Table 1
Demographic, laboratory and clinical characteristics of all patients at baseline

N patients/HCV genotype 1b (%)	16/16 (100%)
Male (%)	12 (75%)
Age (median years, range)	47 (25–64)
Duration of haemodialysis (median years, range)	14.5 (1–28)
HCV RNA (median IU/mL, IQR[a])	18350 (910–469,000)
Outcome of antiviral therapy (SVR/NR/R[a])	11 (68.8%)/3 (18.8%)/2 (12.5%)
Previous renal transplant (*N*, %)	13 (81.3%)
Retained graft *in situ* (*N*, %; median months, range)	6 (37.5%); 14 (4–240)

[a] *Abbreviations:* IQR, interquartile range; SVR, sustained virologic response; NR, non-responder; R, relapse.

hepatitis C with intermediate necroinflammatory activity and fibrosis. Antiviral therapy was begun in June 2006. Despite the withdrawal of ribavirin at week 4 due to anaemia, the patient attained SVR. However, ten weeks after the onset of therapy, he experienced chills, fever and a painful graft. Antiviral treatment was stopped at week 14 and the patient underwent graftectomy. Compatible with the acute-on-chronic allograft rejection, the histology showed almost complete haemorrhagic necrosis of the graft.

In line with previous case records [3,4] and a recent cohort study [5], and contrary to the study of Rendina et al. [2], we conclude that even haemodialysed patients with non-functional renal allograft retained *in situ* may experience acute allograft rejection following peginterferon-α2a treatment of hepatitis C. Before the start of antiviral therapy, these patients may need to be advised of this eventuality and should be aware of the possible need for graftectomy. In patients who present with graft failure less than one year prior to treatment with peginterferon-α and who seem to be most prone to acute rejection, it may seem reasonable to endorse preemptive graftectomy.

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Acute rejection of non-functional allograft in kidney transplant recipients with hepatitis C treated with peginterferon alpha-2a: Reply

To the Editor:

We have read very carefully the Letter to the Editor by Dr. Sperl and collegues and the answers to the questions they raised are provided below:

1. In our series, of the 19 haemodialysed patients, who had previously been transplanted, 14 had retained the graft *in situ*. Five patients, before treatment, had already been submitted to graftectomy on account of symptoms such as, for example, acute rejection which occurred after a mean time of 3–7 months after graft failure. As far as concerns immunosuppressive treatment, the policy in our centre is to stop Calcineurin inhibitors and to taper steroids within 3 months of graft failure. Our patients received anti-viral treatment only after a mean period of 12–36 months after graft failure.
2. In the series described by Dr. Sperl, interferon treatment was started very early after graft loss (i.e., 4 and 8 months) and no information is provided regarding either immunosuppressive treatment or the immunological background of the two patients. We chose to start treatment after a period of clinical and immuno-

logical stabilisation (>12 months after restarting dialysis) on the basis of previous experience showing that most cases of rejection occurred before this time interval.

With regard to the rate of side-effects, this may be due to the fact that a high dose of interferon was used by the authors, while ribavirin was avoided in most cases. Conversely, and as reported in our paper, we carefully assessed the type and dosage of pegylated interferon (i.e., peginterferon alpha-2a, 135 μg/weekly) due to the presence of chronic renal failure and individually tailored the dosage of ribavirin to avoid even more severe anaemia which was controlled by administration of erythropoietin. This strategy resulted in a strong anti-viral effect and avoided, in most instances, major side-effects and, probably, also rejection.

To better define the use of graftectomy, further studies, on a larger series, are necessary focusing on comparisons between the rejection rates in patients receiving interferon and those not receiving interferon. On the basis of our experience and available data, anti-viral treatment should, in our opinion, be delayed until 12 months after graft loss.

We wish to thank Dr. Sperl who gave us the opportunity to emphasize these points which complete our results and would, no doubt, contribute to improving the pharmacological approach with pegylated interferon and ribavirin in this particular category of patients.

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Neonatal classical galactosaemia presenting as citrin deficiency

To the Editor:

We read with interest the recent study by Feillet et al. [1]. This is an elegant paper which eloquently analyses and explains what turned out to be a secondary metabolic abnormality. We agree that neonatal citrin deficiency needed to be considered as a differential diagnosis, albeit a rare one. Clinical presentation of citrin deficiency is heterogeneous and non specific and galactosaemia is a well recognised biochemical feature. Although citrullinaemia is common it is not specific, may be absent and the diagnosis of citrin deficiency today largely depends on mutation analysis.

In neonatal liver failure, in particular, it is often difficult to distinguish between what are primary metabolic abnormalities causing liver dysfunction and secondary abnormalities as a result of liver dysfunction. The situation can become particularly complicated when there has been a delay in diagnosis or partial treatment as in this case.

In the setting of such a complex multi-systemic disease, it is important to have some simple guiding principles. Galactosaemia is one of the commonest causes of neonatal liver failure. A galactose free diet should be given to all children with neonatal liver failure until galactosaemia has been specifically excluded by enzyme analysis. A galactose challenge is neither appropriate nor necessary as a diagnostic test for galactosaemia and is not without risk. An approach to the diagnosis or exclusion of possible galactosaemia when the situation is complicated is summarised in a recent review [2].

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