

Available online at [www.sciencedirect.com](http://www.sciencedirect.com)

ScienceDirect

journal homepage: [www.e-jds.com](http://www.e-jds.com)

## Correspondence

# Pernicious anemia – Diagnosis, treatment, and clinical outcome of a case



## KEYWORDS

Pernicious anemia;  
Diagnosis;  
Treatment;  
Clinical outcome

Pernicious anemia (PA) is a macrocytic normochromic anemia.<sup>1–3</sup> Here, we reported a case of PA in a 64-year-old female patient who was treated with intramuscular injection of vitamin B12 and oral administration of folic acid and iron tablets and showed a good clinical outcome with the abnormal blood data returning to the normal values in two months.

This 64-year-old female patient complained of fatigue, shortness of breath, rapid heart rate, loss of appetite, and unsteadiness when walking for more than 2 months. She came to our dental clinic for evaluation and treatment. Blood examination showed decreased number of red blood cells (1.20 M/ $\mu$ L) and platelets (102 k/ $\mu$ L), reduced blood values of hemoglobin (5.1 g/dL), hematocrit (15.3%), and vitamin B12 (<50 pg/mL), and increased mean corpuscular volume (MCV, 127.5 fL), red blood cell volume distribution width-coefficient of variation (RDW-CV, 22.5%), and serum homocysteine level (79.89  $\mu$ M). The patients also had serum gastric parietal cell antibody (GPCA) positivity with the autoantibody titer of 1:80 and diabetes mellitus with the HbA1c of 7.4% (Table 1). The clinical diagnosis was PA according to the abnormal blood examination data. After discussing with the patient and obtaining the signed informed consent, the patient was treated with intramuscular injection of hydroxocobalamin (2 mg of hydroxocobalamin in 2 cc of distilled water, once per two days for two weeks, once per week for 6 weeks, and once per month thereafter), and oral administration of folic acid tablet (one tablet per day, each tablet contained 5 mg of folic acid) and iron tablet (one tablet per day, each tablet

contained 100 mg of Fe(OH)<sub>3</sub> polymaltose complex). The patient's PA improved quickly and the abnormal blood data returned to normal after 2 months of vitamin B12, folic acid, and iron supplement treatment (Table 1). In addition, the patient's diabetes mellitus was also well treated by the endocrinologist with the abnormal HbA1c level (7.4%) returning to the normal value (5.5%) after 2-month treatment.

PA is a macrocytic anemia caused by the lack of vitamin B12. Etiologies of vitamin B12 deficiency include inadequate intake, food-bound vitamin B12 malabsorption, lack of intrinsic factor or parietal cells, ileal malabsorption, biologic competition, and deficiency of transcobalamin.<sup>1–3</sup> The intrinsic factor, which is produced by the parietal cells of the stomach lining, can avidly bind dietary vitamin B12. The vitamin B12-intrinsic factor complex is carried to the terminal ileum, where it is absorbed after binding to intrinsic factor receptors on the luminal membranes of ileal cells. In the PA patient, the presence of GPCA and/or intrinsic factor autoantibodies in the body can result in failure of intrinsic factor production or inactivation of intrinsic factor, and in turn lead to the vitamin B12 deficiency.<sup>1–3</sup> Because our patient had the GPCA that could be the major factor causing the malabsorption of vitamin B12, the vitamin B12 deficiency, and finally PA in our patient. Vitamin B12 and folic acid are necessary for DNA synthesis and both are hematopoietic factors that promote the production of blood cells, especially the red blood cells. Iron is the structure component of hemoglobin.<sup>4</sup> We suggest that although the main etiologic cause of PA is

<https://doi.org/10.1016/j.jds.2022.09.006>

1991-7902/© 2022 Association for Dental Sciences of the Republic of China. Publishing services by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

**Table 1** Blood examination data of the patient with pernicious anemia before and after 2-month treatment.

	Baseline	6 days later	11 days later	21 days later	25 days later	61 days later
Red blood cells (M/ $\mu$ L)	1.20	1.71	2.33	3.15	3.41	4.71
Hemoglobin (g/dL)	5.1	6.3	8.2	10.1	10.8	13.6
Hematocrit (%)	15.3	21.6	27.1	33.5	35.1	43.9
Mean corpuscular volume (MCV, fL)	127.5	126.3	116.3	106.3	102.9	93.2
Mean corpuscular hemoglobin (pg)	42.5	36.8	35.2	32.1	31.7	28.9
White blood cell (k/ $\mu$ L)	5.15	6.20	9.56	9.18	9.59	9.64
Platelet (k/ $\mu$ L)	102	221	686	527	480	452
RDW-CV (%)	22.5	23.0	20.3	17.7	16.5	13.6
Vitamin B12 (pg/mL)	<50	26,501	—	—	—	4488
Folate (ng/mL)	—	—	—	—	—	37.1
Homocysteine ( $\mu$ M)	79.89	—	—	—	5.37	6.10
Gastric parietal cell antibody (GPCA)	1:80 (+)	1:80 (+)	—	—	—	—
HbA1c (%)	7.4	5.7	—	—	—	5.5

RDW-CV: Red blood cell volume distribution width-coefficient of variation; HbA1c: Glycated hemoglobin.

vitamin B12 deficiency, in addition to giving vitamin B12 to the patient, simultaneous supplementation of folic acid and iron can result in a quick improvement of PA in a short period.<sup>5</sup>

### Declaration of competing interest

The authors have no conflicts of interest relevant to this article.

### References

1. Sun A, Wang YP, Lin HP, Jia JS, Chiang CP. Do all the patients with gastric parietal cell antibodies have pernicious anemia? *Oral Dis* 2013;19:381–6.
2. Sun A, Chang JYF, Wang YP, Cheng SJ, Chen HM, Chiang CP. Do all the patients with vitamin B12 deficiency have pernicious anemia? *J Oral Pathol Med* 2016;45:23–7.
3. Chang JYF, Wang YP, Wu YC, Cheng SJ, Chen HM, Sun A. Hematinic deficiencies and pernicious anemia in oral mucosal disease patients with macrocytosis. *J Formos Med Assoc* 2015; 114:736–41.
4. Wang YP, Chang JYF, Wu YC, Cheng SJ, Chen HM, Sun A. Oral manifestations and blood profile in patients with thalassemia trait. *J Formos Med Assoc* 2013;112:761–5.
5. Wu YH, Hwang MJ, Lee YP, Chiang CP. Atrophic glossitis in pernicious anemia patients can be treated to normal in two weeks by intramuscular injection of vitamin B12. *J Dent Sci* 2020;15:558–9.

Jia-Lin Chiang  
Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical  
Foundation, Hualien, Taiwan

Ming-Jane Lang\*  
Department of Dentistry, Hualien Tzu Chi Hospital,  
Buddhist Tzu Chi Medical Foundation, Hualien, Taiwan

Chun-Pin Chiang\*\*  
Department of Dentistry, Hualien Tzu Chi Hospital,  
Buddhist Tzu Chi Medical Foundation, Hualien, Taiwan  
Department of Dentistry, National Taiwan University  
Hospital, College of Medicine, National Taiwan University,  
Taipei, Taiwan  
Graduate Institute of Oral Biology, School of Dentistry,  
National Taiwan University, Taipei, Taiwan

\*Corresponding author. Department of Dentistry, Hualien  
Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, No.  
707, Section 3, Chung-Yang Road, Hualien 970, Taiwan.  
E-mail address: [dentist.artistl@gmail.com](mailto:dentist.artistl@gmail.com) (M.-J. Lang)

\*\*Corresponding author. Department of Dentistry, Hualien  
Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, No.  
707, Section 3, Chung-Yang Road, Hualien 970, Taiwan.  
E-mail address: [cpchiang@ntu.edu.tw](mailto:cpchiang@ntu.edu.tw) (C.-P. Chiang)

Received 7 September 2022  
Available online 20 September 2022