation.

The disease may be precipitated by infections, stress, exposure to the sun, or pregnancy and abortion.

The pathogeneis of SLE is due to the deposition of antigen-antibody complex. The autoantibodies may attack an antigen within an organ, or they may bind to cells, like erythrocytes, causing them to be removed from the circulation by the reticulo-endothelial system.

SLE patients experience exacerbations interspaced by periods of relative quiscence. It affects almost all systems of the body.

The diagnosis of SLE relies upon the American Rheumatology Association revised criteria for the classification of SLE.

SLE may affect different structures of the eye. It affects the eye lids by cutaneous rash, or telangiectasia formation. The conjunctiva is involved in keratoconjunctivitis sicca. Corneal affection occurs either in the form of perilimbal punctate staining or deposition of yellow-white material in the deep stroma. Episcleritis and scleritis may also occur. The retina is affected

in different ways;

* Retinal soft exudates.

* Retinal vaso occlusion.

* Retinal microaneurysms. and

* Serous retinal detachment secondary to RPE damage.

Pathogenesis of retinal affection includes immune complex deposition in the vessel wall with subsequent vessel wall necrosis and sheathing. Persistent narrowing of the lumina occurs secondary to chronic endothelial and medial proliferation

Chloroquine, an antimalarial drug, used in the treatment of SLE, may cause pigmentary mottling of the macula (Chloroquine retiropathy), which may progress to the classic Bull's eye configuration.

The prevalence of optic neuropathy among SLE patients is around 1%. The patient may present with:

* Anterior ischaemic optic neuropathy.

* Retrobulbar ischaemic optic neuropathy, or

* Insidious and progressive visual loss secondary to optic neuropathy.

Thirty-six patients were included in the study, SLE activity was quantified using the lupus activity criteria count (LACC). Fifteen patients ran an inactive course during the time of examination, while twenty-one cases had an active disease.

Full ophthalmic examination as well as fundus fluorescein angiography were performed to all patients. Suprathreshold automated perimetry was done to one patient to aid in the diagnosis.

Five patients (13.89%) were found to have retinal affection, in the form of retinal microaneurysms in one patient (2.78%), retinal vaso-occlusion in three patients (8.33%), and anterior ischaemic optic neuropthy in one patient (2.78%.).

One case with retinal affection had an inactive SLE disease, while the other four cases had an active SLE disease, denoting more prevalence of retinal affetion among patients with active course.

Two clinical, findings, active cerebral disease and skin vasculitis, as well as two laboratory findings, positive antiphospholipid antibodies and thrombocytopenia, showed significant statistical correlation with SLE retinopathy.