—Case Reports—

Concomitant Severe Normocytic and Normochromic Anemia in Poststreptococcal Acute Glomerulonephritis

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Abstract

Although anemia frequently occurs in poststreptococcal acute glomerulonephritis (PSAGN), severe anemia is rare. We report severe normocytic, normochromic anemia (hematocrit, 19.8%) in PSAGN in a 6-year-old girl with edema, macrohematuria, and proteinuria for 1 month. The potential causes of severe anemia found in this case were: 1) longer duration of massive hematuria from onset of macrohemauria to treatment, 2) a level of erythropoietin much lower than that in cases of iron deficiency anemia, and 3) hemodilution. We speculate that these factors combined to cause an unusual case of severe anemia in PSAGN.
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Key words: anemia, poststreptococcal acute glomerulonephritis, erythropoietin

Introduction

Poststreptococcal acute glomerulonephritis (PSAGN) is typically a benign disease. Although anemia frequently occurs in PSAGN, probably due to hemodilution, severe anemia is rare[1]. Here, we report a case of severe normocytic and normochromic anemia in PSAGN.

Case Report

A 6-year-old girl was admitted to our hospital with edema, macrohematuria, and proteinuria. Macrohematuria had been confirmed 3 weeks before admission and continued without specific treatment. Body weight was 25.5 kg (23.4 kg, 2 months earlier). Laboratory findings on admission were as follows: leukocytes, 7,480/μL (segmented leukocytes, 59.0%; lymphocytes, 34.0%; monocytes, 4.0%; eosinophils, 2.0%); red blood cell (RBC) count, 227 × 10^4/μL; hemoglobin, 6.6 g/dL; hematocrit, 19.8%; mean corpuscular volume (MCV), 87.2 fL (normal, 80–101 fL); mean corpuscular hemoglobin (MCH), 29.1 pg (normal, 26.4–34.3 pg); mean corpuscular hemoglobin (MCHC), 33.3% (normal, 31.3%–36.1%); reticulocytes, 1.1%; and platelets, 17.1 × 10^4/μL. A hemogram showed no abnormalities, such as fragmentation or poikilocytosis. Blood urea nitrogen (BUN) was 63.7 mg/dL, and serum creatinine was 1.02 mg/dL, while serum total protein was 5.9 g/dL, albumin was 3.4 g/dL, complement 3 was 9 mg/dL (normal, 80–140 mg/dL), complement 4 was 15.5 mg/dL (normal 11.0–34.0), anti-streptolysin O (ASLO) was 640 X (normal, <160), and anti-streptokinase (ASK) was 20480 X (normal, <128). Examinations for other

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Table 1 Clinical and laboratory findings

Day after admission	1	2	3	5	8	15	27
Water balance (mL)	+ 170	+ 260	+ 300	− 10	− 250	+ 100	+ 130
Blood pressure (mmHg)	120/70	100/60	100/62	110/62	110/60	98/60	90/50
Body weight (kg)	25.9	25.5	26.0	24.0	23.5	22.5	21.4
Hemoglobin (g/dL)	6.6	7.4	7.9	8.0	8.3	9.9	10.6
Hematocrit (%)	19.8	22.5	24.2	23.8	25.2	29.8	31.5
Reticulocytes		1.1% (2,810/μL)		1.3% (3,560/μL)			0.4% (1,430/μL)
Blood Urine Nitrogen (mg/dL)	63.9	55.5	49.2	25.8	6.1	10.6	10.5
Creatinine (mg/dL)	1.02	0.90	0.88	0.74	0.61	0.57	0.49
Urinalysis (RBC/HPF)	numerous	50−99	many	many	numerous	numerous	many

Water balance was calculated as follows: volume of water intake plus intravenous drip infusion minus volume of urine
Many RBC showed 100−1,000/HPF on urinalysis
Numerous RBC showed 1,000>/HPF on urinalysis

viruses were not performed because of the absence of symptoms suggesting parvovirus B19 or Epstein-Barr virus infection and because of the presence of low complement values that are characteristic of PSAGN. Urinalysis revealed protein and occult blood of 3+ (dip and read method). Urinary sedimentation tests revealed numerous RBCs/high-power field (HPF), 30 to 40 WBCs/HPF with 10 to 19 hyaline casts/HPF, and 50 to 99 granular casts/HPF. Coagulation tests showed a prothrombin time of 85.7% (normal, 80%−100%), activated partial thromboplastin time (APTT) of 32.2 seconds (normal, 26.0−38.0 seconds), hepaplastin test of 91.8% (normal, 70%−130%), fibrinogen of 263 mg/dL (normal, 170−410 mg/dL), factor XIII of 77% (normal, >70%), and fibrin degradation products (FDPs) of 12 μg/mL (normal, <10 μg/mL). We diagnosed PSAGN with severe normocytic and normochromic anemia.

While treating PSAGN, we attempted to determine the cause of anemia. Examination for anemia showed: serum iron, 75 μg/dL; total iron binding capacity, 218 μg/dL; serum erythropoietin, 13.4 mIU/mL (normal, <29.0 mIU/mL); total bilirubin, 0.2 mg/dL; haptoglobin, 78 mg/dL (normal, 19−170 mg/dL); transferring, 135 mg/dL (normal, 190−320 mg/dL); and stool occult blood, negative. One week after admission, hemoglobin was 8.3 g/dL, hemotocrit was 25.2%, MCV was 87.5 fL, MCH was 28.8 pg, MCHC was 32.9%, BUN was 6.1 mg/dL, creatinine was 0.61 mg/dL, and body weight was 23.5 kg (**Table 1**). The anemia gradually resolved and was accompanied by improvement of hematuria, normalization of transferrin ASLO, and ASK values and nephritis without specific treatment for anemia.

Discussion

Severe hematological disorders are rare in PSAGN; only autoimmune hemolytic anemia has been reported in PSAGN[2]. In the present case, hemolytic anemia[3] was unlikely because of normal haptoglobin and reticulocyte levels. Hemophagocytic syndrome was also unlikely because of normal levels of ferritin, β_2-microglobulin, and lactate dehydrogenase. The findings that were possible causes of severe anemia in the present case were: 1)

duration of massive hematuria from the onset of macrohemauria to treatment (21 days) longer than the mean duration in 10 other patients with PSAGN (average 4.6 ± 2.2 days); 2) levels of erythropoietin (13.4 mIU/mL) much lower than those in patients with iron-deficiency anemia (314 ± 93 mIU/mL) or aplastic anemia with low hemoglobin levels (305 ± 68 mIU/mL)[4]; and 3) 17% hemodilution, which is usually considered a cause of anemia in PSAGN[1]. The low level of transferrin might be due in part to hemodilution. The mild anemia of PSAGN might be caused by hemodilution alone, but the moderate anemia of PSAGN is caused by hemodilution with bone marrow depression, as indicated by the low reticulocyte index[1]. In severe anemia with renal disease, normal, rather than increased, erythropoietin levels have also been reported in cases of acute crescentric glomerulonephritis with severe anemia[5], as they were in the present case. Also it has been reported that massive hematuria could cause reversible acute renal failure as a consequence of tubular cell damage[6] and might decrease production of erythropoietin[5]. We considered that these combined factors caused an unusual case of severe anemia in PSAGN. This case demonstrates that we must be aware of hematologic complications in PSAGN.

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