Transient pure red blood cell aplasia has been observed following viral infections, treatment with several drugs, acute renal failure, kwashiorkor, deficit of vitamin B12, riboflavin and folate, and associated with immunological disorders and carcinoma. Only in two reports it has been described in association with viral hepatitis. We are reporting a new case of this association.

A 26-year-old male patient was admitted to our hospital in March 1979 due to an acute hepatocellular insufficiency. A diagnosis of acute HBsAg-negative hepatitis was made at that time. On March 26, 1979, the blood tests showed: hemoglobin 6.2 g/dl; hematocrit 18%; reticulocytes 0.2%; WBC 6.1×10^3/µl; platelets 240×10^3/µl; direct and indirect Coombs tests were negative. Bone marrow aspirate revealed a complete depletion of erythroblastic cells with normal granulocytic and megakaryocytic lines. The clinical picture was interpreted as a severe acute hepatitis, possibly of viral origin with pure red blood cell aplasia. Several transfusions of concentrated red cells were performed. On April 26, 1979, the patient presented an obvious improvement in peripheral blood findings: hemoglobin 13 g/dl; hematocrit 40%; and reticulocytes 9%. A new bone marrow aspirate showed a recovery of the erythroblastic cell population. A liver biopsy revealed a histologic picture compatible with an acute hepatitis. The patient was discharged on May 6, 1979. The patient has been feeling well until the present time, and all biochemical parameters have been normalized. A new liver biopsy, on October 15, 1979, showed a persistent chronic hepatitis.

After the initial description, by Sears et al. [1], of an association of acute viral hepatitis with a pure red cell aplasia in 2 brothers, an additional case has been reported by Wilson et al. [2]. A positive HBsAg was found in only 1 of these 3 patients. In several aspects, our case is similar to those previously reported. As the other 3 patients, ours had severe acute hepatitis, and several weeks later developed a sudden anemia, without signs of bleeding or hemolysis. The bone marrow aspirate showed a complete depletion of erythroblastic cells. Sears et al. described the first 2 cases in 2 brothers, both of whom developed, within a 4-year period, hepatitis associated with selective erythroblastopenia, suggesting a possible genetic link. A brother of our
patient had hepatitis 8 years ago, but it was not studied serologically. It seems that he never developed a simultaneous anemia. In 2 of the 3 reported cases the patient developed chronic hepatitis. Our patient also has persistent chronic hepatitis. The immunological studies were completely negative in 3 of the 4 cases studied, including the one described here. Only in 1 case a positive LE cell phenomenon and the presence of 142
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rheumatoid factor in serum, without clinical signs of rheumatoid arthritis or systemic lupus erythematosus, was observed [1]. Antibodies against erythroblasts could not be detected in the one case investigated.

References
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Neoplasms Associated with Hairy Cell Leukemia
To the Editor,
Montserrat et al. [1982] have described recently in this journal a neoplasm complicating a case of hairy cell leukemia (HCL). While studying a series [unpublished] of 35 cases of HCL, we observed similar associations in 4 cases: squamous cell carcinoma of the skin (1 case), piriform sinus carcinoma (1 case), and carcinoma of the lung (2 cases). Patients were respectively 76, 71, 69, and 65 years old. No prior chemotherapy was given before the emergence of carcinomas. However, in our opinion, further reports in larger series are needed for suggesting a close relation between the processes.
Reference

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