Re: Diagnostic Accuracy of Renal Pelvic Dilatation in Determining Outcome of Congenital Hydronephrosis

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Dear Editor,

Sharifian and colleagues recently reported in the *Iranian Journal of Kidney Diseases* the diagnostic accuracy of postnatal renal pelvic dilatation in determining the outcome of prenatally-detected hydronephrosis in 178 newborns included in a 4-year period. In particular, the authors focused their research on the ability of a specific postnatal renal pelvic dimension to predict the need for postnatal surgery, ie, pyeloplasty.[1] This is a welcome study, and it emphasizes the importance of following such newborns in the postnatal period, with renal ultrasonography and other appropriate imaging. However, after careful reading, we have the following comments to the authors.

This is not the first study on that matter. Several authors already tried to find predictors of postnatal surgery requirement in newborns with prenatally detected hydronephrosis. Longpre and colleagues[2] found that initial anteroposterior pelvic dimension was the only independent predictor of postnatal pyeloplasty. Dias and colleagues[3] found that a combination of pre- and postnatal renal pelvic measurement was able to increase the prediction for postnatal surgery. Similarly, Yang and coworkers[4] used both postnatal ultrasonography and isotope diuretic renography to predict, again, the need for pyeloplasty. Decramer and colleagues[5] studied the accuracy of urinary proteomics in predicting the need for surgical ureteropelvic junction obstruction (UPJO) relief. It should be noted that in all these above-mentioned studies, and many others, newborns with other diagnosis than ureteral obstruction, such as vesico-ureteral reflux (VUR), multicystic dysplastic kidney (MDK), or posterior urethral valves, were all excluded. This only makes sense since these newborns, although presenting with similar symptoms in the prenatal period, can be readily and easily distinguish from newborns with UPJO in the postnatal period. Having a more homogenous population will only increase the positive and negative predictive value of the cutoff value the authors found,[6] its accuracy,[7] and to a lesser extent the sensitivity and specificity of the test and the area under the curve.[8-10]

Therefore we think the authors missed an opportunity to easily improve the performance of their postnatal ultrasonographic cut-off value they found by retaining newborns with VUR or MDK. Voiding cysto-urography allows clinicians to rapidly differentiate infants with VUR from infants with obstruction. Furthermore, the management of infants with VUR is totally different from infants with obstruction. Unless VUR is severe, they are treated conservatively, sometimes with antibioprophylaxis, rarely with surgery, and not with pyeloplasty. In the same vein, infants with MDK usually present already in utero with characteristic ultrasonographic findings, which easily differentiate them from patients with obstruction.[11] Very occasionally, newborns with MDK can be confused with an upper urinary tract obstruction if a single large cyst is present.[12] Was it the case with patients included in their study? Even then, radionuclide study should make the correct diagnosis and classification. Finally, infant with MDK do not benefit from surgery, and their management is mostly conservative.

The goal of this study was to find predictors of surgery (pyeloplasty) for obstruction (UPJO). Including newborns with conditions that will never benefit from pyeloplasty, and that can readily be distinguish from UPJO, will only dampen their findings. There is no doubt that the predictive performance of their test will dramatically increase with a more homogenous population.

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Re: Association of Leptin With Mortality in Patients on Maintenance Hemodialysis: a Prospective Study

Dear Editor,

We Read with interest an article published by Bian and colleagues in the previous issue of the Iranian Journal of Kidney Diseases on leptin in dialysis patients.[1] Leptin, a protein produced by the *obese* gene is expressed, synthesized and secreted by white adipocytes. The exact pathophysiology of leptin is unclear. Metabolic effects of leptin include a wide range. It regulates adipose tissue mass and body weight by a feedback mechanism.[2-4]

In addition to the main known effects of leptin, recent studies have demonstrated that leptin has a wide variety of effects in different tissues.[5] These different functions develop through its action in peripheral tissues or in the central nervous system. Some other lines of evidence which consider the renal clearance as the main route for leptin metabolism propose that the calculated plasma half-life for leptin and molecular weight are as same as the other peptide hormones that are degraded by the proximal renal tubule. Moreover, recent studies showed that the leptin receptors are often expressed in the lung and the kidney.[6]

Leptin may act in relation to cardiovascular homeostasis. It indicates its potential role in cardiovascular system via p38 MAPK.[7] Leptin takes part in the regulation of sympathetic tone and arterial blood pressure.[8] Also, elevated circulating plasma leptin level revealed to be in association with chronic intravenous infusion of leptin.[9] Various mechanisms are suggested for leptin-induced atherosclerosis namely, endothelial dysfunction after great amounts of nitric oxide, induction of a pro-inflammatory state, abnormal lipid metabolism in the vessel wall, increased proliferation and migration of vascular smooth muscle cells, and increased platelet aggregation and abnormal haemostasis.[10,11]

Leptin is cleared by the kidney. As a result, the plasma leptin concentration rises in patients with end-stage renal disease. Leptin has an impact on the kidney through different mechanisms such as modulation of growth, induction of transforming growth factor-□, natriuresis, upregulation of