the lysoPLD activity [8,9]. The increase of the LPA concentration (upon LPC addition) was correlated well with the lysoPLD activity in the urine samples (Figure 2B).

Our present results suggesting that lysoPLD activity exists in the urine and that LPA can be formed in the urine may be important from the perspective of the reported results of a number of studies on the effects of exogenous LPA on renal systems. Sphingosine 1-phosphate is the major sphingosine-based lyosphospholipid, structurally similar to LPA. Recent evidence has implicated this sphingolipid mediator in the (patho)physiology of kidney diseases [10]. Lyosphospholipid, a relatively new family of bioactive lipids, may play important and hitherto unexpected roles in the pathophysiology of kidney diseases, and further studies must be conducted in this field.

Conflict of interest statement. None declared.


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Efficient treatment of crescentic glomerulonephritis associated with hepatitis B virus with lamivudin in a case referred with acute renal failure

Sir,

Glomerulonephritis (GN) associated with hepatitis B virus (HBV) and concomitant acute renal failure (ARF), though rare, has been previously reported [1]. In recent years, lamivudine has been used frequently in GN cases related HBV due to its few side effects and ease of use [2,3].

Case. A 40-year-old female patient referred to our clinic with nausea, vomiting, oedema in the lower extremities and reduced urine amount. Laboratory findings were: urea: 212 mg/dl, creatinine: 9.4 mg/dl, albumin: 29 g/l, AST: 414 U/l, ALT: 261 U/l and 3206 mg/day proteinuria. C3, C4 and Clq were normal. Antinuclear antibodies (ANA), pANCA, cANCA, and glomerular basement membrane antibody were detected as negative. Serological tests revealed: HbsAg (+), HBeAg (+), AntiHbs (−), AntiHbc (−), AntiHbc IgM (−) and HBV DNA was detected to be $5.81 \times 10^5$ copies/ml. A renal biopsy had shown global sclerosis (7 of the 10 glomeruli), crescent formation and mesangial proliferation with IgA and C3 deposits in the glomeruli. Monoclonal antibodies gave positive results with HbsAg in the glomeruli. Treatment was planned with interferon-α (IFN-α). Because of an anaphylactic reaction observed after the first dose, IFN-α therapy was stopped, and the patient was followed-up with lamivudine treatment and haemodialysis 3 days a week. Thirty-six weeks after the onset of 30 therapy, HBV DNA levels were 454 copies/ml, creatinine levels were 1.5 mg/dl, and the proteinuria level was 334 mg/day. Though in low titers, Anti-HBs antibody turned positive. Renal biopsy was repeated in this period. Global sclerosis (6 of the 14 glomeruli), mild or moderate mesangial proliferation with IgA deposits were observed in glomeruli. Monoclonal antibodies were detected as negative with HbsAg. Patient was still in the 20th month of the therapy and levels of creatinine were observed as 1.9 mg/dl.

Comment. Antiviral therapy has taken first place in the treatment of HBV-related GN [1]. Our case is the first adult case demonstrating crescentic GN and ARF secondary to HBV, in which an efficient improvement in renal functions, if for short duration, was obtained with lamivudine therapy. Taking into consideration other data in the literature, we thought that lamivudine might be an alternative for its advantages, particularly in developing countries and in cases where IFN-α cannot be used due to its side effects.

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**Utility of cystatin C measurement—precision or secretion?**

Sir,

Recently, van Rossum and colleagues [1] evaluated the renal extraction of cystatin C compared with $^{125}$I-iothalamate in 40 patients with unilateral renal artery stenosis. The authors reported that the mean difference between these two measures of glomerular filtration rate was small (0.002); however the limits of agreement by Bland and Altman technique [2] were quite large (−0.271 to 0.267). The authors concluded that this difference may reflect previously unrecognized tubular secretion of cystatin C and that cystatin C may not be a useful endogenous measure of kidney function.

The cystatin C assay employed by van Rossum and colleagues [1] uses a turbimetric immunoassay which has relatively poor intra-assay precision [3]. Using their controls, the authors report an intra-assay coefficient variation of 11%, substantially greater than that for creatinine [1]. The authors repeated cystatin C measurements in triplicate. Whereas this may improve the precision of the mean value, it would do little to improve the variation between measurements. Cystatin C is freely filtered at the glomerulus [4], more than 99% is catabolized by proximal tubular cells [5], and the urinary concentration of cystatin C is very low under normal physiological conditions [6]. Perhaps the imprecision in measurement of cystatin C led to the larger limits of agreement, rather than previously unrecognized renal tubular secretion. A nephelometric assay that has lower coefficients of variation is commercially available [7]. Would the results have been similar with this assay?

Despite its limitations, creatinine measurement is precise, readily available, and inexpensive. Cystatin C may eventually prove useful in clinical situations where serum creatinine concentrations might erroneously mislead clinicians to believe that the glomerular filtration rate is normal, rather than as a universal replacement of serum creatinine and associated derived estimating equations. Examples where cystatin C measurement might prove most useful include evaluation of kidney function among persons with advanced age [8] and low muscle mass [8,9], and among persons with normal or near-normal glomerular filtration rate [10].

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