Renal failure in the neonate associated with in utero exposure to non-steroidal anti-inflammatory agents

Key words: Renal failure – Fetus and neonate – Indomethacin

Sirs,

Kaplan et al. [1] documented five patients with renal insufficiency related to intrauterine exposure to non-steroidal anti-inflammatory agents. We described six cases of irreversible renal failure in such patients and performed immunohistochemical studies of renin distribution on five renal biopsy specimens. In four cases we found a remarkable increase in renin content in the juxtaglomerular apparatus compared with that observed in specimens of normal singleton and twin controls [2]. Fetuses with suboptimal circulation, as can be the case in one of the twin pair in twin pregnancies as well as in fetuses under stress conditions leading to preterm labour, may have high levels of circulating angiotensin II, favouring the vasoconstrictory effect of prostaglandin inhibitors. Our findings strongly support this hypothesis for the underlying mechanism of renal insufficiency and renal histological changes in these neonates.

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References

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2. Heijden AJ van der, Carlus C, Narcy F, Bavoux F, Delezoide AL, Gubler MC (1994) Persistent anuria, neonatal death, and renal microcystic lesions after prenatal exposure to indomethacin. Am J Obstet Gynecol 171: 617–623

Improved absorption of cyclosporin A from a new microemulsion formulation: implications for dosage and monitoring

Key words: Cyclosporin A – Pharmacokinetics – Renal Transplant – Side-effects

Sirs,

Thank you for publishing our paper in the April issue of Pediatric Nephrology.

Unfortunately the formula for the calculation of the AUC stated in the text is incomplete which might lead to false results if this formula is applied by other groups.

The correct formula, which we used for the analysis of our data, should read:

$$AUC = \sum_{i=1}^{n-1} (c_{ti} + (c_{ti+1} - c_{ti})/2) \cdot (ti + 1 - ti)$$

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