Nomoto et al. [1] have reported the frequency of scleritis in patients with IgA nephropathy. Scleritis is an inflammatory lesion of the eye, often associated with systemic diseases such as rheumatoid arthritis, periarteritis nodosa, Wegener’s granulomatosis, or with infectious and/or allergic disorders [2].

We report our study of the association of this condition with glomerulonephritis (GN). Scleritis was looked for, in a blind manner, in 53 GN patients and 40 healthy controls. A renal biopsy was obtained in all 53 GN patients. IgA nephropathy was diagnosed by means of immunofluorescence in 30 of them, as primary IgA nephropathy (Berger’s disease) in 27, secondary to anaphylactoid pur-pura in 2 cases, and associated with liver cirrhosis in 1 patient. 4 patients had isolated mesangial deposits of C3 [3]. Minimal glomerular lesions were observed in 5 biopsies, and histological and immunofluorescence examination of the others revealed 4 focal GN, 5 membrano-proliferative GN and 2 extramembraneous GN. Vascular lesions were only found in 3 biopsies from patients with microscopic hematuria.

All 40 healthy controls were free of scleritis at ophthalmic examination, while this lesion was observed in 10 GN patients. Analysis of these results confirmed the report of Nomoto et al. [1]: scleritis was found in 5 cases of IgA nephropathy, and in 3 out of 4 cases of isolated mesangial deposits of C3, a GN considered as very similar to mesangial IgA nephropathies. The last 2 cases of scleritis were observed in completely different patients, one with membranoproliferative GN and one extramembraneous GN.

No known cause of scleritis was found in these positive patients whose ocular lesions may be considered as related to their GN. There was no correlation between the presence of scleritis and the level of serum IgA, but circulating immune complexes activating the alternate pathway of the complement system were present in most of them. These complexes do not bind Clq and were assessed by their ability to fix bovine conglutinin. They were found in the serum of 15 out of 25 IgA nephropathy patients without scleritis (60%), 4 of 5 IgA nephropathy patients with scleritis (80%), and 1 patient with isolated mesangial C3 deposits and scleritis.

Our results (18% of IgA nephropathy patients presenting scleritis) are very similar to those reported by Nomoto et al. [1] (15%). They bring no new data regarding the etio-pathology of
scleritis, but suggest that this condition might occur more frequently in patients presenting circulating immune complexes activating the alternate pathway of complement and, hypothetically, tissue abnormalities favoring the deposition of such complexes. This last point is suggested by the peculiar mesangial localization of IgA and/or C3. Similar conditions might exist in ocular tissues, but the reality of immune complex deposits in this area still needs to be demonstrated.

References