per patient was 50 months (31–108). On susceptibility testing, no resistance was detected in G-sensitive bacteria: the MCI was <4 μg/mL, except in two cases of CVCB due to methicillin-resistant *Staphylococcus aureus*, and one case with BC(−).

No patients had a diagnosis of ototoxicity. The mean trough level of G per patient was 0.17 μg/mL (0.05–0.31). The mean G lock per limb per patient was 3 mg (2–5), equivalent to 1.1–1.7 mg/mL/limb depending on the volume of the limb according to the type of catheter.

Patients diagnosed with CVCB: 11(8.7%). Patients admitted to hospital for CVCB: 4 (3.2%). Number of CVCB/CVC/1000 days: 0.17. CVC removed due to CVCB: 3 patients (2.4%). Mortality due to CVCB: 1 (0.8%). Number of CVCB: 15, *Staphylococcus aureus*: 8; *Staphylococcus epidermidis*: 4; *Escherichia coli*: 1; *Streptococcus bovis*: 1, and BC(−): 1. CVC was removed due to recurrent CVCB in one patient, for failure to improve clinically of in one patient, and due to BC(−) in one patient with clinical remission. There were no other CVCB complications (endocarditis, spondylodiscitis), except in one patient who died due to sepsis.

Discussion. The scientific literature demonstrates that in HD patients, prophylaxis with post-HD antibiotic locking of CVC limbs, including G, reduces morbidity and mortality from bacterial infection associated with CVCB (number of CVCB/CVC/1000 days, mortality and hospital admission due to CVCB) compared with patients with heparin lock alone.[3] Bacterial resistance to G has been reported.[4] However, our experience since July 2003 in patients with CVC attending to the unit and treated with G locking in doses lower than those given in other units (a detail we consider fundamental due to iatrogenic effects), no bacterial resistance or ototoxicity was demonstrated after 9 years of follow-up.[3] Having seen our results, we must refer to the publication by Beathar and Urbanes[1] in which they rate the quality of care of a HD unit according to the number of CVCB/CVC/1000 days it obtains when complete asepsis is employed, an excellent result being a value ≤1. In our case, the practice of complete asepsis + prophylaxis meant that the number of CVCB/CVC/1000 days was 0.17. Although we are unable to compare another study, in 9 years, to obtain a mortality, removal of CVC, and hospital admission due to CVCB of 0.8%, 2.4%, and 3.2%, respectively, is an appreciable standard, obtained thanks to G prophylaxis + universal asepsis. This is further underlined by the absence of endocarditis or spondylodiscitis, except for one patient who died due to sepsis. Strict complete asepsis[5] for all handling procedures of CVC is integral to prophylaxis in reducing morbidity and mortality from bacterial infection associated with CVCB.

Conclusions. This prospective observational study of 9 years' duration in 126 HD patients with a CVC showed: (1) Prophylaxis with intraluminal G locking in CVC limbs does not cause antibiotic resistance in microorganisms sensitive to the antibiotic. (2) There were no diagnoses of clinical ototoxicity, and (3) Prophylaxis with administration of low-dose G (compared with higher doses in other studies)[3] can result in the absence of resistance and ototoxicity.

REFERENCES

1. Beathard GA, Urbanes A. Infection associated with tunneled hemodialysis catheter. Semin Dial. 2008;21:528–38.
2. NKF K/DOQI Guidelines. Clinical practice guidelines for vascular access: guidelines 7: prevention and treatment of catheter and port complications. Am J Kidney Dis. 2006;48 Suppl. 1:S176–247.
3. Fernández Gallego J, Martín M, Gutiérrez E, Cobelo C, Frías P, Jironda C, et al. La profilaxis con sellado de gentamicina de las ramas del catéter venoso central crónico tunelizado no causa resistencia bacteriana. Nefrologia. 2011;31:308–12.
4. Wolley MJ, Taylor SL, Hossain F, Abbas SA, Marshall MR. Association between antimicrobial locks for hemodialysis central venous catheters and antibiotic resistance. Hemodial Int. 2012;16 Suppl. 1:S2–9.
5. Albalate M, Pérez García R, de Sequera P, Alcázar R, Puerta M, Ortega M, et al. ¿Hemos olvidado lo más importante para prevenir las bacteriemias en pacientes portadores de catéteres para hemodiálisis? Nefrologia. 2010;30:573–7.

The relationship between serum and urine NGAL and graft function in pediatric renal transplant recipients

Relación entre niveles de NGAL en suero y orina y función del injerto en pacientes pediátricos trasplantados renales

Sepideh Hekmat[a,*], *Hasan Otukesh*[b], *Nahid Rahimzadeh*[b] *Rozita Hoseini*[b]

[a] *Iran University of Medical Sciences, Iran*

[b] *Iran University of Medical Sciences, Pediatric Transplantation and Dialysis Research Center, Iran*

DOI of original article:
http://dx.doi.org/10.1016/j.nefro.2015.02.005.

* *Corresponding author.*
E-mail address: sepidhekmat@hotmail.com (S. Hekmat).

Dear Editor,

We previously serially measured the serum and urine neutrophil gelatinase associated lipocalin (NGAL) during the first

week after renal transplantation and found that mild ischemic injury may increase this lipocalin.[1] In this study, we investigated the level of this marker after one month post-transplant with the aim of finding a relationship between NGAL quantities and graft function.

Twenty-one pediatric renal transplant recipients without any infection at the time of assessment were included in this study. Glomerular filtration rate was estimated by Schwartz equation and DTPA scan concurrently. Scintigraphic measurement of GFR was performed using an ADAC single-headed gamma camera with the following formula:

$$\text{Total renal uptake percent (\%)} = (k - b)/e - \mu x$$

Pre-post – k: kidney count; b: background count; x: renal depth; e: constant; μ: attenuation coefficient of 99mTc in soft tissue (0.153 cm^{-1}); GFR = total renal uptake percent (%) × 100 × 9.81270 − 6.82519.

The mean age of patients was 9.9 ± 3 years old. Nine patients (43%) were male. The mean time from transplantation was 6.8 ± 2.47 years. The mean serum creatinine was 1.16 ± 0.18 mg/dl. The mean Schwartz calculated GFR was 69.8 ± 12.2 cc/min/1.73 m^2. The mean DTPA measured GFR was 50.6 ± 16 cc/min/1.73 m^2. All patients had GFR less than 90 cc/min/1.73 m^2 by scan and Schwartz formula. The mean serum NGAL was 140 ± 94 ng/ml (15–324 ng/ml). The mean urine NGAL was 17.8 ng/ml (3.2–68 ng/ml).

We assessed the correlation between serum NGAL and serum creatinine, Schwartz GFR, and DTPA-related GFR. The coefficient of correlation with serum creatinine was 0.67 ($P = 0.09$), −0.2 ($P = 0.3$) with Schwartz GFR, and −0.26 ($P = 0.46$) with DTPA GFR. Regarding urine NGAL, the correlation coefficient with serum creatinine was 0.2 ($P = 0.37$), −0.007 ($P = 0.9$) with Schwartz GFR, and −0.24 ($P = 0.48$) with DTPA GFR.

We did not find any significant association between the transplant time and serum NGAL ($r = 0.05$, $P = 0.8$) and urine NGAL ($r = 0.06$, $P = 0.77$). Three patients had slow graft function in this study without need for dialysis in the first week post-transplant. The mean serum and urine NGAL was not different between patients with SGF and those without SGF (for serum NGAL 106 vs 145.5 ng/ml and for urine NGAL 12.2 vs 21.6 ng/ml).

Studies have shown that expression of NGAL protein is significantly increased during ischemic insults in renal transplant recipients with delayed graft function.[2] Magnusson et al. have shown that plasma NGAL levels were significantly higher than normal in renal transplant recipients.[3] Malyszko et al. also found a strong correlation between serum NGAL and serum creatinine in 100 kidney transplant recipients.[4]

This study is the first study in pediatric renal transplant recipients in which the association between serum and urine NGAL with graft function was assessed long term. We did not find any significant association between the amounts of NGAL in serum and urine with serum creatinine and GFR estimated by Schwartz formula or measured by DTPA scan. We think we cannot use serum and urine NGAL as markers of graft function in pediatric renal transplant recipients, but this result needs confirmation by more studies with more cases.

REFERENCES

1. Rahimzadeh N, Otukesh H, Hoseini R, Sorkhi H, Otukesh M, Hoseini S, et al. Are serum and urine neutrophil gelatinase associated lipocalin predictive of renal graft function in short term? Pediatr Transplant. 2012;16:796–802.
2. Mishra J, Ma Q, Kelly C, Mitsnefes M, Mori K, Barasch J, et al. Kidney NGAL is a novel early marker of acute injury following transplantation. Pediatr Nephrol. 2006;21:856–63.
3. Magnusson NE, Hornum M, Jørgensen KA, Hansen JM, Bistrup C, Feldt-Rasmussen B, et al. Plasma neutrophil gelatinase associated lipocalin (NGAL) is associated with kidney function in uraemic patients before and after kidney transplantation. BMC Nephrol. 2012;10:8.
4. Malyszko J, Malyszko JS, Mysliwiec M. Serum neutrophil gelatinase-associated lipocalin correlates with kidney function in renal allograft recipients. Clin Transplant. 2009;23:681–6.