Regarding the letter, we thank Dr. Gambaro et al. for the interest in our recent paper published in *Nephrology Dialysis Transplantation*.

We agree with the observation that the majority of patients with medullary sponge kidney (MSK) do not present the typical distal renal tubular acidosis (dRTA) clinical phenotype (e.g. growth failure from infancy). However, dRTA is characterized by a wide clinical heterogeneity, and only in the last few years, has this metabolic disease been studied in all its various clinical and molecular aspects. Some cases of dRTA can show low clinical expression, so the diagnosis may be delayed until the appearance of classical complications. We recently studied two sisters affected by dRTA, one with the typical clinical manifestations of the disease and the other (30 years old) with a metabolic history that remained essentially silent. Both have the same mutation in one of the causative dRTA genes. Furthermore, as reported in our article, Kasap and colleagues described a 5-year-old girl with MSK associated with dRTA [1]. Despite the fact that a molecular analysis was not performed, the clinical features described by the authors are really convincing that this patient could have both conditions.

Because the urographic pictures were taken several years ago, it is difficult to answer point by point the comments of Gambaro et al. However, usually, provocative manoeuvres are applied when it is necessary to increase the sensitivity of the procedure, but the Rx images of the reported patients show the typical features of MSK, as our current consultant radiologists, who reviewed the X-ray films, confirmed.

We cannot conclude that MSK is ever a consequence of dRTA, but since MSK is a heterogeneous condition, as also stressed by Gambaro et al., for the first time, our study has highlighted the association of two rare renal diseases coupled with the genes responsible of dRTA, and it revealed a possible correlation between MSK and dRTA.

Further studies are certainly needed to clarify not only the relationship between these two clinical conditions but also the phenotypic expression of dRTA, which is still not well known.

*Conflict of interest statement.* None declared

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Is *Chlamydia pneumoniae* the causative agent of microscopic polyangiitis?

Sir,

Iyoda et al. have reported that *Chlamydia pneumoniae* infection might be involved in the pathogenesis of myeloperoxidase (MPO)—antineutrophil cytoplasmic antibody (ANCA)-associated glomerulonephritis (GN) because the prevalence of immunoglobulin M (IgM) antibody against *C. pneumoniae* was significantly higher in the active MPO–ANCA-associated GN group than in the remission and control groups (60%, 30% and 26%, respectively) [1]. However, rheumatoid factors (RF) interfere with the detection of anti-*C. pneumoniae* IgM antibodies in the presence of anti-*C. pneumoniae* immunoglobulin G (IgG) antibodies, leading to false-positive results [2,3]. Hence, the serum IgG should be absorbed before the measurement of anti-*C. pneumoniae* IgM antibodies. The prevalence of circulating RF [2] and the incidence of MPO–ANCA-associated microscopic polyangiitis (mPA) [4] increase with age. Iyoda et al. have not commented whether RF were present or IgG was depleted from the serum samples [1]; therefore, their results might be affected by the presence of RF.

We measured the level of anti-*C. pneumoniae* IgM antibodies to investigate the true prevalence of anti-*C. pneumoniae* IgM antibodies in patients with active MPO–ANCA-associated mPA using the revised enzyme-linked immunosorbent assay (ELISA) kits (Hitachi Chemical Co. Ltd, Tokyo, Japan), which were introduced in December 2007, for reducing the influence of RF and other confounding factors.

Our study group comprised 15 patients with MPO–ANCA-associated mPA (5 males and 10 females; mean age, 70.0 years). All patients had rapidly progressive GN. All the serum samples were collected at the active phase (14 patients, at diagnosis; 1, at flare). We measured the levels of anti-*C. pneumoniae* IgM antibodies in these sera and the corresponding IgG-depleted sera, which were prepared using the Protein G spin column (GE Healthcare, Uppsala, Sweden), and corrected by serum IgM concentration before IgG depletion. Out of 15 patients, seven (47%) were positive for RF. Although two of the 15 patients (13%) were positive for both anti-*C. pneumoniae* IgM antibodies and RF, this frequency was low when compared to that reported by Iyoda et al. (Figure 1). Similar results
were obtained for the IgG-depleted serum samples (data not shown). The two patients with interstitial lung disease had dry cough at the first manifestation; therefore, they might have developed an acute *C. pneumoniae* infection. The decrease in the prevalence of anti-*C. pneumoniae* IgM antibodies in patients at the remission phase in their study [1] is intriguing because we did not find any relation between anti-*C. pneumoniae* IgM antibodies and MPO–ANCA-associated mPA in our study. It could be possible that the patients in their study at remission had low prevalence and/or titre of RF and anti-*C. pneumoniae* IgG antibodies after immunosuppressive therapy.

Our study revealed that the prevalence of anti-*C. pneumoniae* IgM antibodies in patients with MPO–ANCA-associated mPA was low, and it is not highly probable that *C. pneumoniae* infection is involved in the pathogenesis of MPO–ANCA-associated vasculitis.

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Reply

I thank Dr Ikenoya and colleagues for their comments and for indicating the probability of interference of rheumatoid factors (RF) in the detection of anti-*C. pneumoniae* IgM antibodies. I am pleased to receive their interesting comments. Although our results are consistent with recently reported data by Fujita *et al.* [1] and Kawaguchi *et al.* [2] indicating that the anti-*C. pneumoniae* IgM-positive results were 33 and 39.4% in MPO-ANCA-associated vasculitis, respectively, I agree that the exclusion of the influence of RF is necessary to avoid false-positive results.

Conflict of interest statement. None declared.

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**Differential impact of dialysis modality on circulating endothelial progenitor cells**

Dear Sir,

With great interest, we read the recent publication by Ueno *et al.* in your journal. They found that circulating levels of endothelial progenitor cells (EPC) are higher in patients with end-stage renal disease treated with continuous ambulatory peritoneal dialysis (CAPD) than in those on haemodialysis (HD) [1]. The authors speculate on a possible