Surgical repair of the leakage would probably be the most satisfying option in this situation. However, this would usually require a prolonged rest of PD and the risk of bleeding complications without the possibility to stop anti-coagulation in the patient. Therefore we decided on a conservative management.

Fibrin glue instillation was performed. We used the following approach: after instillation of dialysis solution, and guided by ultrasound, we carefully injected 1ml fibrin glue into the space slightly above the peritoneal gap. A compression bandage was applied for 12 h. Three days later a low echogenic mass became visible in the region where the fibrin glue had been injected and a thin echogenic line indicated the restoration of the peritoneal membrane (Figure 2B). After five additional days, we started to instill 500 ml dialysate per exchange increasing the volume up to 1000 ml within 10 days. She is presently doing well with nocturnal attenuated peritoneal dialysis (APD) and 1000 ml icodextrin at daytime 4 months after discharge.

Experiences with fibrin glue in peritoneal leakage are still limited. Joffe [3] described his positive experience with fibrin glue in the treatment of pericatheter leakage. As far as we know, this is the first report that demonstrates the successful treatment of early internal leakage using fibrin glue.

Fibrin glue should be considered as an effective and safe tool in cases where standard treatment of dialysate leakages have failed or if a fast resumption of PD is required.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfl080
Advance Access publication 31 January 2006

Combined renal replacement therapy for severe metformin-induced lactic acidosis

Sir,

This report is on a 68-year-old woman with a history of type 2 diabetes mellitus treated with metformin 850 mg thrice daily, and mild chronic renal failure, who underwent cardiopulmonary resuscitation for cardiovascular collapse because of severe metformin-associated lactic acidosis. Even after 12 h of continuous venovenous haemofiltration (CVVH), lactic acidosis persisted and the patient required increasing doses of norepinephrine. After starting simultaneous haemofiltration via a second vascular access, lactic acidosis resolved, norepinephrine infusion was discontinued and the patient subsequently made a complete recovery.

Metformin is the only medication for type 2 diabetes mellitus which has been demonstrated to reduce the risk of macrovascular complications of diabetes [1]. Lactic acidosis is a rare but potentially fatal adverse effect of metformin (30–50% mortality rate [2]) with an estimated risk of 1–15/100 000 patient-years [2]. The significance of lactic acidosis due to accumulation of metformin in renal insufficiency in the absence of other precipitating factors is the subject of controversial debate [2,3]. Tissue hypoxia triggering lactic acidosis is presumed to be present in most cases.

The patient was admitted to the stroke unit for sudden visual loss, nausea, vomiting and faintness. Soon after diagnosis of a severe metabolic acidosis (pH 6.5), the patient had a cardiovascular collapse. After successful resuscitation, she was transferred to our medical ICU, mechanically ventilated and placed on norepinephrine infusion. Blood gas analysis revealed severe lactic acidosis: pH 6.83, base excess −28 mmol/l, bicarbonate 7.9 mmol/l, lactate 35.3 mmol/l.
History and clinical presentation led to the suspicion of a metformin-associated lactic acidosis [4]. Other possible causes for lactic acidosis and circulatory failure, specifically cardiogenic or hypovolaemic shock, bowel or limb ischaemia, severe sepsis or septic shock were ruled out. The plasma level of metformin was 41.9 mg/l (therapeutic range 0.3–1.2 mg/l) and confirmed our diagnosis. Despite immediate CVVH (AV 400, with an ultrafiltration rate of 1500 ml/h and bicarbonate-based substitution solution), the patient’s condition deteriorated, the norepinephrine dose had to be increased to 1.65 \( \mu \)g/kg/min, the serum lactate level rose (Figure 1). Since vascular access did not allow for higher ultrafiltration rates, and following the suggestion of Panzer et al. [5], we started an additional discontinuous haemofiltration (high-flux) via a separate venous catheter. Under this combined renal replacement therapy, serum lactate decreased promptly and norepinephrine doses could be tapered off (Figure 1). Subsequently, the patient recovered completely from the acute illness.

The benefit of simultaneously combining intermittent and continuous renal replacement therapy in case of severe metformin-induced lactic acidosis with circulatory failure, as described by Panzer et al. [5], was confirmed in our case. Thus, the approach may be considered in other cases of this frequently fatal complication of diabetes therapy.

Conflict of interest statement. None declared.

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doi:10.1093/ndt/gfl011
Advance Access publication 27 April 2006

Remission of polyomavirus-induced graft nephropathy treated with low-dose leflunomide

Sir,

The incidence of polyomavirus (BK) induced allograft nephropathy (PVAN) has gained in clinical importance as a cause of renal graft dysfunction [1]. Treatment options are at present confined to reduction of immunosuppression with or without low-dose cidofovir [2]. Leflunomide is an immunosuppressive drug with in vitro and suspected in vivo antiviral potency and has been applied to PVAN with promising results [3].

Two living donor renal transplant recipients were diagnosed with PVAN, 117 and 70 days after transplantation, respectively [characteristics of patient 1/patient 2 respectively: age 39/51 years; renal disease: MPGN/IgA-nephritis; blood group donor/recipient: (0/0)/(A/0); CMV-serostatus donor/recipient: (+/+)/(+/C0; HLA-mismatches: 3/4, cellular rejection episodes prior to PVAN: 2/1]. The diagnosis was confirmed by renal biopsy and positive quantitative BK RT-PCR (graded PVAN B/PVAN A according to [2]; 3.4 and 2.1 million copies/ml plasma, respectively). Preceding leflunomide therapy, immunosuppressive therapy was reduced and a course of immunoglobulins administered in patient 1 without effect within 3 weeks.