Single-center experience of encapsulating peritoneal sclerosis in patients on peritoneal dialysis for end-stage renal failure

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To the Editor: In the November issue of *Kidney International*, the 5-year experience of encapsulating peritoneal sclerosis (EPS) in patients on peritoneal dialysis in a single center is reported.[1] This retrospective analysis, in which the EPS rate was 3.3% overall, concluded that there were no differences in peritoneal history or peritonitis rates between those who developed EPS and those who did not. Careful radiological monitoring of those on peritoneal dialysis for over 5 years, with early catheter removal if peritoneal thickening was detected, was recommended, with the suggestion that tamoxifen may be beneficial.

This study is a valuable contribution to the literature on EPS, but does not reflect the increasing evidence that a fulminant variant of EPS can occur as a second phase phenomenon immediately following the treatment of acute bacterial peritonitis, and that rapid resolution is reported with corticosteroid treatment alone.[2]

In this setting, an unrelenting acute inflammatory state persists despite appropriate treatment for infective peritonitis, and it may not be recognized that this is due to an acute form of EPS rather than on-going sepsis. Aggressive immunosuppression might be thought potentially hazardous in these circumstances, but the greater risk is to miss the diagnosis and opportunity for treatment. Corticosteroid monotherapy appears particularly effective for these patients,[2,3] who may comprise up to 25% of cases.[4]

The continuing high mortality of EPS suggests that this subgroup of treatment-responsive patients may not be well recognized by nephrologists. Future studies should seek to determine if there is a clinical distinction between the acute and chronic presentations of EPS in terms of pathogenesis and response to immunosuppression.

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Racial differences in response to cinacalcet as a treatment for uremic hyperparathyroidism

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To the Editor: In a recent issue of *Kidney International*, Martin *et al.*[1] reported the results of a randomized controlled trial in which parathyroid hormone (PTH) levels were compared for 205 patients with uremic hyperparathyroidism treated with cinacalcet versus 205 patients treated with placebo. We believe that this analysis may have obscured important PTH outcomes for black subjects.

In a sample of dialysis patients, one of us (AG) reported that average PTH levels were approximately 650 pg/ml in black subjects and 350 pg/ml in white subjects, after adjusting for age, sex, diabetes, serum calcium, and phosphorus.[2] In multivariate regression analysis, black background was the strongest independent correlate of maximum PTH.

Severe uremic hyperparathyroidism may not lead to severe bone disease in black subjects, because black subjects may have a relative resistance to PTH action as suggested by decreased Gla protein and bone turnover.[3] Based on studies mostly among white subjects, PTH level of 150–300 pg/ml is optimal in end-stage renal disease.[4] Because treating black subjects with parathyroid disease by these guidelines may be inappropriate, it is critical to examine racial differences in uremic hyperparathyroidism and response to PTH. Unfortunately, the Martin *et al.* trial compared white subjects with a pooled group of black subjects and 'others'. If the 'other' group did not have PTH levels as elevated as those of black subjects, collapsing these subjects into one category would have created a downward bias in the analysis – thus obscuring the clinical relevance of the findings for black patients with uremic hyperparathyroidism. We recommend repeating the analysis without collapsing the black people and 'other' groups.

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