[CASE REPORT]

A Case of Severe Anemia Caused by Gastric Antral Vascular Ectasia and Autoimmune Gastritis

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Abstract:

An 80-year-old man presented to our hospital with general fatigue on exertion that had gradually worsened over 6 months. His blood test revealed severe anemia, and gastroscopy revealed findings consistent with gastric antral vascular ectasia (GAVE) and autoimmune gastritis. We diagnosed the patient with severe anemia caused by GAVE and autoimmune gastritis. The present case suggested that GAVE is triggered by autoimmune gastritis, and the mechanism is likely related to hypergastrinemia. The reporting of this rare case may help elucidate the cause of GAVE, which is currently unknown.

Key words: iron deficiency, vitamin B₁₂ deficiency, gastric antral vascular ectasia, autoimmune gastritis, hypergastrinemia, severe anemia

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Introduction

Gastric antral vascular ectasia (GAVE) is a condition in which vasodilation is observed mainly in the gastric antrum. GAVE is a common cause of gastrointestinal bleeding. GAVE is often associated with chronic liver disease or chronic kidney disease and is detected by the chief complaint of anemia (1-3). However, the pathogenesis of GAVE is not clearly understood.

Autoimmune gastritis is a type of atrophic gastritis characterized by gastric corporal atrophy via endoscopy and by the anti-gastric parietal cell antibody. This disease can result in achlorhydria, hypergastrinemia, iron deficiency, vitamin B₁₂ deficiency, and neuroendocrine tumors (4, 5). Since both iron and vitamins play an important role in red blood cell formation, autoimmune gastritis is often associated with anemia via iron deficiency and vitamin B₁₂ deficiency. The ionization of iron by gastric acid is important for efficient iron absorption. The efficiency of iron absorption is reduced by achlorhydria, which is caused by severe and chronic mucosal atrophy in the gastric fundic gland area (6). Massive

atrophy of the corporal mucosa markedly reduces the secretion of the intrinsic factor, which is necessary for vitamin B₁₂ absorption; the activity of the intrinsic factors is also reduced with the appearance of anti-intrinsic factor antibodies (7).

We herein report a case of severe anemia caused by GAVE and autoimmune gastritis in a patient with no notable medical history. Since this is a rare case, our reporting may help elucidate the cause of GAVE.

Case Report

An 80-year-old Japanese man with no notable medical history, including *Helicobacter pylori* eradication or the use of medications, such as proton pump inhibitors (PPIs), presented to our hospital with general fatigue on exertion that gradually worsened over six months. His initial blood test findings revealed severe microcytic hypochromic anemia: red blood cell count $1.64 \times 10^6/\mu\text{L}$, hemoglobin 3.4 g/dL, and hematocrit 13.1%. There were no abnormalities in his liver or renal functions. Additional blood tests to investigate the cause of the anemia revealed iron deficiency and a reduction

Table. Blood Test Results.

Items (unit)	Measured value	Reference value range from the Osaka Medical and Pharmaceutical University Hospital
White blood cell counts (/μL)	4,620	3,300-8,600
Red blood cell counts (×10 ⁴ /μL)	164*	435-555
Hemoglobin (g/dL)	3.4*	13.7-16.8
Hematocrit (%)	13.1*	40.7-50.1
Mean corpuscular value (fL)	79.9*	83.6-98.2
Mean corpuscular hemoglobin (pg)	20.7*	27.5-33.2
Platelet count (×10 ³ /μL)	196	158-348
Aspartate aminotransferase (U/L)	30	13-30
Alanine aminotransferase (U/L)	8*	10-42
Lactate dehydrogenase (U/L)	262*	124-222
Alkaline phosphatase (U/L)	72	38-113
γ-Glutamyl transpeptidase (U/L)	22	13-64
Blood urea nitrogen (mg/dL)	12	8/20
Creatinine (mg/dL)	0.80	0.65-1.07
Iron (μg/dL)	14*	40-188
Ferritin (ng/dL)	2.8*	39.9-465.0
Vitamin B ₁₂ (pg/mL)	≤50*†	180-914
Folic acid (ng/mL)	15.5	≥4.0
Anti- <i>Helicobacter pylori</i> antibody (U/mL)	<3†	<10
Anti-gastric parietal cell antibody	×40*	<×10
Anti-intrinsic factor antibody	Positive*	Negative
Gastrin (pg/mL)	2,700*	≤200

*: outside the reference range; †: outside the detection range.

in vitamin B₁₂ levels below the measurement sensitivity (Table). Gastrosocopy revealed massive atrophy in the corporal area, which is a typical finding of autoimmune gastritis (Fig. 1). In addition, longitudinal capillary dilatation radiating to the vestibular area was observed; these findings are typical of GAVE, watermelon stomach type (Fig. 1). There were no findings suggestive of gastric cancer or neuroendocrine tumors (Fig. 1). Colonoscopy showed no abnormalities. Subsequent blood tests revealed hypergastrinemia, positive anti-gastric parietal cell antibodies, and positive anti-intrinsic factor antibodies (Table). The impaired absorption of vitamin B₁₂ and iron was considered to be caused by autoimmune gastritis. In addition, a further cause for iron deficiency was chronic persistent bleeding from GAVE. Both diagnoses were believed to be the cause of the severe anemia in the present case. The patient's anemia was treated with red blood cell transfusion and intravenous and oral vitamin B₁₂. To prevent gastrointestinal bleeding from GAVE, treatment with endoscopic argon plasma coagulation was performed (Fig. 2).

One month after endoscopic argon plasma coagulation, his anemia had not worsened. However, gastroscopy revealed that GAVE partially remained. We will re-treat the remaining GAVE with endoscopic argon plasma coagulation if bleeding occurs.

Discussion

In this rare case, severe anemia in our patient was caused by both GAVE and autoimmune gastritis. This case may help elucidate the cause of GAVE, as this presentation of this disease has not been previously reported in the medical literature.

As previously mentioned, the causes of GAVE remain unknown. However, several factors have been speculated. Gostout et al. reported that chronic gastritis occurred in GAVE cases, with hypergastrinemia associated in many instances (8). In a recent Japanese case report, GAVE in a cirrhotic patient with hypergastrinemia was induced by long-term PPI administration, but the patient discontinued follow-up after PPI treatment was completed (9). Furthermore, it has been reported that estrogen or prostaglandin E₂ is involved as other endocrinological factors. Liver cirrhosis and chronic kidney disease are considered to cause metabolic disturbances in these hormones, resulting in increased blood levels (10, 11). However, in contrast to these previous cases and studies, our patient did not have liver cirrhosis or chronic kidney disease, had not taken a PPI, and did not have autoimmune thyroid disease or autoimmune polyendocrine syndrome. Therefore, we hypothesized that hypergastrinemia caused by autoimmune gastritis might have triggered GAVE in our patient.

Autoimmune gastritis is not a rare disease; its prevalence

Figure 1. Gastroscopy findings. Gastroscopy revealed massive atrophy in the corporal area, and the findings were typical of autoimmune gastritis. There were no findings suggesting gastric cancer or a neuroendocrine tumor (a, b). Longitudinal capillary dilatation radiating to the vestibular area was observed, and the findings were typical of gastric antral vascular ectasia, watermelon stomach type (c).

Figure 2. To prevent gastrointestinal bleeding from gastric antral vascular ectasia, treatment with endoscopic argon plasma coagulation was performed.

in asymptomatic individuals undergoing medical checkups has been reported to be 0.49% in Japan (12). Autoimmune gastritis often causes iron deficiency and an impaired absorption of vitamin B₁₂, resulting in anemia (4, 5). Simple iron deficiency anemia results in a microcytic hypochromic anemia pattern, whereas simple vitamin B₁₂ deficiency ane-

mia results in a macrocytic hyperchromic anemia pattern. In the present case, although the patient was deficient in both iron and vitamin B₁₂, the microcytic hypochromic anemia pattern suggests that iron deficiency anemia was predominant. Vitamin B₁₂ deficiency should not be overlooked because of the assumption that microcytic hypochromic ane-

mia is simply due to iron deficiency, as chronic vitamin B₁₂ deficiency can cause irreversible peripheral neuropathy (13).

In conclusion, the present case suggested that GAVE is triggered by autoimmune gastritis, and the mechanism is likely related to hypergastrinemia. Therefore, it is necessary to study more similar cases in the future.

The authors state that they have no Conflict of Interest (COI).

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