fluorescent staining of renal biopsy specimens revealed no deposition of IgG, IgM or C3 in the glomeruli (data not shown). Examination of the nasal cavity revealed an oozing ulcer of the septum. Nasal bleeding was stopped after nasal tamponade.

An initial diagnosis of primary SS with crescentic glomerulonephritis was made and she was treated with intravenous methylprednisolone (125 mg/day, three successive days) and followed by plasma exchange (3 l/day, total 6 l). Thereafter she was given oral prednisolone (40 mg/day). These therapies resulted in a marked decrease of MPO-ANCA. Low dose of cyclophosphamide (25 mg/day) was also tried during, but had to be stopped because of myelosuppression. Renal function had markedly improved (BUN 32 mg/dl, Cr 1.8 mg/dl) when she was discharged after 99 days.

Comment. The most common renal lesion of SS is interstitial nephritis with interstitial lymphocytic infiltration, fibrosis and tubular atrophy. In contrast, glomerulonephritis is relatively rare [1,2]. The present case had extraglandular manifestations affecting the lungs and kidneys. The present case fulfilled the criteria of primary SS [3], i.e. dry eyes with positive Schirmer’s test, abnormal biopsy findings of salivary gland and presence of specific autoantibodies. MPO-ANCA, the serological marker for systemic vasculitis, was also present. Cryoglobulin-mediated glomerulopathy is occasionally found in primary SS [4], although cryoglobulins were detected in our patient, but immunofluorescence study showed no immunoglobulin deposits in the glomeruli.

Antibodies to MPO have been reported in collagen disease such as SLE [5,6], but only two cases of SS with MPO-ANCA-related glomerulonephritis have been reported [7,8].

References

**Seizures as the presenting feature of post-streptococcal glomerulonephritis**

Sir,

During the last decades many diseases have become progressively rare mainly due to better hygiene and vaccination of the population. For this reason diagnosis can be difficult, especially when presenting symptoms are confusing or unusual.

**Case.** A 33-year-old female was admitted to the emergency ward because of seizures and blurred vision. The previous week she had been treated with tablet tetracycline for sinusitis, but was otherwise healthy. On admission she was somnolent, with mild periorbital oedema, and diastolic hypertension, 110–120 mmHg. The rest of the physical examination was unremarkable. A CT (computed tomography) of the brain and a lumbar puncture were performed, followed by an MR (magnetic resonance). Localized oedema was noticed in the occipital, temporal, frontal and parietal lobes (Figure 1). Cerebro spinal fluid (CSF) cell count was normal. Virus encephalitis was suspected and treatment with acyclovir parenterally was initiated but discontinued 2 days later when virologic examination of blood and CSF did not support this diagnosis. A chest X-ray showed bilateral perihilar pulmonary congestion and oedema, particularly in the right lower lobe, while sonography of the kidneys, including renal artery circulation, were normal. The sedimentation rate was 50 mm (0–20), C-reactive protein 33 mg/l (<9 mg/l), serum creatinine 71 µmol/l (60–120 µmol/l), serum albumin 27 g/l (36–48 g/l). Urinalysis showed albuminuria reaching 1.3 g/l and microscopic haematuria. An acute inflammatory reaction and hypoalbuminaemia were found in plasma electrophoresis the following day, with complement C3 and C4 within the lower normal range. Serologic tests for ANA, p- and c-ANCA, anti-GBM antibodies, and RF gave no evidence of connective tissue or vasculitic diseases. A 24 h urine catecholamines sample was normal. Renal crisis due to scleroderma was excluded on the basis of the clinical and laboratory findings. Eye fundoscopy and urinary bladder cystoscopy disclosed no abnormalities. The initial patho-
Comment. This interesting case has many instructive points beginning with the presenting symptom which was dramatic and highly suggestive of a cerebral process. The MR findings were first thought to represent an infection, which led to treatment with acyclovir. Hypertensive encephalopathy due to post infectious glomerulonephritis is very rare but the diagnostic approach could be improved by a detailed medical history [3]. The complement levels, which usually decline 3 to 5 weeks from the beginning of a streptococcal pharyngitis, were normal [4]. Our interpretation of this result is that the examination was performed too soon after the onset of symptoms. A low level of suspicion due to the current rarity of post-streptococcal nephritis partially explains the difficulty in making a correct diagnosis.

Safety and efficacy of corticosteroids in childhood nephrotic syndrome with concomitant hepatitis B

Sir, Chronic hepatitis B infection is a well-recognized cause of membranous nephropathy and membranoproliferative glomerulonephritis and generally manifests as nephrotic syndrome (NS) [1,2]. Clinical trials using corticosteroid (C) therapy have been disappointing, in that a benefit has not been demonstrated consistently [3]. Conversely, in several reports therapy has been noted to be, in fact, detrimental, by increasing viral replication [4]. Moreover, the sudden cessation of C may be associated with rebound activation of the immune system, with resultant acute hepatic decompensation [5].

Case. We are reporting our experience in managing a 3-year-old boy with new onset NS who was found to be positive for hepatitis B surface antigen (HBsAg) with a hepatitis B DNA titre (by PCR) of greater than 10,525 pg/ml (normal <5.0 pg/ml). Laboratory work-up confirmed the nephrosis, with mildly elevated transaminases. Liver and kidney biopsies performed subsequently showed evidence of chronic active hepatitis, and much to our surprise, minimal change renal disease (MCD) without immune complexes on electron micrography. Due to concern that C therapy could foster viral activation, he received a 6-month course of interferon alpha (IA) 2-b (3 million units thrice weekly) without improvement. Due to his persistent nephrosis, a trial of C therapy in tapering doses (oral prednisolone 2 mg/kg/day) was initiated, in combination with a second course of IA 2-b. This has resulted in a prompt and sustained remission of his NS, without clinical deterioration of his liver function. He however, continues to have evidence of active viral replication and mild transaminase elevations.

Comment. The patient presented herein, differs from children previously reported with hepatitis B associated NS in several aspects, most importantly in that his underlying renal histopathology was that of MCD. Manna et al. [6] noted a higher prevalence of HBsAg carriage among male children with MCD compared to controls, although less than half of the patients whom they classified as having MCD had undergone a renal biopsy to confirm the histologic diagnosis. In addition, it is unclear from their data at what stage during the course of the disease hepatitis B serology was initially checked, and whether any of these children had previously received a course of C therapy which potentially, could have altered immune responsiveness, thereby resulting in increased susceptibility to, and chronic carriage of, hepatitis B virus. It is certainly possible that the two conditions could coexist in the same patient without being related pathogenically. The fact that our patient’s heavy proteinuria failed to respond to a trial of IA alone, but promptly resolved after initiation of C therapy in spite of persistent hepatitis B antigenemia, strongly supports an incidental rather than a cause and effect relationship between his NS and hepatitis B.

In conclusion, our opinion all patients with new onset NS should be evaluated for serologic evidence of hepatitis B infection prior to initiation of C therapy, which may enhance viral replication, and that the presence of hepatitis B antigenemia in the setting of newly diagnosed childhood NS, whether felt to be related or not, is not a contraindication to the use of C in a slow tapering regimen, with concomitant interferon therapy and careful clinical monitoring, and should be offered to patients who have failed a course of IA alone.