

Table 1.

Subjects	HGV RNA ⁺	Anti HGV/E2 ⁺
Controls	7/249 (2.8%)	177/506 (35%)
HCV+ B-NHL (n=33)	1/33 (3%)	26/33 (78.8%)

HGV infection by HGV RNA assays (RT-PCR) and anti-HGV antibodies directed against the E2 structural protein (immunoenzymatic method), a reliable serologic marker of past HGV infection followed by viral clearance.⁸ Serum specimens were taken at the onset of the disease and stored at -20°C; we found only one patient with HGV positivity by RT-PCR (3%). Twenty-six of 33 patients were positive for anti HGV/E2 antibodies (78.8%) suggesting past infection.

This prevalence of HGV RNA was similar to that found in a population of healthy subjects matched for age and sex, tested as controls (Chi-square test: NS). In contrast, the prevalence of HGV antibodies was significantly higher in the NHL population than in the controls (Chi square test: $p < 0.0001$: see Table 1).

Even if we compare our findings with the prevalence of anti HGV/E2 reported in healthy subjects, drug abusers and blood donors by Tacke *et al.*,⁹ the detection of antibodies against HGV/E2 protein is much higher in our HCV positive NHL patients. It must be stressed that the immunoenzymatic method we used for anti HGV/E2 antibodies was tested as highly specific: therefore, cross reaction with anti-HCV-E2 antibodies is unlikely.

If confirmed, our preliminary data seem to suggest a higher incidence of past HGV infection in our group of HCV positive patients with B cell NHL. Whether or not this has any role (together with HCV) in the etiology or evolution of the neoplastic disease needs to be elucidated.

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Serum transferrin receptor in polycythemia

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We measured serum transferrin receptor (sTfR) levels in 22 patients with polycythemia vera and in 26 cases of secondary polycythemia. In our study, raised sTfR levels in both polycythemia groups were related to iron deficiency.

In normal adults a strong correlation has been shown to exist between serum transferrin receptor (sTfR) and standard ferrokinetic measurements of erythropoiesis¹ with the result that the level of erythropoietic activity is the most important determinant of sTfR. Increased expression of transferrin receptor (TfR) has also been documented on the surface of malignant tumor cells such as erythroleukemic cells.² Whether the shedding of these receptors can contribute to sTfR levels is still unclear. We measured sTfR in polycythemia patients to investigate its potential clinical usefulness. The sTfR was measured using a commercial immunoassay (ELISA) (Quantikine™, transferrin receptor EIA kit, R&D Systems, Minneapolis, USA) in 26 patients with secondary polycythemia (SP), 22 patients with polycythemia vera (PV) and 63 normal controls (REF group).

The sTfR levels (mean±SD) were 2.6±0.5 mg/L in the REF group, 5.9±3.9 mg/L in PV group and 4.7±2.9 mg/L in the SP group; sTfR was higher in both polycythemia groups than in the REF group (ANOVA, $p < 0.0001$). After excluding patients with iron deficiency, however, the sTfR levels in both polycythemia groups were 2.8±0.9 mg/L in the SP group (n=15), and 2.9±0.8 mg/L in the PV group (n=8). Moreover, there was no difference with respect to

sTfR levels in the REF group (ANOVA, $p=0.96$).

Raised sTfR levels have been reported in cases with polycythemia^{3,4} although it is unclear whether this increase is related to red cell mass, disease activity or iron status. Iron deficiency is closely associated with high sTfR values and sTfR levels progressively increase in parallel with the different iron deficiency stages, from the earliest stages with only low ferritin values to fully expressed iron deficiency anemia.⁵ In our study, the difference in sTfR levels between polycythemia and REF groups was probably due to iron status. Thus, at variance with serum erythropoietin,⁶ the possible role of sTfR in evaluating erythroblastic mass in polycythemia remains unresolved.

Key words

Polycythemia, serum transferrin receptor.

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Elective splenectomy in relapsing thrombotic thrombocytopenic purpura

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Between 20 and 40% of surviving patients with thrombotic thrombocytopenic purpura (TTP) have relapses.

Plasma exchange therapy is usually effective in treating relapses, but this treatment does not prevent TTP recurrence. The role of splenectomy in relapsing TTP is still controversial. We describe a patient with multiple relapses of TTP who was successfully treated with elective splenectomy.

Thrombotic thrombocytopenic purpura (TTP) is a rare, life-threatening disorder of unknown pathophysiology. Without treatment, TTP is a rapidly progressive and fatal disease, with 90% of patients surviving less than 3 months. Treatment with plasma exchange, often used in combination with corticosteroids, antiplatelet agents, splenectomy and vincristine has reduced mortality to 20%.¹⁻³ However, between 20 and 40% of surviving patients have relapses. Relapses can occur as early as a few weeks after recovery, but also after an interval of many years.^{4,5} Plasma exchange therapy is usually effective in treating relapses, but this treatment exposes the patient to blood products from numerous donors and does not reduce the relapse rate. Various interventions, including antiplatelet agents, corticosteroids and splenectomy have been used to prevent relapses.² Some studies have suggested that splenectomy has a role in the management of relapsing TTP, since it seems to reduce the frequency of relapse.⁶⁻⁸ We describe a patient with multiple relapses of TTP who was successfully treated with elective splenectomy during remission.

A 27-year-old female was diagnosed as having TTP in May 1992. Clinical manifestations in the initial episode of TTP were fatigue, headache, hematuria, petechiae and hematomas. At admission, biological findings were as follows: hemoglobin 8.2 g/dL, platelet count $15 \times 10^9/L$, LDH 897 U/L, bilirubin 1.8 mg/dL and serum creatinine 1 g/dL. The initial episode and two early recurrences through 1992 were successfully treated with fresh plasma transfusions, plasma exchange, corticosteroids and vincristine (Figure 1). The patient remained in remission until 1994. Between March 1994 and February 1996, this patient had five relapses (incidence of 2.5 relapses/year). The disease-free interval varied from 3 weeks to 9 months (Figure 1). Presenting signs and symptoms during relapses were similar but less severe than those observed in the first episode. The patient repeatedly responded to therapy with plasma infusions and prednisone. Splenectomy was performed 18 days after the last relapse when the platelet counts and LDH levels had returned to normal values with the treatment schedule mentioned before (Figure 1). One week before splenectomy pneumococcal vaccine was given. There were no perioperative complications. The spleen was not enlarged (weight 110 g). The histologic study showed a moderate hyperplasia of follicles in the white pulp. Microthrombi were absent. In the 2 years of follow-up since the splenectomy, the patient had no further relapses.

The pathophysiology of TTP is still poorly under-