that we started a preventive programme based on universal precautions to minimize the risk of exposure to hepatitis C virus (HCV), with good results [2]. Actually, from 1990 to March 1997 we had no cases of seroconversion to HCV antibodies among new patients starting haemodialysis treatment. Surprisingly, from March 1997 to January 1998 we had five new cases, as reported in Table 1. We were seriously considering the possibility of a new cluster of HCV infection which might have been favoured by our lowering attention for universal precautions of blood-borne infections.

In all our patients who had positive HCV antibodies (Abbott HCV EIA 3.0, Abbott Labs, USA) we analysed HCV genotype with inverse hybridization (Ambicor extraction, Roche, immunoblotted Innolipa, Nuclear Laser) and classified them as suggested by Simmonds et al. [3]. In January 1997 we had 36/143 haemodialysis patients with HCV antibodies. We found genotype 1B in 16/36 (44.4%), genotypes 2 in 15/36 (41.6%), and undetermined genotype in 5/36 haemodialysis patients (13.9%). In our geographical area the presence of HCV antibodies is 3–6%, depending on age. We therefore controlled the distribution of different genotypes in a non-selected, successively enrolled group of 453 patients with community acquired HCV infection. We found genotype 1A in 41/453 haemodialysis patients (9.1%), genotype 1B in 204/453 (44.9%), genotypes 2 in 158/453 (24.9%), genotype 3A in 33/453 (7.3%), genotype 4 in 5/453 (1.1%) and undetermined genotype in 12/453 (2.6%).

We then controlled the day and turn of dialysis of all five patients from June 1996 to January 1998. We found that only patients No. 4 and 5 were regularly dialysed in the same room and turn. Moreover, the genotyping of our five new cases revealed different HCV subtypes.

We conclude that this apparent cluster of HCV infection was not an epidemic but corresponded to independent infections, possibly related to surgical procedures (patients Nos 1, 3 and 4) or community acquired infection (patient No. 2). Only one out of the five cases was potentially acquired in the dialysis unit. After January 1998 we followed the usual procedure to avoid exposure to HCV and until January 1999 no other cases have occurred. This observation led to a debate with dialysis staff as to the importance of respecting universal precaution measures in the unit. We think that it is useful to maintain permanent alertness on blood-borne infections.

Apparent epidemic of HCV infection in a haemodialysis unit

Sir,

Haemodialysis units have been recognized as a high-risk environment for the transmission of blood-borne infections. In our unit we had an outbreak of non-A, non-B hepatitis involving over 40 patients between 1980 and 1985 [1]. After

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Table 1.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age</th>
<th>Sex</th>
<th>Date of last negative HCVAb</th>
<th>Date of GPT elevation</th>
<th>Date of first positive HCVAb</th>
<th>Date of blood transfusions and units</th>
<th>Surgical procedure</th>
<th>HCV genotype</th>
</tr>
</thead>
<tbody>
<tr>
<td>2 B.F.</td>
<td>20</td>
<td>F</td>
<td>Oct 1996</td>
<td>Apr 1997</td>
<td>Apr 1997</td>
<td>None</td>
<td>None</td>
<td>1B</td>
</tr>
<tr>
<td>5 P.R.</td>
<td>49</td>
<td>F</td>
<td>Oct 1997</td>
<td>Jan 1998</td>
<td>Jan 1998</td>
<td>None</td>
<td>One finger amputation None</td>
<td>2C</td>
</tr>
</tbody>
</table>

*All donors controlled in January 1998 remained negative for HCV antibodies.
and azathioprine generally suppress all immune responses, function:
correlation of histopathology and transplant outcome.

Dial Transplant and invasion of leukocytes in renal tissues cause injury by
parameters creates confusion. graft function after renal transplantation in patients undergoing

et al. advised to use a highly biocompatible membrane for
acute renal failure after cadaveric renal transplantation

E et al. et al. in the study, if biopsies had confirmed a diagnosis of ATN.

statement, that ARF after renal transplantation is a good
three had died, cause not given) contradicts the authors' in critically ill patients [9,10].

had primary non-function, two had vascular thrombosis and cellulosic membranes adversely a
the study (three patients had biopsy proven rejection, one the hypothesis presented in 1994, namely that the use of
incidence of dialysis dependent ARF (53 out of 95, 56%) as of ATN after renal transplantation. The data of these authors
between 20–30% [4,5]. In Romao's study, both the high per se or choice of membrane has an impact on the course
post-transplantation phase. The prevalence of ATN after of IDGF [8]. The study by Romao

et al. et al. Ect of dialyser biocompatibility on recovery from

blood–membrane interactions resulting in complement- peritoneal dialysis and hemodialysis.

J Gen Virol phylogenetic analysis of the NS5 region.

Nephrol Dial Transplant outbreak of Non-A, Non-B hepatitis in centre haemodialysis reasons to argue that immunosuppressive drugs could attenu-


