Nephrol Dial Transplant (1996) 11: 1484–1500

Nephrology Dialysis Transplantation

Letters

MPGN and HCV infection in Istanbul, Turkey

Sir,

In a recent editorial in this journal D'Amico [1] discussed the possible link between HCV infection and non-cryoglobulinaemic membranoproliferative GN. In support of this argument Professor D'Amico cites the experience of Johnson *et al.* [2] in the USA and Yamabe *et al.* [3] in Japan, who found this to be a common association amongst their patients. He then goes on to point out that this association is infrequent in Southern Europe, notably France [4], Spain [5], and Italy [1].

With 2nd-generation ELISA testing, of 36 patients (24 males, 12 females; mean age 32 ± 12 years) with biopsy-proven membranoproliferative GN presenting at our outpatient clinic in the last 2 years and followed for a mean of 11 ± 6 months, no patient has been demonstrated to have HCV infection. HCV infection is, however, a problem on our haemodialysis unit, where at present 25% of patients are infected. Additionally, approximately 1.5 % of blood donors screened at our hospital blood bank are HCV-antibody positive. Athough we do not yet know the incidence of cryoglobulinaemia amongst our patient group, our experience appears to reflect that of Southern Europe rather than that of the USA or Japan.

Departments of Nephrology and Haematology, Marmara University Hospital, Istanbul, Turkey

R. Lawrence
Ç. Özener
M. Çobanoğlu
N. Şahin
E. Akoğlu
M. Bayık

1. D'Amico G. Is type II mixed cryoglobulinaemia an essential part of hepatitis C virus (HCV)-associated glomerulonephritis? *Nephrol Dial Transplant* 1995; 10: 1279–1281
2. Johnson RJ, Gretch DR, Yamabe H *et al.* Membranoproliferative glomerulonephritis associated with hepatitis C virus infection. *N Engl J Med* 1993; 328: 465–470
3. Yamabe H, Fukushi K, Ohsawa H *et al.* Hepatitis C virus (HCV) infection may be an important cause of membranoproliferative glomerulonephritis (MPGN) in Japan (abstract). *J Am Soc Nephrol* 1993; 4: 291
4. Rostoker G, Deforges L, Ben Maadi A *et al.* Low prevalence of hepatitis C antibodies among adult patients with idiopathic membranoproliferative type 1 glomerulonephritis in France. *Nephron* 1995; 69: 97
5. Gonzalo A, Fernandez M, Navarro J, Ortuno J. Searching for hepatitis C virus antibodies in chronic primary glomerular diseases. *Nephron* 1995; 69: 96

Iatrogenic acute oxalate nephropathy

Sir,

Further to C. G. Winearls' recent Editorial Comment on iatrogenic acute oxalate nephropathy [1], it is of interest to recall that the use of pharmaceutical agents which contain piridoxilate may induce oxalate nephropathy.

Dequiedt *et al.* [2] reported in 1985 a case of acute oxalate nephropathy in an individual who had taken 6 g of piridoxilate in an attempted suicide, and two cases of chronic oxalate nephropathy in patients receiving long-term treatment with piridoxilate have been reported, also by French authors, a few years ago [3,4]. Like those patients with naftidrofuryl-induced oxalate nephropathy, these patients had a rise of serum and urinary oxalate concentrations and crystalluria. Renal biopsy showed tubulo-interstitial nephropathy with calcium oxalate deposition.

Piridoxilate is an association of glyoxylic acid and pyridoxine. Glyoxylic acid is mainly metabolized *in vivo* to oxalic acid, pyridoxine being supposed to facilitate its transformation to glycine rather than to oxalic acid. However, piridoxilate administration causes a rise of serum and urinary oxalate concentrations. These observations are important: indeed, if the two French pharmaceutical agents that contained piridoxilate have been withdrawn, some similar preparations may still be on the market in other countries.

Service de Néphrologie
Hôpital Bichat
Paris
France

J. P. Méry

1. Winearls CG. Iatrogenic acute oxalate nephropathy. *Nephrol Dial Transplant* 1995; 10: 2171
2. Dequiedt Ph, Gosselin B, Benoit O *et al* Insuffisance rénale aiguë par oxalose aiguë après ingestion massive de piridoxilate. *Néphrologie* 1985; 6: 228–230
3. Vigeral P, Kenouch S, Chauveau D, Mougenot B, Méry JPh. Piridoxilate-associated nephrocalcinosis: a new form of chronic oxalate nephropathy *Nephrol Dial Transplant* 1987; 2: 275–278
4. Mousson Ch, Justrabo E, Rifle G, Sgro C, Chalopin JM, Gérard C. Piridoxilate-induced oxalate nephropathy can lead to end-stage renal failure. *Nephron* 1993; 63: 104–106

When dialysis becomes worse than death

Sir,

The review of Sessa [1] focuses on a problem in which nephrologists are increasingly involved. The increasing number of patients with advanced age or severe and multiple comorbid conditions can strongly reduce the quality of life and favour the consideration of the opportunity of withdrawing dialysis treatment. The article outlines the different rate of withdrawal from dialysis between US and Europe and points out the cultural, ethical, and legal factors that can influence the decision of stopping dialysis, problems which are largely debated in US or in Canada [2].

As European nephrologists we wonder if we should really hope that cultural changes will favour an increase in the choice of stopping dialysis. In particular we believe that implementation of 'living wills' or advance directives is ethically questionable. They are based on the principle of autonomy whose value is well recognized for the competent patient, but the value of these acts cannot be considered permanent and the possibility that the opinion of the patient may have changed over time cannot be excluded. Some people may be induced to subscribe such directives for depressive conditions or moved by the fear that the disease may involve an excessive burden for their family. In addition many problems remain unsolved: when and who has the right to say at what moment the patient had decided to die.