Sir,

We appreciate the interest and contribution of Liberek et al. to the controversy regarding timely initiation of dialysis. We agree that the findings of our study should not be used to advocate a late or low eGFR start on dialysis. In fact, as an observational cohort study, our study simply describes outcomes of those in whom dialysis was started at different levels of eGFR, and the data suggest that eGFR values should not be used in isolation, either to make a judgement on starting dialysis or as an auditing tool for the quality of ESRF care [1]. In fact, we describe an excellent example of ‘confounding by indication’: those with higher eGFR appear to be sicker, thus leading physicians to commence dialysis. Interestingly, however, the European Best Practice Guidelines [2] recommend ensuring that all patients have started dialysis before eGFR < 6 ml/min/1.73 m², regardless of the presence or absence of signs and symptoms.

Methods of assessing residual renal function that consider weight, and thus indirectly muscle mass and nutrition, such as creatinine clearance, are preferable in this setting to assess the significance role. Traynor et al. [4] published results on survival and dialysis initiation utilizing the Cockcroft–Gault formula, which at least incorporates weight. The results are consistent with our findings and also contradict the concept of ‘early’ or ‘healthy’ start.

While our study uses age and diabetes as indicators of comorbidity, both van Manen et al. [5] and Stel et al. [6] have found that after adjusting for age and diabetes, further adjustments for comorbidity made little further difference in European populations. Furthermore, it is interesting that Stel et al. found that the distribution of causes of death was similar in high, medium and low eGFR groups [6].

Finally, we welcome the sentiment that the individual decision of dialysis timing involves a clinical judgement of nephrologists on the basis of a variety of clinical features, particularly fluid overload in patients with left ventricular dysfunction. This is valuable to consider not only when met with studies that counter-intuitively appear to suggest a benefit in delaying the start of dialysis, but also when met with contradictory recommendations of a healthy early start. While we hope that the randomized controlled IDEAL trial [7] may be able provide some answers next year, meanwhile our study and that of Stel et al. demonstrate that nephrology is a specialty that values the physician who adopts a patient-centred approach.

Conflict of interest statement. KS is the Chair of the Scottish Renal Registry. AL is provincial executive director of the BC Provincial Renal Agency, BCPRA.


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Non-diphtheria corynebacteria and CAPD infections

Sir,

The comprehensive analysis of the large scale ANZDATA presented by Dr D. Johnson and co-workers [1] highlights the increasing importance of non-diphtheria Corynebacteria as emerging pathogens in the population of patients on continuous ambulatory peritoneal dialysis. This is in accordance with smaller reports of Corynebacteria peritonitis [2,3] or exit-site/tunnel infections [4,5] by Corynebacteria. Regrettably, the incomplete data of the Register question both the correct diagnosis of peritonitis as well as the therapeutic recommendations for this infectious complication.

Firstly, rapid identification of potentially multi-resistant non-diphtheria Corynebacteria to the species level may be difficult with commercially available systems. However, as not all Corynebacteria play a role in peritonitis, it is essential to identify correctly pathogenic Corynebacteria. Currently, the genus Corynebacteria consist of 59 validly described species, of which 35 species are considered to be clinically relevant. Only a few C. species (predominantly C. striatum, C. aquatum, C. jeikeium) have been implicated in CAPD infection [6]. These organisms are even today frequently...
dismissed as contaminants if isolated together with other pathogens or from otherwise culture negative isolates [7]. Using the genus Corynebacteria as the source of peritonitis, the ANZDATA analysis gave no correct information about the Corynebacteria species causing the peritonitis, nor whether peritonitis relapse or repeated peritonitis were caused by the same species. The causal relationship between a causative microbe and a disease (Koch’s postulates) has not been established for a significant proportion of Australian patients with Corynebacteria isolated from peritonitis drainage.

Secondly, the authors’ recommendation to start empiric therapy with cefazolin rather than with vancomycin for a total antibiotic treatment duration of 2 weeks is not supported by the registry data and stands in sharp contrast to the data of smaller studies or case series [2]. The authors did not give the results of antibiotic susceptibility testing, neither the dosage nor the routes of antibiotic administration. In contrast to cephalosporins, all Corynebacteria species are in vitro susceptible to vancomycin, particularly C. jeikeium. CAPD-peritonitis caused by pathogenic Corynebacteria is not a benign disorder, it often relapses, affects patients’ quality of life and causes significant morbidity and even mortality in this population. Current recommendations based on few published data include early diagnosis of infectious complications of CAPD, isolation of the causative Corynebacteria and differentiation to the species level, administration of vancomycin as part of empirical therapy and duration of antibiotic therapy of 3 weeks.

**Conflict of interest statement.** None declared.

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**Reply**

Sir,

We thank Professors Schiffl and Lang for their interest in our article discussing *Corynebacterium* peritonitis in Australian peritoneal dialysis (PD) patients [1]. They specifically comment that (1) the lack of availability of data on *Corynebacterium* species identification calls into question the correct diagnosis of *Corynebacterium* peritonitis since only a proportion of the 59 described species are considered clinically relevant, and (2) they believe that a 3-week course of vancomycin is more appropriate than cephalosporin therapy because all *Corynebacterium* species are susceptible to vancomycin and because *Corynebacterium* peritonitis is a potentially serious condition.

We agree that correct speciation would have provided useful additional information that may have helped to establish the pathogenic significance of the isolate. However, as Professors Schiffl and Lang concede, identification of corynebacteria to the species level is often difficult with commercially available systems and is not done by many microbiology laboratories. A diagnosis of *Corynebacterium* peritonitis was therefore made on clinical grounds by the treating nephrologist. We acknowledged in our paper that the lack of speciation data was a limitation of our study, as it was in a previous case series [2]. Indeed, we are not aware of any previously published case series of *Corynebacterium* peritonitis of a reasonable size where species identification has been provided.

With respect to initial empiric antibiotic therapy of PD-associated peritonitis prior to availability of antibiotic susceptibility data, our results clearly demonstrated that *Corynebacterium* peritonitis outcomes were similar regardless of whether patients were initially treated with vancomycin, cephalosporins or other antimicrobial agents. Indeed, the small case series that Professors Schiffl and Lang cited to support their argument did not, in fact, find any significant difference in either the primary response rate or the complete cure rate of *Corynebacterium* peritonitis episodes treated with vancomycin initially as opposed to a cephalosporin initially [2]. In line with international recommendations to minimize vancomycin usage in renal units to curtail emergence of vancomycin-resistant enterococci [3,4], it would seem appropriate to commence initial empiric treatment with a cephalosporin-based regimen. However, once antimicrobial susceptibility data become available, we agree with Professors Schiffl and Lang that, as with any other case of peritonitis, antimicrobial therapy should then be tailored to these specific susceptibilities. Indeed, our data showed that the most common antibiotic used as a second or third antibiotic regimen was vancomycin. Moreover, *Corynebacterium* peritonitis outcomes were not significantly associated with antibiotic treatment duration and were generally excellent in spite of a median treatment course duration of 13 days. We would therefore argue based on our data that a 2-week course is sufficient in most cases of *Corynebacterium* peritonitis.

**Conflict of interest statement.** None declared.