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LETTER TO THE EDITOR

Poiseuille's law in polyuria

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Dear Sir,

In their recent paper, Caletti et al. describe urinary tract findings in children with nephrogenic diabetes insipidus (NDI) and show that seven out of ten patients have renal pelvic dilatation that improved or even normalized during follow-up [1]. Treatment with a low osmolar diet in combination with diuretics and indomethacin reduced the urine output from 10.5 to 4.4 ml/kg/h.

This transition in urine production is similar to the physiological changes around birth. In the last weeks of pregnancy, a fetus produces on average 10–15 ml/kg/h of urine. Indeed, fetal renal pelvic dilatation is regularly found, making it the most frequently found abnormality during prenatal ultrasound screening [2]. In approximately 50–70 % of cases of antenatal hydronephrosis, postnatal follow-up shows no significant urinary tract abnormalities and are therefore labeled as transient or physiologic. Postnatal renal ultrasound will even be without any abnormalities in about 21–28 % of cases, generally associated with only mild antenatal hydronephrosis [2]. In analogy with NDI during treatment, urine production after birth is greatly reduced to around 4 ml/kg/h, which will further decrease with increasing age.

A likely explanation for the reduction in renal pelvic dilatation with a reduction in urine volume, the common denominator, can be found in Poiseuille's law stating that a higher flow through a tube will increase the pressure. When applied to the urinary tract, polyuria will lead to a higher pressure in the renal pelvis, which generally results in dilatation. With the use of the Whitaker test, a higher flow rate was indeed found to lead to an increase in renal pelvic pressure, and some patients only showed signs of obstruction at higher urine flow rates [3]. Dilatation of the upper urinary tract can be exacerbated by suboptimal emptying of the bladder, as was rightfully stated by Caletti et al. [1].

Whether the genetic mutation leading to NDI has an additional influence on the development of the urinary tract, and therefore directly results in hydronephrosis, remains to be determined. The absence of developmental defects of peristalsis in the renal pelvis of animal models of NDI, such as mice with an aquaporin-2 mutation [4], seems to suggest that this does not play a role.

Normal fluid physiology provides a perfect explanation for the urinary tract findings in NDI, and therefore reducing urine volume and optimizing voiding habits remains the cornerstone of treatment for hydronephrosis in NDI patients.

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