addition, the pathological process in the affected pediatric cases is owing to the acute obstruction from melamine crystal that is unlikely to cause proteinuria. Therefore, the rational for screening for proteinuria in pediatric cases exposed to melamine is not supported. Some limitations of this work should be noted. First, this is a bioinformatics study that still requires further in vitro and in vivo confirmation. Second, there are some new information on uric acid crystallized melamine in urine, but it is not observed in all cases and is not studied in this work due to lack of basic information on its structure.

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Reno-renal Syndrome
Cross-talk Between Kidneys

SIR,

Various pathophysiological syndromes have thronged nephrology. These include hepato-renal, pulmonary-renal, and cardio-renal syndromes. We herein describe a new syndrome characterized by injury (partial or total) to one kidney, cumulating in pathophysiological changes in the other, and we shall now call it as *reno-renal syndrome*. The notion that a single kidney enlarges to compensate for the loss of its partner has been entertained since antiquity. Aristotle (384 to 322 BC) noted that a single kidney was able to sustain life in animals, and that such kidneys were enlarged. The enlargement of the glomeruli is mainly due to increase in volume. Numerous hypotheses accounting for this renal hypertrophy include large amount of metabolic solute load, renotropic factors such as renotropin, hormones, and growth factors such as insulin-like growth factor I, vascular endothelial growth factor, etc. It appears that there is increased sensitivity of kidney cells to growth promoting factors.

Both hemodynamic and nonhemodynamic responses occur after renal mass ablation. The hemodynamic responses comprise of renin-angiotensin-aldosterone system, endothelines, eicosanoids, natriuretic peptides, bradykinin, and nitric oxide. Nonhemodynamic factors again include angiotensin II, aldosterone, and others such as transforming growth factor β, bone morphogenic protein-7, hepatocyte growth factor, and altered glomerular permselectivity to proteins.[1]

The common clinical conditions which manifest as reno-renal syndrome include: postuninephrectomy condition, unilateral renal dysplasia, unilateral renal agenesis, oligomeganephronia, segmental hypoplasia, vesicoureteric reflux, the condition following urinary diversion, accident-related reduction of renal mass, condictions associated with renovascular disease, unilateral obstruction, and cortical necrosis.[2] The end results of these pathologic syndromes are proteinuria, hypertension, and progression to chronic kidney disease by means of focal segmental glomerulosclerosis. Therefore, interventions aiming to control of hypertension, dyslipidemia, weight gain, anemia, calcium-phosphate metabolism, decrease protein intake, and renin-angiotensin-aldosterone system blockade may not only prevent but may also reverse reno-renal syndrome.

To summarize, reno-renal syndrome is a

pathophysiological syndrome resulting from injury to one kidney affecting the other. Early suspicion for development of this syndrome and adequate interventions may prevent the increase in the number of patients with chronic kidney disease. Internists and surgeons should be aware of this entity so that timely preventive and curative measures can be taken.

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Erratum

In volume 3, number 3 of the *Iranian Journal of Kidney Diseases*, the following error occurred: on pages 164 and 165, the unit for the oxidized LDLC in Tables 1 and 2 should have read *U/L* instead of mg/dL. The editor regrets this error.