in the negative group of patients, three (7.8%) had current raised AST values and two (5.3%) had current raised ALT levels (AST and/or ALT > ULN). These increased levels had three negative HD patients). We demonstrated that a significant proportion (40.6%) of HD patients negative for other blood-borne viruses such as HBV, HCV, HIV and HTLV-I/II, tested positive for GBV-C/HGV infection markers. It is noteworthy that contrary to what was found in previous studies [7–10], the presence of these markers was significantly associated with lower prevalence of markers of previous HBV infection, lower rate of previous history of blood transfusions and lower mean number of transfused units. Our findings could be attributed to the particular composition of our study group since the latter consisted of individuals without any sign of infection with several blood-borne viruses, and to the inclusion of a significant percentage of HCV infected HD patients in most of the former studies. The lack of association between GBV-C/HGV infection markers and parenteral transmission events in our study, probably indicates that although GBV-C/HGV can be transmitted by transfusions [1,7], acquisition in the HD unit by other means, like nosocomial ones as it has already been reported for HCV [12], should be kept in mind. These patients undergo many percutaneous and intravenous procedures having disease-associated and sometimes iatrogenic immunosuppression, which may facilitate the transmission of GBV-C/HGV. Alternatively, although our study does not establish how HD patients contracted the virus, a very high rate of contacts with contaminated devices or persons such as the staff, seems attractive as it has been suggested recently by our group and others for hepatitis E virus infection [6,13]. The possibility of community-acquired or patient-to-patient transmission of GBV-C/HGV in HD patients has already been suggested [10]. We did not observe any significant difference of transaminases values between GBV-C/HGV-positive and -negative HD patients. These findings are similar with that reported by others [7–9] particularly when the HD patients were HCV negative. It remains unclear, therefore, whether GBV-C/HGV may be a causative agent for liver disease in HD patients. Case-control studies are required to confirm whether or not GBV-C/HGV is an aetiologic agent of viral hepatitis or any other disease. Anti-E2 and GBV-C/HGV-RNA were found to be mutually exclusive confirming the notion that anti-E2 has to be considered as a marker of past infection [7,9,14]. A surprisingly high frequency of GBV-C/HGV-RNA detection was also recorded very recently in groups other than HD patients without known risk factors for blood-borne viral infections, indicating that chronic GBV-C/HGV infection might also be transmitted by social or sexual contacts or other routes yet to be defined [15]. Therefore, in the HD setting GBV-C/HGV infection may be an indirect marker for potentially intraunit transmission of viral diseases since new infections are detected in the absence of blood transfusions. More studies, however, are needed in order to assess the transmission routes other than transfusions (e.g. patient-to-patient or by the staff) and the role of GBV-C/HGV in liver disease among HD patients.

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10. Kallinowski B, Ahmadi R, Seipp S, Bommer J, Stremmel W. Clinical impact of GB-C virus in hemodialysis patients. HD patients contracted the virus, a very high rate of contacts with contaminated devices or persons such as the staff, seems attractive as it has been suggested recently by our group and others for hepatitis E virus infection [6,13]. The possibility of community-acquired or patient-to-patient transmission of GBV-C/HGV in HD patients has already been suggested [10]. We did not observe any significant difference of transaminases values between GBV-C/HGV-positive and -negative HD patients. These findings are similar with that reported by others [7–9] particularly when the HD patients were HCV negative. It remains unclear, therefore, whether GBV-C/HGV may be a causative agent for liver disease in HD patients. Case-control studies are required to confirm whether or not GBV-C/HGV is an aetiologic agent of viral hepatitis or any other disease. Anti-E2 and GBV-C/HGV-RNA were found to be mutually exclusive confirming the notion that anti-E2 has to be considered as a marker of past infection [7,9,14]. A surprisingly high frequency of GBV-C/HGV-RNA detection was also recorded very recently in groups other than HD patients without known risk factors for blood-borne viral infections, indicating that chronic GBV-C/HGV infection might also be transmitted by social or sexual contacts or other routes yet to be defined [15]. Therefore, in the HD setting GBV-C/HGV infection may be an indirect marker for potentially intraunit transmission of viral diseases since new infections are detected in the absence of blood transfusions. More studies, however, are needed in order to assess the transmission routes other than transfusions (e.g. patient-to-patient or by the staff) and the role of GBV-C/HGV in liver disease among HD patients.

HCV infection in haemodialysis and CAPD patients

Sir,

Hepatitis C virus (HCV) is a major cause of hepatitis among chronic dialysis patients. The prevalence of HCV infection varies according to countries and centres. In 1995, data collected by EDTA questionnaires from 30 different dialysis centres showed that the prevalence of HCV antibodies (HCV-Ab) using second generation ELISA, was 49.9% in Turkey [1]. Blood or blood products appear to be the primary risk factor for HCV infection, but a growing number of studies also indicate nosocomial transmission [2]. We evaluated the status of HCV infection and its relation with risk factors, in 59 haemodialysis (HD) and 29 continuous ambulatory peritoneal dialysis (CAPD) patients attending a university hospital dialysis unit in Izmir, Turkey. None of the patients was an intravenous drug abuser or infected with HIV. Blood samples of all patients were tested for HCV-RNA (Amplicor-HCV, Roche) and for HCV-Ab by ELA.
Influence of peritoneal loss of GHBP, IGF-I and IGFBP-3 on serum levels in children with ESRD

Sir,

Growth retardation is an important consequence of end stage renal disease (ESRD) in childhood. There have been several reports on the role of growth hormone (GH) and GH-related growth factors such as insulin-like growth factor (IGF-I) in ESRD [1–3]. GH binds to the GH binding protein (GHBP), a 60 kDa protein, and forms a 85 kDa complex in serum. About 50% of the GH in serum is present in this form [4]. The 60 kDa GHB is identical to the extracellular domain of the GH receptor that mediates normal GH action. IGF-I is bound to specific binding proteins in the circulation. In human serum, IGFBP-3, a 150 kDa glycoprotein complex, was found to be the predominant protein, binding >95% of IGF-I. In uremia, there is an excess of IGFBP-3, with molecular fragments of 12–150 kDa, caused by reduced renal clearance. IGFBP-3 has been shown to strongly inhibit IGF-I action [5]. In peritoneal dialysis (PD), substantial amounts of proteins like albumin, immunoglobulins and complement factors are lost with the dialysate. We investigated the loss of GHBP, IGF-I and IGFBP-3 in order to assess whether these losses contribute to the growth retardation in children on peritoneal dialysis.

Serum and peritoneal fluid concentrations of GHBP, IGF-I and IGFBP-3 were studied in eight children on PD. The mean age was 10.9 ± 3.5 (SD) years. Puberty was defined as a testicular volume of >4 ml in boys and in girls breast development more than stage 2 according to Tanner [6]. The creatinine clearance was less than 5 ml/min/1.73 m². Mean duration of dialysis was 21 months. Four patients were treated with GH (3 IU/m²/day) for at least 6 months.

Samples from 24-h dialysate collection were analysed for GHBP, IGF-I and IGFBP-3. Dialysate losses of IGF-I and IGFBP-3 per day were calculated using the concentration of protein present in the dialysate (concentration x volume/day).

GHBP levels were measured by separation of the GHBP-bound and free fraction using fast protein liquid chromatography. Levels of GHBP below 10 nmol/l were not detectable. IGF-I was determined by radioimmunoassay after acidification and C18 extraction of plasma or dialysate samples. The concentrations of IGFBP-3 in plasma and dialysate were determined by a commercial direct radioimmunoassay (Nichols Institute, The Netherlands). Methods of determination were the same in dialysate and serum.

Data are expressed as mean ± SD, but when the data were not normally distributed, median values are given. Differences between groups were analysed using the Mann-Whitney U test. A P value <0.05 was taken to indicate statistical significance.

Mean serum GHBP was low (271 ± 131.5 nmol/l). GHBP was not detectable in peritoneal fluid. Mean serum levels of IGF-I were normal to elevated (38.9 ± 24.6 nmol/l). Mean serum levels of IGFBP-3 were also elevated (6.1 ± 1.34 µg/ml). Serum GHBP, IGF-I and IGFBP-3 were significantly higher in the two pubertal children as normal ranges are age- and sex-dependent. The median daily peri-