‘Too-early’ initiation of dialysis?
Kidney International (2008) 73, 511; doi:10.1038/sj.ki.5002753

To the Editor: In ‘Diagnosis and salvage of an immature fistula’ by Bhimani and Asif, the interrelationship of several issues—the importance of fistulas, the salvage of fistulas, and the movement toward earlier initiation of dialysis—is apparent.

In the case presentation, the patient began dialysis therapy through a percutaneous catheter with a recent serum creatinine level of 5.2 mg per 100 ml. Assuming reasonably typical body weight, GFR (glomerular filtration rate) was $\sim 10-15 \text{ ml min}^{-1}$. Therefore, the initiation of dialysis was probably in the absence of overt uremic symptoms, in accordance with the recent K/DOQI (Kidney Disease Outcomes Quality Initiative) guidelines that stress earlier initiation and GFR as an important metric—first specified in 1997, pp 17–22.

Unfortunately, the patient suffered complications of percutaneous dialysis—sepsis and malfunction—requiring several catheter replacements.

It is worth stressing that K/DOQI does not propose the GFR metric as a mandate, and especially not as an urgent mandate. Instead, it notes that ‘It is difficult to make a recommendation for initiating renal replacement therapy based solely on a specific level of GFR,’ and discusses the difficulty, to date, that clinical studies have had in demonstrating the benefit of early dialysis. Importantly, three conditions have been listed that ‘may indicate that dialysis is not yet necessary’ despite reaching the proposed GFR, including stable body weight, acceptable nutritional indices, and absence of symptoms.

The case presentation suggests the need to specify a fourth restraint to early initiation of dialysis: the absence of acceptable access. The putative benefits of earlier dialysis are certainly incremental; the purported risk of waiting will accrue slowly over time, not all at the instant of 15 ml min $^{-1}$. The possible risk of deferring dialysis for a short period while waiting for appropriate access must be weighed against the frequent complications of percutaneous catheters. In a risk vs benefit calculation, it might be concluded that placing a catheter in the relatively asymptomatic patient to begin ‘early’ dialysis is not warranted. Acceptable alternatives include the salvage of an immature fistula, the placement of a first or new fistula, ‘bridging’ peritoneal dialysis, even placement of a graft. Many fistulas are usable after only 4–6 weeks; salvaged fistulas or peritoneal dialysis catheters even sooner and newer graft material allows immediate cannulation.

Taken a step further, if a fistula irreversibly thromboses shortly after dialysis initiation, it may be reasonable, in some patients, to stop dialysis until a new appropriate access is available, rather than to reflexively continue dialysis, in all patients, through placement of a percutaneous catheter.

One reason for the epidemic of catheter prevalence may be the ‘too-early’ initiation of dialysis through catheters.


$^S$ Hirsch

1 Renal, Lakeside Nephrology, Chicago, Illinois, USA
Correspondence: $^S$ Hirsch, Renal, Lakeside Nephrology, 55 East Washington St, Chicago, Illinois 60602, USA. E-mail: shelman100@aol.com

Response to ‘‘Too-early’ initiation of dialysis?’
Kidney International (2008) 73, 511; doi:10.1038/sj.ki.5002762

We concur with Sheldon Hirsch. There should be a high threshold for tunneled hemodialysis catheter insertion. Such devices should only be placed when renal replacement therapy is required.


A Asif$^1$ and B Bhimani$^2$

1 Section of Interventional Nephrology, Department of Medicine, University of Miami Miller School of Medicine, Miami, Florida, USA
2 University of Kentucky at Louisville, Division of Nephrology, Department of Medicine, Louisville, Kentucky, USA
Correspondence: A Asif, Section of Interventional Nephrology, Department of Medicine, University of Miami School of Medicine, 1600 NW 10th Ave (R 7168), Miami, Florida 33136, USA. E-mail: Aasif@med.miami.edu

Iron sucrose causes greater proteinuria than ferric gluconate in non-dialysis chronic kidney disease
Kidney International (2008) 73, 511–512; doi:10.1038/sj.ki.5002756

To the Editor: Agarwal et al.$^1$ demonstrated that a single dose of iron sucrose causes greater proteinuria than ferric gluconate in a crossover trial of 12 patients with stage 3–4 chronic kidney disease. The toxicity of iron preparations on renal tubular epithelial cells is becoming increasingly recognized in experimental settings.$^2$ Although not addressed in the original design of the trial, using the blood sampling obtained during the first and second phase of the study, a complementary analytical approach would be to examine whether the use of either parenteral iron preparation was associated with a higher incidence of transient elevation in serum creatinine, which would define...
Response to ‘Bertram Jaber’s letter to the editor’

Kidney International (2008) 73, 512; doi:10.1038/sj.ki.5002758

We did not collect post-infusion serum creatinine, so we are unable to comment on the occurrence of acute renal failure. The time frame of the study is simply too short to detect progression of renal injury. The benefits and risks of iron use in nephrology need to be clarified further. A larger, NIH-funded, randomized trial is under way to examine the effects of iron administration in patients with chronic kidney disease on GFR (glomerular filtration rate) slopes.

R Agarwal

Department of Medicine, Indiana University School of Medicine, Indianapolis, Indiana, USA
Correspondence: R Agarwal, Department of Medicine, Indiana University School of Medicine, 1481 West 10th St, Indianapolis, Indiana 46033, USA.
E-mail: ragarwal@iupui.edu

Gabapentin as a therapeutic option in uremic pruritus

Kidney International (2008) 73, 512; doi:10.1038/sj.ki.5002757

To the Editor: We read with interest the ‘Renal consult’ on uremic pruritus by Keithi-Reddy et al., but we believe that a major point regarding this topic has not been considered by the authors. An emerging and intriguing pathogenetic hypothesis on pruritus suggests that it can be due to peripheral nerve fiber damage associated with a central sensitization, leading to a diminished threshold of perception of itch. This hypothesis is similar to that proposed for neuropathic pain, in which nerve fiber damage and central ‘windup’ phenomenon are thought to be major pathomechanisms. Ongoing studies are now trying to clarify the neurophysiologic pathways shared by itch and neuropathic pain. Gabapentin, a medication widely used for a spectrum of neuropathic pain syndromes, has recently been added to the therapeutic armamentarium of uremic pruritus. Our experience and other published randomized trials have demonstrated that gabapentin can be effective and safe for uremic pruritus, and that it also ameliorates neuropathic symptoms (that is, restless leg syndrome, diabetic neuropathy, and insomnia) that often affect hemodialysis patients’ quality of life. On the basis of these considerations, we believe that Keithi-Reddy et al. should have discussed this point and included gabapentin among the therapeutic options for uremic itch.


L Manenti, A Vaglio and PP Borghetti

Department of Nephrology, ASMN Reggio Emilia, Reggio Emilia, Italy
Department of Clinical Medicine, Nephrology and Health Science, University of Parma, Parma, Italy
Correspondence: L Manenti, Department of Nephrology, ASMN Reggio Emilia, Viale Risorgimento 80, Reggio Emilia 42100, Italy.
E-mail: lucio.manenti@asmn.re.it

Response to ‘Gabapentin as a therapeutic option in uremic pruritus’


In response to our article, Manenti et al. suggest a role for gabapentin in the management of uremic pruritus. They make a similar point in a recent article. Gabapentin is structurally related to γ-aminobutyric acid (GABA). However, gabapentin does not bind to GABA \( \alpha \) or GABA \( \beta \) receptors, and does not appear to influence synthesis or uptake of GABA. High-affinity gabapentin-binding sites have been located throughout the brain and correspond to the presence of presynaptically located voltage-gated calcium channels. This channel appears to modulate the release of excitatory neurotransmitters that participate in epileptogenesis and pain sensation. Hence, gabapentin has been used for the treatment of neuropathic pain. However, its role in uremic pruritus is not well established. In a randomized controlled crossover trial, Gunal et al. evaluated the effect of gabapentin (300 mg given thrice weekly after dialysis) versus placebo in treating 25 hemodialysis patients for pruritus. Gabapentin lowered the mean pruritus score (from 8.4 to 1.2 for gabapentin versus 8.4 to 7.6 with placebo-treated patients). However, this study had several limitations, including a small sample size, limited information on demographics of the study population, subjective evaluation of itching, only