Letters

Advance Access publication 20 June 2008

Update on reintroduction of epoetin in a patient with pure red cell aplasia

Sir,
A recent review of recombinant human erythropoietin (rHuEPO)-associated pure red cell aplasia (PRCA) cautioned re-challenge with the same or alternative rHuEPO products in the face of continued anaemia in patients with end-stage renal failure [1]. The mainstay of treatment should involve withdrawal of epoetin therapy, immunosuppression and supportive correction of anaemia with blood transfusion. Whilst epoetin re-challenge can be attempted, caution is advised in the face of reported relapse, even with an alternative rHuEPO [2].

Our unit initially reported the case of an 81-year-old man who developed PRCA with positive anti-EPO antibodies whilst on subcutaneous epoetin-alpha in May 2002 [3]. His epoetin was discontinued, and following the 4-month treatment with cyclosporin his anti-EPO antibodies became negative. He became transfusion dependent and after a further 9 months the decision was made to restart him on alternative EPO therapy because of persistent anaemia. He was commenced on darbepoetin and had a successful response to therapy. He remained on cyclosporin throughout this time although subsequent withdrawal had been discussed. The patient chose to remain on cyclosporin as he had suffered no adverse effects. This was one of the first reports of the successful reintroduction of rHuEPO in a patient with PRCA. He remained well and transfusion independent with a haemoglobin level between 10.5 and 11.5 g/dL for 4 years. He subsequently died, aged 84, following complications after a fractured neck of femur. To date there are no reports of any patients having tolerated reintroduction of epoetin following PRCA for a longer duration.

This case illustrates the potential benefits, and safety, of long-term immunosuppressant therapy supporting the reintroduction of rHuEPO following PRCA.

Conflicts of interest statement. None declared.

(See related article by S. Summers et al. The (re)challenging question of erythropoiesis-stimulating agents inducing pure red cell aplasia. Nephrol Dial Transplant 2008; 23: 3053–3055.)

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PD underutilization in Europe: a call to action

Sir,
We read with interest the editorial comment by Van Biesen et al. [1] regarding the detailed list of factors leading to peritoneal dialysis (PD) underutilization in Europe. However, as we ‘cannot teach an old dog new tricks’, all our effort should focus on the training of the renal fellows around Europe. Most of them can easily say by heart all the contraindications (haemodialysis has none!) and complications (mainly peritonitis!) of PD, but rarely a few of its advantages.

More than two-thirds of incident ESRD patients do not have medical contraindications for either haemodialysis (HD) or PD, but the vast majority starts HD [2] and this situation will become worse, as young nephrologists usually do not feel very confident with the modality. Not only in the USA [3], but also in many European countries and Greece, a lot of medical centres with nephrology training programs do not treat enough (or have no PD patients!), or devote not enough time for renal fellows in order to develop expertise in the care of PD patients. Most of the PD training is based mainly on the complications the fellow will face in the nephrology wards. So, the new nephrologist will remain with the bad experiences of resistant, or sclerosing encapsulating peritonitis and severe fluid overload. Successful PD and patients’ real satisfaction with the modality can be appreciated only in the PD outpatient clinic.

Selecting PD is a complex situation for the ESRD patient, who might be aware of HD or transplantation of course, but almost never of PD! The ‘hidden curriculum’ in the society and the ‘hidden persuaders’ in the medical community will always be in favour of HD. The fake dilemma regarding the best modality for ESRD (HD or PD?) should be terminated as soon as possible. The modern nephrologist should be wise enough to recognize the possible contraindications of each modality and confident enough to offer both of them to the right patient. Offering HD to a patient who stays 100 km away from the nearest HD unit sounds equally ridiculous to offering PD in an obese, anuric octogenarian without any assistance at home.

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Our association ERA/EDTA, in collaboration with the ISPD, must focus on all these educational issues and take action to offer more adequate training and exposure to PD, in order to equip the young nephrologists in the future with the appropriate knowledge, regarding the best therapeutic options for the individualized ESRD patient.

Conflict of interest statement. None declared.

Letters and Replies

Advance Access publication 5 August 2008

Correcting hypervolaemic hypernatraemia

Sir,

Nguyen and Kurtz [1] have provided a formula to help achieve a negative Na\(^+\) balance, and a negative water balance to treat hypervolaemic hypernatraemia by infusing a modelled volume of isotonic glucose (D5W) and furosemide.

The formula, however, is not easy to use. Besides knowing the initial [Na\(^+\)] and deciding on a required endpoint in [Na\(^+\)] and a desired reduction in total body water (VMB), you will have to know the initial total body water (TBW\(_1\)), and what is more demanding, you will have to know the combined [E\(_{\text{urine}}\) = [Na\(^+\)] + [K\(^+\)] in urine over the treatment course.

In practice, knowing both TBW\(_1\) and [E\(_{\text{urine}}\)] is not straightforward. In their patient example, the authors happen to know in advance that [E\(_{\text{urine}}\)] is going to be 80 mmol/l, and from that they compute a volume of D5W infusate of 5.6 l. If instead of assuming that [E\(_{\text{urine}}\)] is known, we let it vary between, say, 60 mmol/l and 140 mmol/l, we get from the authors’ formula a volume of infusate between 2.4 l and 8.2 l. Even more important, if we keep the volume of infusate at 5.6 l and investigate the final [Na\(^+\)] resulting solely from changing the [E\(_{\text{urine}}\)] within the range 60–140 mmol/l, we get a new [Na\(^+\)] ranging from 123 mmol/l to 146 mmol/l. This uses a new ancillary formula we have devised solely with the intent of avoiding accidents secondary to using the formula given by Nguyen and Kurtz at face value (ignoring, as they do in their example, non-renal outputs and non-infusate inputs):

\[
Na_2 = [(Na_1 + 23.8)TBW_1 - V_{IVF} \times 1.03[E_{urine}] + 1.03[E_{urine}]V_{MB})/(TBW_1 + V_{MB}) - 23.8
\]

The patient’s history, which the authors use to illustrate the utility of their formula, is extraordinary. The patient is not stated to be demented, to have diabetes insipidus or osmotic diuresis, or to have been denied access to water, or to have been given hypertonic saline, yet this elderly lady with congestive heart failure develops hypernatraemia that is said to be secondary to furosemide treatment. This is indeed a rare occurrence, and hyponatraemia would be more likely under these circumstances.

Besides that, as previously demonstrated [2], the modelled intercept term 23.8 from Edelman [3] is very uncertain with 99% CI including 0 since all measurements of (exchangeable sodium + exchangeable potassium)/total body water were very far from 0.

Hence, returning to the patient’s history, we fully agree with the authors that any formula is dangerous without frequent and comprehensive assessment of the response to treatment. In practice, administering furosemide and D5W (or water per os?) would not be much helped by using a formula with at least two unknown central entries always in need of an update. Rather, the existence of a pseudo-accurate formula might comfort the caretakers unduly into becoming less scrupulous. This pertains in particular to a situation in which the underlying pathophysiology is not well understood, for instance, the absence of the expected increased Na excretion under hypernatraemia [4] as in the patient described.

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