Beta-2-Microglobulin Excretion in Neonates Treated with Gentamicin

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Dear Sir,

In a recent number of Nephron [1], Assadi and Chow-Tung state that 'Persistent elevations in fractional excretion of beta-2-microglobulin (FE β 2M) during gentamicin therapy... may reflect proximal tubule cell injury. Measures such as interrupting treatment or lowering the drug dosages are indicated if permanent damage is to be avoided'.

The fact that aminoglycosides (AMG) increase the excretion of beta-2-microglobulin (β 2M) is well known [2, 3]. It is also recognized that measurement of this protein's excretion in urine does not discern accurately between tubular damage induced by AMG or by other causes [4]. The authors' patients have statistically significant differences in FE β 2M among the two groups, but they could find no differences when monitoring other parameters during 14 days. Thus, one can state that a raise in β 2M excretion is a marker of tubular dysfunction, but does not correlate with the development of clinical nephrotoxicity. Besides, I do not think that the authors have demonstrated that an increase in the excretion of β 2M, followed by treatment interruption or modification, prevents the appearance of nephrotoxicity, simply because they have not done so in their paper. Statements like this need to be confirmed by means of well-controlled studies. The authors also write that 'serum aminoglycoside levels are even less sensitive markers of tubular damage than serum creatinine concentrations', and I do agree with them. Nevertheless, monitoring of AMG levels has not been developed as a measure of tubular dysfunction or necrosis, but as a way to prevent it from appearing.

The finding of a valid marker of tubular damage during AMG treatment is certainly both necessary and desirable. Nevertheless, when dealing with patients, we need to know that modifications in the excretion or concentration of the substance will correlate with an increased risk of developing clinical nephrotoxicity. No marker like this has been described yet, although amy-lase-creatinine clearance ratio seems to have a promising future [5]. On the other hand, maintaining of trough serum AMG levels in nontoxic concentrations is a reasonable, though not infallible, way of preventing nephrotoxicity [6–8].

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