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Letters to the editor

A patient with a preoperative hemoglobin concentration of 1.8 g·dl[1]: How was the life-threatening anemia tolerated without any intensive care?

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To the editor:

We report a case with a preoperative hemoglobin concentration of 1.8 $g \cdot dl^{-1}$.

A 61-year-old Japanese man, 162 cm tall and weighing 49 kg, became aware of hematuria in the spring, and had general fatigue with systemic edema in September 1997. With the exacerbation of these symptoms, the patient had mild chest pain and dyspnea and was admitted to the hospital while still ambulatory in October 1997.

The patient had had a partial gastrectomy for duodenal ulcer in 1994. He had smoked half a pack of cigarettes per day for several years and quit smoking with this operation as an incentive.

On admission, he showed paleness, tachypnea, tachycardia, and systemic edema. His laboratory tests revealed severe hypochromatic and microcytic anemia with a hemoglobin (Hgb) concentration of 1.8 $g \cdot dl^{-1}$ as determined by a sodium lauryl sulfate method (NE 1500, Toa Medical Electronics, Kobe, Japan), red blood cell count of $1.24 \times 10^6 \cdot mm^{-3}$, hematocrit of 7.3%, mean corpuscular Hgb of 14.5 pg, mean corpuscular Hgb concentration of 24.7%, mean corpuscular volume of 58.9 μm^3, serum iron of 49 $\mu g \cdot dl^{-1}$, unsaturated iron-binding capacity of 146 $\mu g \cdot dl^{-1}$, total iron-binding capacity of 195 $\mu g \cdot dl^{-1}$, and ferritin of 4.79 $ng \cdot dl^{-1}$, while serum vitamin B_{12} (1200 $pg \cdot ml^{-1}$) and folic acid (5.9 $ng \cdot ml^{-1}$) levels and the findings of a bone marrow examination were normal. An analysis of arterial blood in FiO_2 of 0.21 revealed pH, 7.48; Pao_2, 63 torr; $Paco_2$, 25 torr; and base excess, −2.2 mM under spontaneous respiration. The patient's chest radiograph showed marked cardiomegaly and pleural effusions, i.e., signs of heart failure (Fig. 1). His electrocardiogram showed sinus tachycardia (120 $beats \cdot min^{-1}$) with no myocardial ischemic changes, and the echocardiogram indicated normal cardiac function but with mild mitral regurgitation and left ventricular hypertrophy. Macroscopic hematuria was observed. The patient showed normotension, clear consciousness, normal concentrations of serum electrolytes, lactic acid, and total protein, and normal hepatic, renal, and respiratory function.

Continuous inhalation of oxygen (3 $l \cdot min^{-1}$) via a face mask was initiated 30 min after admission, and homologous transfusion of packed red blood cells (PRBC) was initiated 5 h after admission. After transfusion of 2 units of PRBC in 2 h, Hgb concentration increased to 3.1 $g \cdot dl^{-1}$, resulting in the prompt alleviation of tachycardia and tachypnea. Inhalation of oxygen was continued for 48 h, and S_po_2 was maintained at 98%–99%. A total of 8 units of PRBC given in the following 9 days, supplemented by intravenous furosemide (20 mg per day for 5 days), increased the Hgb level to 7.5 $g \cdot dl^{-1}$, accompanied by an appropriate urine output. Consequently, the patient recovered from the heart failure symptoms. He needed no more intensive care such as mechanical ventilation, invasive hemodynamic monitoring, or administration of catecholamines throughout the clinical course. A bladder tumor with a diameter of 4 cm was detected by cystoscopy.

In November 1997 patient underwent a transurethral biopsy of the bladder tumor under subarachnoidal block uneventfully, followed by total cystectomy involving an ileal conduit under general anesthesia in March 1998.

It has been generally accepted that a Hgb concentration less than 6 $g \cdot dl^{-1}$ almost always requires blood transfusion [1]. However, some clinical studies have demonstrated that normovolemic profound hemodilution to Hgb values as low as 4 $g \cdot dl^{-1}$ can be well tolerated under intensive care in patients who refuse transfusion therapy because of religious beliefs [2–5]. Thus, the accepted minimal Hgb levels appear to be becoming lower with the times. To our knowledge, the lowest intraoperative Hgb ever reported for a survivor is 1.1 $g \cdot dl^{-1}$, demonstrated by Zollinger et al. in a patient extremely hemodiluted due to massive blood loss [6]. Why have reported patients survived despite intraoperative critical Hgb levels of

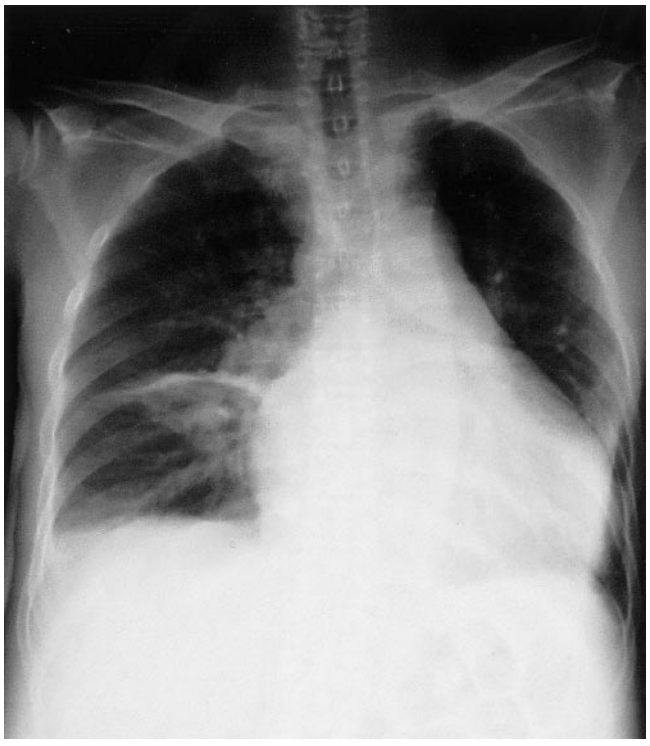

Fig. 1. Chest radiograph of the patient, a 61-year-old man, showing marked cardiomegaly and pleural effusions

less than 4 g·dl^{-1}? The reasons might be attributable to the fact that they were isovolemically hemodiluted under general anesthesia and placed under intensive care. In such cases, the higher FIO_2 (mostly 1.0), the support of cardiac output by the use of catecholamines, and the decrease in blood viscosity caused by isovolemic hemodilution may assure the oxygen supply. With respect to the oxygen demand, anesthetic agents and mechanical ventilation using muscle relaxants might suppress it, resulting in the prevention of anemic organ damage [7]. Additionally, the duration of threatening anemia was quite short.

In contrast to those cases, the present patient survived the threatening anemia with the nadir Hgb of 1.8 g·dl^{-1} in his daily life for a considerable duration. How his anemia was so well tolerated without any intensive care remains unknown. We suspect that his anemia had developed chronically by bleeding from the bladder carcinoma and the deficiency in ferritin caused by the previous gastrectomy. Empirically, we know that tolerance to low Hgb levels is stronger in patients with chronic rather than acute anemia. In chronically anemic patients on dialysis, it has been reported that an advantageous adaptation to anemia occurs by a decreased affinity of Hgb for oxygen in association with an increase in 2,3-diphosphoglycerate concentration [8]. In addition, the functions of the organs of the present patient had remained comparatively normal. These factors might be contributory to our patient's anemic tolerance. Further clinical and experimental studies on the acceptable minimal Hgb concentration in perioperative patients are of great importance.

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