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CASE PRESENTATION – IgA nephropathy

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The patient was a 48 year-old man. He had suffered for 2 days from a sore throat. He had a pink and cloudy discolouration of his urine. He had difficulty in swallowing and was feverish. He did not have dysurai and no increase or decrease in urinary frequency. He had also had discomfort in his left knee for about 36 hours.

He was a non-smoker, but drank about 6 pints of beer per week. He was married but there was no family history of renal disease. He himself had had 3 episodes of glomerulonephritis when aged 14, 21 and 28. Each of these followed pharyngytis. There was nothing else in his personal history.

On examination his temperature was 38.1degrees centigrade. He had no rash, no cyanosis, and no splinter haemmorrhages. His blood pressure was 120/76. There were no symptoms in his cardiovascular system, in his chest, and he had a normal abdomen. On examination of his pharynx, it was found to be inflamed, with enlarged tonsils. As a result of this a preliminary diagnosis of post-streptococcal glomerulonephritis was made.

His haematological tests showed that his haemoglobin was 13.6 g/L his white cell count was increased to 23.7 and his platelet count was 178. His biochemistry tests showed that his sodium was 134 meq/L his potassium was 3.7 meq/L his urea was 7.3, his creatinine was 167, and he had more than 100 red cells per ml and ++ protein in his urine. Ultrasound studies indicated that his kidneys were of normal size and cortical thickness, there was no hydronephrosis and the bladder appreared to be normal. No renal tract calcification was seen on plain X-ray film.

Advice from a renal physician suggested that it could be post-streptococcal glomerulonephritis or possibly something else. He suggested that the fibrinogen level and the prothrombin time be measured; he also suggested that his treatment should be changed to benzylpenicillin. Finally he suggested that the patient should be transferred to the Renal Ward and that a renal biopsy should be done.

The renal biopsy showed that the renal medulla and coretx appeared to be normal, as did the glomeruli. But immunohistochemistry showed that the glomeruli contained small but significant mesangial deposits of immunoglobulin A. As a result the diagnosis was changed to IgA nephropathy.

IgA Nephropathy

IgA nephropathy was first described about 25 years ago. It was suggested that it might be due to a genetic susceptibility and that it caused mesangial proliferation glomerulonephritis. It is one of the most common forms of glomerulonephritis, occurs in children and young people, and gives rise to asymptomatic microscopic haematuria and sometimes proteinuria. About 5% of patients can suffer from the nephrotic syndrome. Usually, however, the prognosis is good.

It is caused by an increase in levels of IgA in the blood, especially due to gastrointestinal inflammation, and especially in those who are genetically predisposed. It can be compared to Henoch-Schonlein purpura. Some claim that steroid therapy is of benefit, especially prednisolone; but meta-analysis indicates that the benefit may not exceed that risks of the treatment. Steroids can be used in those especially at risk.

It is stated that approximately 20% may develop renal failure. But complete remission can occur after about 10 years in over 30% of children who develop the disease.

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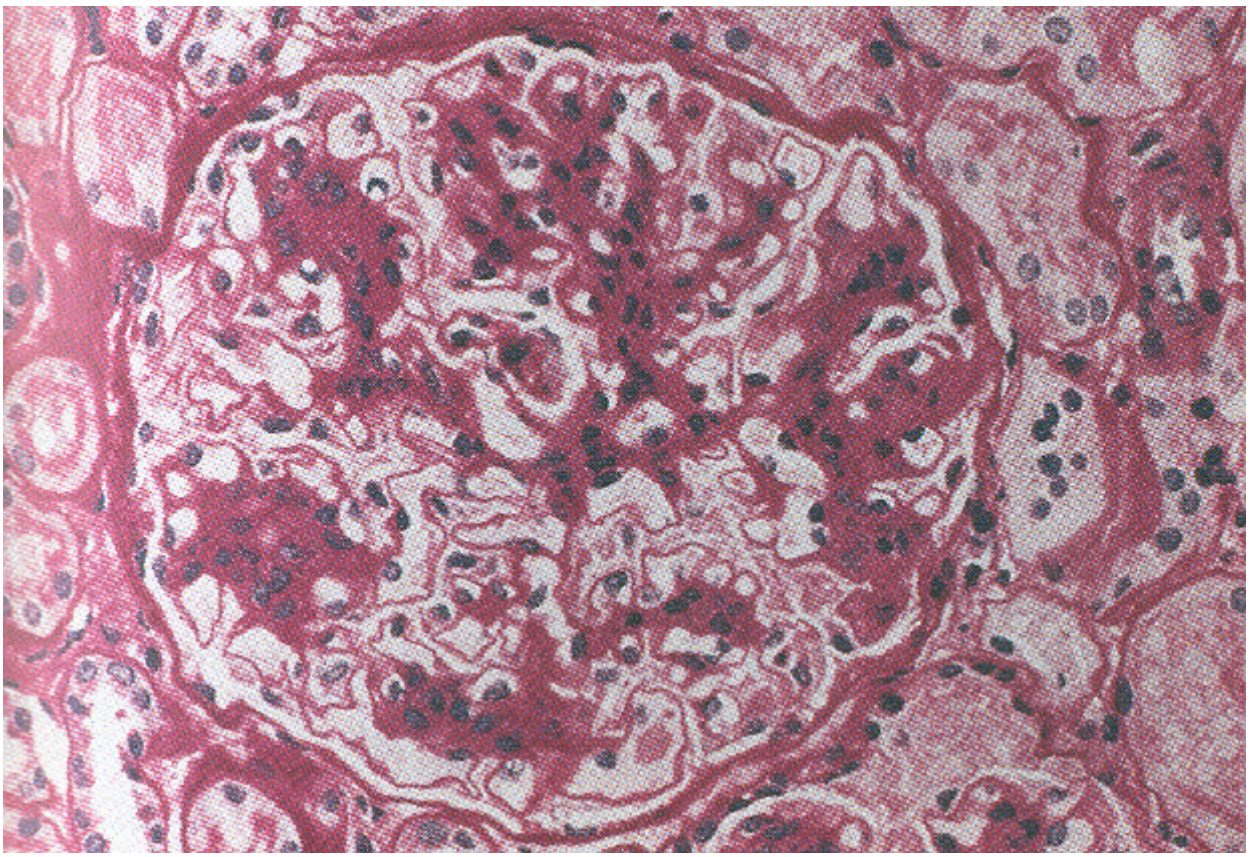

Figure 1 – Appearance of the glomeruli

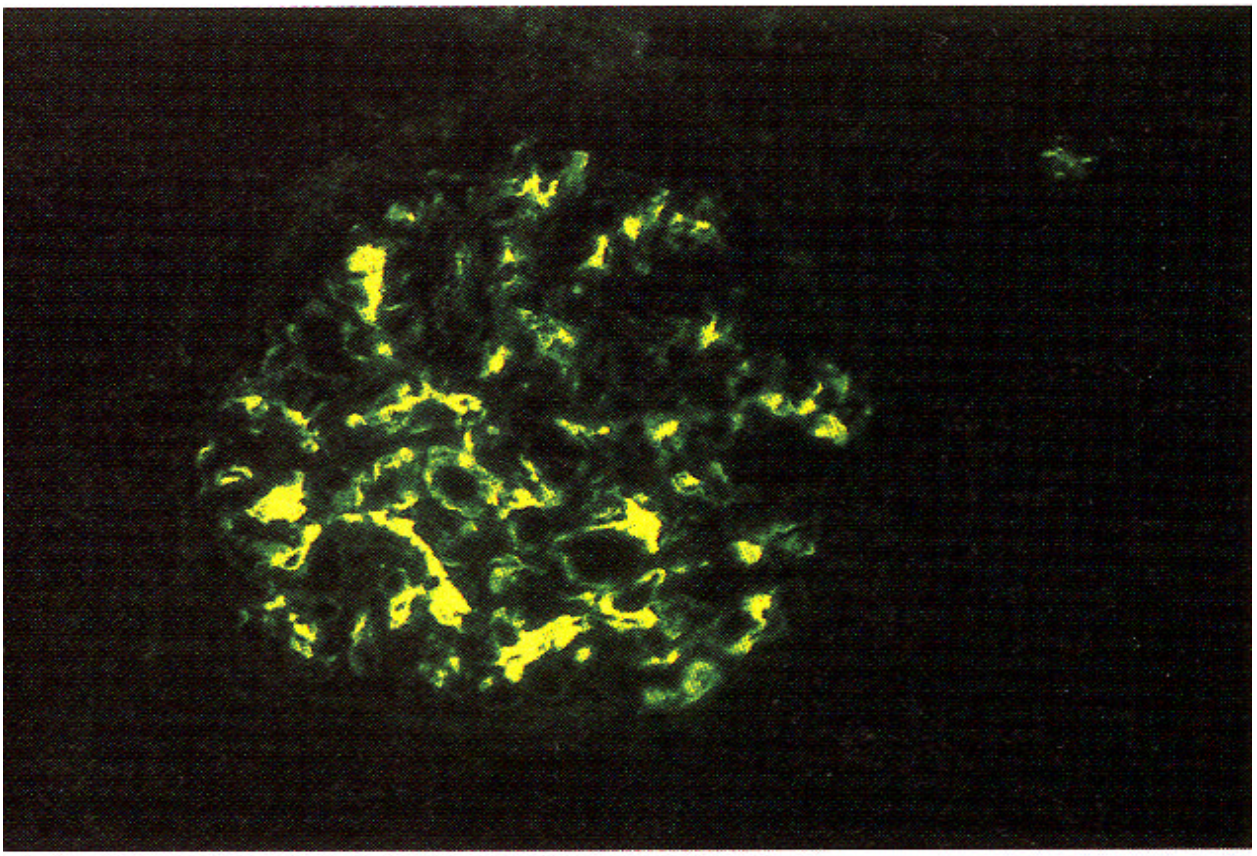

Figure 2 – The Glomeruli when immunohistologically examined with IgA antibody