more flare-ups of hepatitis than patients without \((P < 0.005)\) [2]. Although Tang et al. [4] described one patient who had a relapse of nephropathy after 2 years of complete remission when lamivudine was withdrawn, there was no patient whose nephropathy was in complete or partial remission and who was being treated by an angiotensin-converting enzyme inhibitor at 12 months ended up with end-stage renal disease by 3 years of follow-up. It is supposed that the renal function is conserved when patient’s proteinuria declines into remission levels. Therefore, long-term lamivudine therapy for the patients with HBV-associated nephropathy should be considered unnecessary, perhaps even unreasonable. Although the optimal duration of treatment and the criteria for stopping it have not been established, maintaining lamivudine therapy for 4–6 months following chemotherapy was suggested [5].

We would like to reinforce the point that long-term lamivudine therapy should be used only for patients who do not experience remission under supportive treatment, or for those with relapse of nephropathy after lamivudine withdrawal, so as to prevent lamivudine-resistant mutations and hepatitis flare-ups.

Conflict of interest statement. None declared.


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A renal transplant patient with a solitary plasmacytoma in the oral cavity

Sir,

Post-transplant lymphoproliferative disorder (PTLD) is a serious complication of transplantation because it can occur early after transplantation and carries a high morbidity and mortality. Not more than 4% of the malignant tumors detected in organ recipients are plasmacytomas [1,2]. Although the upper respiratory tract and oral regions are favourable sites for the extramedullary or solitary plasmacytoma, solitary plasmacytoma after a renal transplantation is extremely rare [3,4]. We report a renal transplant patient with a solitary plasmacytoma of the mandible that developed 12 years after a living related renal transplantation. A 26-year-old male patient presented with a mild gingival enlargement in the left mandibular molar area. His medications were cyclosporine A (CsA) 200 mg/day, azathioprine 50 mg/day and methylprednisolone 6 mg/day. Physical examination revealed no abnormalities except a buccogingival mass measuring 1×1 cm between the lower left first and second molar teeth (Figure 1). Laboratory investigation revealed: an erythrocyte sedimentation rate 98 mm/h, leukocytes 6910/μl, haemoglobin 10.7 g/dl, serum creatinine concentration 2.1 mg/dl and proteinuria 0.5 g/day. An excisional biopsy was performed and microscopic examination revealed diffuse infiltration of plasma cells with kappa light chain monotype. A pathologic diagnosis of a plasmacytoma was made. Bone marrow biopsy and examination for multiple myeloma revealed no evidence of systemic involvement. Azathioprine was stopped. CsA was switched to rapamycin and surgical excision was performed. After six months of outpatient clinical follow-up, a new buccogingival mass measuring 1×1 cm between the lower right first and second molar teeth was detected. An excisional biopsy was performed again and microscopic examination revealed diffuse infiltration of plasma cells with kappa light chain monotype. Immunosuppressed organ allograft recipients are at risk of developing lymphoproliferative disorders as a consequence of immunosuppressive therapy and long-term antigenic stimulation from both the graft and possible viral infections. In this rare case, although the PTLD regressed after changing the immunosuppressive treatment with concurrent excisional surgery, solitary plasmacytoma recurred in a different site.

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Fig. 1. A buccogingival mass diagnosed as solitary plasmacytoma measuring 1×1 cm between the lower left first and second molar teeth.
Rituximab-induced long-term remission of membranous lupus nephritis

Sir,

Rituximab is a chimeric anti-CD20 monoclonal antibody that has proved to be effective in depleting B-lymphocytes in vivo [1]. We report here a case of a woman with severe membranous lupus nephritis who was treated with rituximab with significant and persistent improvement during a 3 year follow-up. She had previously failed to respond to therapy with cyclophosphamide, prednisolone, cyclosporine and mycophenolate mofetil (MMF).

A 16-year-old female with SLE and nephrotic syndrome had a renal biopsy performed showing membranous lupus nephritis (WHO class V). The patient was treated with prednisolone continuously and a sustained remission of the nephrotic syndrome was achieved. At the age of 22, the patient had a relapse and a new renal biopsy showed WHO class V. The prednisolone dose was increased and i.v. cyclophosphamide was administered monthly for 6 months. A re-biopsy showed persistent WHO class V. Cyclophosphamide i.v. pulse therapy was continued but due to incomplete effect and side effects, therapy was stopped after two infusions. The patient was started on oral cyclosporine, 24-h urine albumin was 5.2 g and glomerular filtration rate (GFR) was 59 ml/min. During treatment, proteinuria decreased to approximately 1–2 g/24 h.

At ~27 months (before start of rituximab) the then 25-year-old woman was treated with cyclosporine 75 mg and prednisolone 5 mg/day (Table 1). At ~12 months, the nephrotic syndrome relapsed and the dose of cyclosporine was increased to 150 mg, prednisolone to 30 mg and MMF 1 g/day was added, subsequently increased to 2 g/day. A year later (~1 month), the patient developed pneumonia with septicaemia and a severe nephrotic syndrome with extensive oedema, pericardial effusion and hypertension developed. GFR was 5 ml/min. Cyclosporine and MMF were withdrawn and the patient was given high doses of i.v. methylprednisolone. Kidney biopsy showed lupus nephritis WHO class Vb and vasculopathy with thrombotic microangiopathy and signs of malignant hypertension. At that time (month 0), i.v. rituximab (Mabthera) 375 mg/m² body surface area was given once weekly for 4 weeks in combination with i.v. cyclophosphamide 0.5 g/m² twice. After treatment, the CD19 positive B-lymphocytes were totally depleted, starting to recover after approximately 1 year. By then, the ongoing low-dose prednisolone treatment was combined with MMF in a dose of 0.5 g/day. 24 months after treatment with rituximab, GFR had increased to 40 ml/min. At 36 months, treatment consisted of prednisolone 5 mg in combination with MMF 1 g/day and antihypertensive drugs. Serum creatinine was 118 µmol/l, serum albumin 36 g/l and 24-h urine albumin 7 mg (Table 1).

In membranous lupus nephritis the optimal treatment is controversial although remission of the nephrotic syndrome can occur following the treatment with steroids in combination with cyclophosphamide, cyclosporine or MMF. Despite extensive immunosuppressive treatment during the disease course, the nephrotic syndrome persisted in our patient and was further complicated by infectious complications and renal failure. At that time rituximab was given in combination with two infusions of cyclophosphamide with long-standing beneficial results. Rituximab-induced remission of refractory nephrotic syndrome has previously been reported in patients with idiopathic membranous nephropathy and membranous lupus nephritis [2,3] and promising effects of rituximab in short-term follow-ups in lupus nephritis patients have been described [4,5]. This strengthens the view that depletion of B-cells with rituximab may be a new therapeutic option in patients with severe therapy-resistant membranous lupus nephritis.

Conflict of interest statement. None declared.

Table 1. Laboratory examinations prior and after treatment with rituximab

<table>
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<th>Months</th>
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<td>−27</td>
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<td>Creatinine (µmol/l)</td>
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<td>24 hrs U-albumin (mg)</td>
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<td>GFR (ml/m²×1.73 m²)</td>
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<td>IgG-dsDNA (FEIA) (IU/ml)</td>
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1. Maloney DG. Pre-clinical and phase I and II trials of rituximab. Semin Oncol 1999; 26 [Suppl 14]: 74–78