of the tunnelled cuffed catheter (TCC) is considered impossible or undesirable. The recommended vancomycin concentration in the lock is at least 1000 times higher than the minimal inhibitory concentration of the microorganism involved [1,3], i.e. at least 2.5 mg/mL [2]. In order to ensure effective and safe systemic vancomycin treatment despite variable removal by residual renal function and dialysis sessions, measurement of pre-dialysis trough levels is also recommended [1]. However, if the catheter has been locked with a solution containing vancomycin, and predialysis serum is obtained, as usual, from the catheter lumens prior to the start of a session, the vancomycin serum level may be considerably overestimated.

We report the case of one patient on chronic haemodialysis by a TCC. She received intravenous vancomycin for a CRBSI. The TCC was further locked with a solution of vancomycin (5 mg/mL) diluted in pure heparin (5000 IU/mL) at the end of every haemodialysis (HD) session. Vancomycin pre-dialysis trough levels measured by immunoassay (Abbott Laboratories, www.abbott.com) in samples taken from the catheter at the beginning of the session were surprisingly high (47 μg/mL). She had received 2 days earlier the first dose of vancomycin (1 g) during the last 60 min of the previous dialysis session. A second blood sample drawn 50 min later from the dialysis circuit showed a level of 10.8 μg/mL.

To confirm our suspicion that this discrepancy in vancomycin levels reflected the residual high concentration in the TCC due to the interdialytic lock, rather than very rapid vancomycin removal by HD, two independent samples were drawn from the TCC and from the dialysis circuit before and 5 min after starting the HD session, respectively, in two additional patients. In both of them, vancomycin trough levels were >100 μg/mL (outside the upper range of the test) and <20 μg/mL (10.5 and 16.6 μg/mL), in samples taken from the catheter and dialysis circuit, respectively.

Our cases are reminiscent of a similar problem observed while monitoring warfarin treatment (by INR measurement) on blood samples drawn directly from HD catheters locked with pure heparin if aspiration of the endoluminal content was incomplete [4]. This problem can easily be avoided by drawing the samples directly from the dialysis circuit a few minutes after the beginning of each session. Clinicians should consider this fact for the routine interpretation of laboratory values and the prescription of drugs such as vancomycin in patients dialysed by a catheter. Otherwise, gross underdosing of systemic vancomycin could seriously increase the risk of treatment failure.

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Sir,

We read with interest the article entitled ‘The increased risk of post-transplant diabetes mellitus (PTDM) in peritoneal dialysis-treated kidney allograft recipients’ by Madziarska et al. [1]. While a number of risk factors for PTDM have been identified, previous literature is scarce concerning the potential influence of dialysis modality on PTDM risk. In this study, the authors examined 306 renal transplant recipients (RTRs) among whom 23.4% developed PTDM. In multivariate analysis, older recipient age, previous treatment, and a positive family history of diabetes and previous treatment by peritoneal dialysis (PD) were significantly associated with an increased risk of PTDM.

We recently formulated an opposite hypothesis for the following reasons. First, we showed that weight gain during the first year after transplantation, a major risk factor for PTDM, was greater in haemodialysis (HD) patients than in PD patients [2]. Ghrelin can significantly increase food intake [3]. Thus, higher ghrelin levels observed in HD patients compared to PD patients can predispose them to an increased risk of weight gain and PTDM [3]. It is interesting to observe that, in the study by Madziarska et al. [1], HD patients had significantly greater weight gain in the first year following transplantation. Second, inflammation is also considered as a risk factor for PTDM and a number of studies have shown a significant increase in inflammatory mediators in HD patients as compared with PD [4]. Third, in our own cohort of RTRs, we observed a lower incidence of PTDM in PD patients [5]. Nevertheless, we believed that a monocentre study is not suitable for consistent conclusions considering the high risk of alpha error of such a result. We performed a multicentre retrospective study including 2010 consecutive RTRs [5]. PTDM was defined as a need for anti-diabetic therapy in the 6 months following renal transplantation. A total of 6.8% of patients developed PTDM. The characteristics of our population were comparable to those of Madziarska’s study. The proportion of patients in each modality of dialysis was similar. The incidence of PTDM was lower probably because of the definition we used. However, most studies on the impact of PTDM on transplant outcomes have used the ‘need for treatment’ as a definition and the observed rate of PTDM is consistent with previous reports from other European centres. In our study, the incidence of PTDM was quite similar in HD and PD patients (7 versus 6.5%, P = 0.85). In multivariate analysis, age, body mass index at transplantation, use of tacrolimus and rejection episodes but not dialysis modality were identified as independent risk factors for development of PTDM.

The results of our study show that pre-transplant dialysis modality does not have any impact on the development of PTDM in RTR. We believe that only large multicentre studies are needed to resolve such complex questions.

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Sir,

We appreciate the interest in our paper [1] expressed by Courivaud and Ducloux. Reading insightfully the original paper ‘Impact of pre-transplant dialysis modality on post-transplant diabetes mellitus after kidney transplantation’ [2] published by the discussants, we found, surprisingly,