In Reply to 'Hemodialysis Catheters: Which Design Is More Cost-effective?' *and* 'Use of an Uncensored Primary Outcome in a Catheter Design Trial?'

We are pleased to learn that AlBugami et al[1] confirm our most important finding: catheter design has little, if any, influence on catheter survival and incidence of thrombosis/infection. In our large sample (>60,000 catheter-days), we used the strict pre-emptive catheter salvage KDOQI (Kidney Disease Outcomes Quality Initiative) protocol, which resulted in relatively low urokinase use (17-35 episodes/1,000 catheter-days).[2] AlBugami et al used 2 mg (lock) or 4 mg (infusion) of tissue plasminogen activator when blood flow rate was <250 mL/min (M.M. AlBugami, personal communication, March 2015), corresponding with an average of 70 locks or 35 infusions/1,000 catheter-days. Comparing thrombolysis outcome across both studies is very difficult in view of the differences in indication, administration mode, and type of thrombolytic. However, we believe that our pre-emptive strategy is efficient and cost-effective and over-rides any potential initial differences in catheter costs.

The question from Drs Ashby and Corbett[3] most likely originates from a difference in interpretation of the concept of censoring. An observation is "censored" when the observation time has been interrupted prematurely and the time to event (in our case, thrombosis/infection) has therefore not been registered. Because our study reflected standard clinical practice, there was a high degree of early censoring (recovery of kidney function, maturation of vascular access, and transfer to peritoneal dialysis therapy). Primary assisted patency was defined as the interval from access placement to catheter removal for infection/thrombosis, with censoring for non–catheter-related removal.[2] This is entirely different from mean survival time (time to thrombosis/infection if all catheters had been followed up until the thrombosis/infection end point was reached), of which the Kaplan-Meier curve gives only a poor estimate given the high degree of censoring.[4]

An De Vriese, MD, PhD[1]
Hans Van Der Meersch, MD[1]
Dirk De Bacquer, PhD[2]
[1]AZ Sint-Jan Brugge, Brugge, Belgium
[2]Ghent University, Ghent, Belgium

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The Myth of the Future Burden of CKD in United States

To the Editor:

We read with interest the article by Hoerger et al about the future burden of CKD in the United States.[1] These authors accept the notion that as humans age, glomerular filtration rate (GFR) falls.[2-4] Consequently, the application of a fixed and arbitrary threshold of GFR as a definition for "CKD," without reference to other signs of kidney damage (such as albuminuria) will always lead to increased "CKD" prevalence as populations age.[5] Without an age-adapted definition, the prevalence of CKD is overestimated, with a high proportion of the elderly in stage 3a.[6] Prognosis is now considered as a key feature of CKD classification; however, it has been shown that remaining life expectancy is not different between individuals in CKD stage 3a versus those with normal kidney function (see the second figure in Gansevoort et al[7]).

If Hoerger et al wish to suggest that individuals can expect a 1 in 2 chance of developing CKD over their lifetimes, they should make the association of CKD risk and aging clearer. Also they need to explain the gap in "CKD" prevalence between stage 3a (around 20% of population older than 65 years of age) and stages 3b (8%) or 4-5 (<3%); clearly stage 3a neither progresses to more severe stages nor shortens life expectancy.[7]

A solution would be to age calibrate the thresholds of estimated GFR used to define CKD in absence of other signs of kidney damage.[8] Then, the alarming prospects of the simulation from Hoerger et al would take a more subtle and less dramatic hue.

Pierre Delanaye, MD, PhD,[1] *Meguid El Nahas, FRCP, PhD*[2]
Richard J. Glassock, MD[3]
[1]University of Liège, Liège, Belgium
[2]Sheffield Kidney Institute, Sheffield, United Kingdom
[3]Geffen School of Medicine at UCLA, Los Angeles, California

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In Reply to 'The Myth of the Future Burden of CKD in United States'

In our *AJKD* article, we reported estimates of the future prevalence of CKD using existing definitions of the disease.[1] In their letter, Drs Delanaye, El Nahas, and Glassock state that the high prevalence of CKD in persons aged 65 and older is primarily due to aging and does not represent an added mortality risk.[2] A few studies have indeed found that estimated GFR values between 45 and 59 mL/min/1.73 m^2 are not associated with increased mortality.[3] However, other large epidemiologic studies have found that the relative and absolute risks of mortality are higher for the elderly with estimated GFR in this range than for the elderly with greater estimated GFR levels, even after controlling for albuminuria.[4,5] Thus, we believe that CKD staging does provide prognostic information for persons aged 65 and older. We agree with Delanaye et al that not all persons who reach CKD stage 3a will progress to more advanced CKD stages; however, we disagree with their statement that "clearly stage 3a neither progresses to more severe stages nor shortens life expectancy" in persons aged 65 and older.

Thomas J. Hoerger, PhD,[1] *Nilka Ríos Burrows, MPH, MT*[2]
Meda E. Pavkov, MD, PhD[2]

[1]RTI International, Research Triangle Park, North Carolina
[2]Centers for Disease Control and Prevention, Atlanta, Georgia

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Discontinuation of Eculizumab Treatment in Atypical Hemolytic Uremic Syndrome: An Update

To the Editor:

We write to update our previously published report of discontinuing eculizumab therapy after successful treatment of atypical hemolytic uremic syndrome in 10 patients,[1] with longer-term follow-up of the original cases and a report of 6 additional cases. When stable clinical remission had been obtained, patients were offered the choice of continuing or discontinuing eculizumab treatment with the rationale and procedure previously described.[1] Patients had received eculizumab for a median of 4.3 (range, 0.5-14.4) months (Table 1). Eight were able to discontinue dialysis therapy, whereas the other 8 had never been dialyzed. During a cumulative time off treatment of 243 months, 5 patients experienced relapse, identified by means of regular home urine dipstick testing, within 6 months of the last eculizumab dose (an average of 1 relapse per 49 months off therapy). In these patients, eculizumab therapy was restarted, followed by rapid improvement in serum creatinine levels and proteinuria to or below baseline values, and maintained every 3 or 4 weeks based on global complement activity.[2] Eleven patients remained in remission with no signs of acute disease.

In conclusion, we believe that in atypical hemolytic uremic syndrome, it is possible and relatively safe to discontinue eculizumab therapy. In general, we discourage discontinuation of eculizumab therapy in kidney transplant recipients with *CFH* mutations and patients with glomerular filtration rates < 20 mL/min/1.73 m^2. In patients with anti-CFH antibodies, we consider discontinuation of eculizumab therapy when antibody titer is <2.5 times the upper limit of normal. We suggest regular home urine dipstick monitoring for early identification of relapses, especially during acute illnesses and when patients feel unwell.

Gianluigi Ardissino, MD, PhD, Ilaria Possenti, MD
Francesca Tel, MD, Sara Testa, MD, Stefania Salardi, BS
Vito Ladisa, PharmD
Center for HUS Control, Prevention and Management
Fondazione IRCCS Ca' Granda Ospedale Maggiore Policlinico
Milan, Italy
Corresponding author: ardissino@centroseu.org

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