Improvement of Anemia Associated with Constrictive Pericarditis in a Hemodialysed Patient

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Dear Sir,

We read with great interest Brunois's report on the relationship between acute hepatitis and erythropoiesis in chronically hemodialysed patients [Nephron 28: 152–153 (1981)]. We observed the same relationship. Furthermore, we found that incomprehensible erythropoiesis seen on rare occasions exists. We here report on a patient whose anemia recovered spontaneously during the end stage of uremic constrictive pericarditis. The important finding concerning this case is that reticulocytosis with erythropoiesis was not recognized in a period of acute hepatitis but during liver congestion. Observations similar to ours have not been found in the past literature. This erythropoiesis could impossibly be explained by means of the known mechanism, such as erythropoietin induced by viral hepatitis. As the pathogenesis of anemia in hemodialyzed patients remains uncertain, we would like to report this rare phenomenon which might give some suggestions on the mechanism of erythropoiesis in chronic renal failure.

Case Report

In March 1980, a 44-year-old man was admitted to our hospital because of fever. For 2 years, he had been receiving hemodialysis treatment (12 h a week using a coil dialyzer) against diabetic nephro-pathy. Physical examination disclosed a temperature of 39 °C and a blood pressure of 150/94 mm Hg. The other objective findings were unremarkable. Laboratory data on admission were as follows: WBC count 14,500/mm³, RBC count 208 × 10⁴/mm³, hemoglobin 6.2 g/dl, reticulocyte count 1.4%, ESR 150 mm/h, BUN 98 mg/dl, creatinine 12.8 mg/dl, SGOT 10 mU/ml, SGPT 14 mU/ml, LDH 160 mU/ml and FBS 138 mg/dl. Chest roentgenogram and ECG on admission showed no specific findings. Although we closely examined the cause of the patient's fever, no particular findings were detected. 2 months after administration of antibiotics the fever was under control. By June 1980, however, hepatomegaly (2–3 fb below right costal margin) and refractory hypotension began to appear and in August ECG findings supported constrictive pericarditis. From September until the patient's death, heart failure progressed rapidly and at the same time the reticulocyte count increased (0.8- > 15.0%) and anemia spontaneously improved (RBC count 234 × 10⁴→366 × 10⁴/mm³, hemoglobin: 7.0→10.8 g/dl). He was never subject to blood transfusion nor to administration of an anabolic steroid. Hepatitis B surface antigen was constantly negative and significant hypoxemia was not observed in his clinical course, except during his last 2 days of life. The schedule of hemodialysis treatment was not changed during this time. Liver function studies carried out each month were constantly within normal limits. At autopsy it was found that the heart was encased by a thick, leathery pericardium measuring 6–8 mm. The parietal and visceral layers were firm,

white and adherent to each other. There was no pericardial fluid. The heart, including adherent pericardium, weighed 500 g. Microscopic examination showed no tuberculous or other specific lesions in the pericardium. Chronic liver congestion was prominent. We diagnosed this case as uremic constrictive pericarditis presenting fever as initial manifestation.

Comment

Chronic inflammation worsens anemia in general and, accordingly chronic pericarditis itself is not an explanation for stimulation of erythropoiesis. Secondary polycythemia could not be considered at all because of no existence of long-term hypoxemia. It was difficult to attribute the reduced anemia to dehydration alone. In our case we could not observe 'liver enzyme abnormalities' as suggested by Brown et al. [1] or 'HB viral or toxic hepatic cytolysis' as reported by Simon et al. [2]. We cannot find a complete explanation of our case in these factors. Reticulocytosis from an unknown cause did not appear often in the past literature.

Reticulocytosis while suffering from uremic pericarditis was pointed out by Silverberg et al. [3], but it was not fully determined whether each patient had accompanying erythropoiesis and which causes were present. The report by Simon et al. [2] also included 2 patients whose hematologic improvement was not attributed to viral or toxic liver cytolysis. Judging from the change of our pa-

376

Letter to the Editor

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Hepatomegaly

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tient's clinical feature (fig. 1), it seemed to us that congestive liver, which might produce erythropoietin or hepatotrophic factors, could be a participating factor in the erythropoiesis of our case. Further reports are needed to confirm this assumption.

References

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Fig.1. Relationship between hematologic data and clinical signs resulting from constrictive pericarditis. Silverberg

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