CASE REPORT

Severe Nutritional Deficiency Behind a Social Facade: A Case of High-Output Heart Failure and Severe Megaloblastic Anemia Caused by Self-Imposed Dietary Restrictions

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Abstract

Our clinical experience with this patient illustrates that severe nutritional deficiencies and thiamine-driven high-output heart failure (HOHF) can occur even in socially independent individuals without traditional risk factors. We introduce "selective malnutrition" as a critical phenomenon where life-threatening metabolic voids hide behind a facade of social stability. Our focus is a 54-year-old homemaker who presented in a state of sudden cardiovascular collapse, marked by a hemoglobin of 2.1 g/dL and mildly elevated blood lactate. Despite her outwardly functional role, her rigid "kodawari"—an uncompromising adherence to idiosyncratic dietary rules—resulted in catastrophic thiamine and vitamin B12 depletion. Following aggressive empirical vitamin rescue, we witnessed a dramatic clinical pivot where hemodynamic stabilization significantly outpaced the recovery of her anemia. This case underscores the need to scrutinize hidden, individual dietary behaviors in primary care, regardless of apparently stable social or household circumstances.

Keywords: *Wet beriberi, kodawari, self-imposed dietary restriction, megaloblastic anemia, high-output heart failure.*

INTRODUCTION

Clinicians occasionally find themselves managing high-output heart failure (HOHF)—a paradoxical state where, despite a surging cardiac output, the body's metabolic demands remain unmet due to a plummeting systemic vascular resistance. While we are trained to prioritize hyperthyroidism or arteriovenous fistulas, we must not let thiamine (vitamin B1) deficiency slip through the cracks of modern practice. It is a dangerous oversight, especially when the clinical presentation deviates from what we expect in classic textbooks [1-3].

The timeline of "wet beriberi" is notoriously unforgiving. It can spiral into a total circulatory collapse within hours, often catching even seasoned clinicians off guard. In Japan, this disease has a long history linked to polished rice. Yet, today, a dangerous bias persists. Many of us still reflexively pigeonhole beriberi as a tragedy of the marginalized—those battling alcoholism, deep poverty, or the isolation of "hikikomori" [4]. This case illustrates that catastrophic nutritional failure can hide in plain sight, serving as a critical reminder that severe vitamin deficiencies may occur even in individuals who appear perfectly stable and socially integrated.

We refer to this pattern as "selective malnutrition"—which we define as a state of severe, life-threatening deficiency in one or more essential micronutrients despite an overall adequate caloric intake and the complete preservation of outward social and occupational functioning. In our observation, even when patients remain socially independent and maintain their daily routines, their physiological reality may be one of severe imbalance. This crisis often stems from a diet highly restricted by deep-seated personal habits or an intense psychological focus on specific food choices ("kodawari"). We have encountered striking instances where patients meticulously prepared nutritious meals for their families while dangerously neglecting their own intake. Because their social functioning remains a convincing facade, these profound nutritional deficits often stay hidden until triggered by the sudden onset of anemia, heart failure, or other metabolic collapses.

Adding to the complexity, vitamin B12 deficiency often joins the fray, further fueling the high-output state by triggering severe megaloblastic anemia [5]. We believe the metabolic synergy between B1 and B12 depletion creates a precarious instability, pushing the patient toward an avoidable cardiovascular collapse [6]. In this report, we describe a 54-year-old homemaker whose survival was threatened by both profound macrocytic anemia and wet beriberi. Her case stands as a stark warning: we must look past a patient's perceived social standing and instead interrogate the hidden, idiosyncratic dietary choices of the individual.

CASE PRESENTATION

Our clinical involvement centered on a 54-year-old woman who presented as an emergency. She had a long-standing history of panic disorder, and for the last three months, before admission, she had experienced progressive generalized edema with exertional dyspnea; she was practically bedbound on admission

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Table 1: Laboratory findings on admission.

Category	Test Item	Result	Reference Range
Hematology	Hb (g/dL)	2.1	11.5-15.0
	MCV (fL)	152.4	83.0-99.0
	WBC (10^3/μL)	4.07	3.04-8.54
	Plt (10^3/μL)	29	150-361
Biochemistry	LD (U/L)	675	124-222
	T-Bil (mg/dL)	2.8	0.4-1.5
	Alb (g/dL)	2.4	4.1-5.1
Cardiac/Lactate	NT-proBNP (pg/mL)	1563	< 125
	Lactate (mmol/L)	2.5	0.5-1.6
Vitamins*	Vitamin B1 (ng/dL)	1.6	2.4-5.0
	Vitamin B12 (pg/mL)	152	180-914

*Results obtained on hospital day 10.
Abbreviations: Hb, hemoglobin; MCV, mean corpuscular volume; WBC, white blood cell; Plt, platelet; LD, lactate dehydrogenase; T-Bil, total bilirubin; Alb, albumin; NT-proBNP, N-terminal pro-brain natriuretic peptide.

and incapacitated due to fatigue. Her dietary history was particularly striking; idiosyncratic beliefs on food restriction drove her and, as a result—afraid of inducing anorexia with stress—had not consumed any meat for an extended period. She had no smoking or alcohol history and was not on any regular medications at this time of her decline.

The initial physical exam quickly and accurately illuminated how truly sick she was. We saw a patient with extreme palpebral conjunctival pallor and generalized, diffuse anasarca. Despite a BP of 130/87 mmHg and an HR of 76 beats/min, her respiratory status was obvious with a rate of 24 breaths/min and a need for 2 L/min of supplemental oxygen. Chest radiograph demonstrated cardiomegaly (cardiothoracic ratio 60%) and moderate pulmonary congestion; however, transthoracic echocardiogram showed a preserved LVEF (62%). There was no significant valvular or structural abnormality, and her high-output failure was therefore considered to be non-cardiac in origin.

The laboratory findings were alarming (**Table 1**). We were faced with a very low hemoglobin level of 2.1 g/dL and evidence of macrocytic anemia, with a markedly increased mean corpuscular volume of 152.4 fL. In addition, her blood counts were significantly reduced and included a white blood cell count of 4.07 ×10^3/μL and a platelet count of 29 ×10^3/μL. The clinical data suggested that her red blood cell production system was not functioning correctly, as demonstrated by an LD of 675 U/L and a bilirubin of 2.8 mg/dL. Another important laboratory finding was an increase in NT-proBNP to 1,563 pg/mL and blood lactate to 2.5 mmol/L. To rule out other common etiologies of high-output heart failure, we investigated thyroid function and evaluated the patient for arteriovenous fistulas. Her thyroid function test on admission showed a free thyroxine (FT4) level of 1.0 ng/dL (reference range: 0.90-1.70 ng/dL) and a thyroid-stimulating hormone (TSH) level of 5.050 μIU/mL (reference range: 0.50-5.00 μIU/mL). These values indicated that her thyroid function was essentially normal, ruling out both thyrotoxicosis and hypothyroidism as potential causes of her heart failure. Physical and imaging examinations also revealed no evidence of arteriovenous shunts. These lab results, in addition to the clinical findings, indicated that she had severe deficiencies of thiamine and vitamin B12 (laboratory values were 1.6 ng/dL of Thiamine and 152 pg/mL of Vitamin B12, respectively).

With all the evidence available to us pointing towards both high-output cardiac failure and hyperlactatemia, combined with her dietary restrictions that led to severe anemia, we felt comfortable making a diagnosis of two concurrent disease states (*i.e.*, wet beriberi and megaloblastic anemia). On Day 1, we began giving her an aggressive dose of intravenous (IV) thiamine (200 mg of Thiamine daily) along with an aggressive dose of parenteral vitamin B12 (500 mcg of B12 three times a week) with concurrent transfusion of 4 units of packed red blood cells (**Fig. 1**).

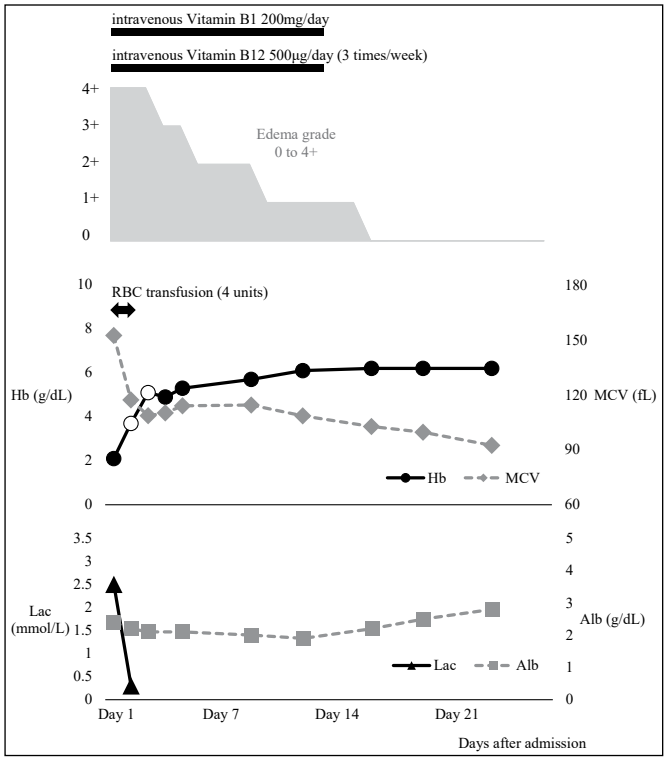


Fig. (1): Clinical course and response to treatment.

The hospital course is indexed by days after admission (Day 1).
Top panel: The duration of intravenous Thiamine (200 mg/day, daily) and vitamin B12 (500 μg/dose, 3 times/week) supplementation is indicated by horizontal bars (Day 1-14). The clinical progression of edema is presented using a semi-quantitative scale (0 to 4+).
Middle panel: Trends in hemoglobin (Hb) levels (solid line, primary axis) and mean corpuscular volume (MCV) (dashed line, secondary axis). The period of red blood cell (RBC) transfusion (4 units) is indicated by a double-headed arrow; open circles (Days 2 and 3) represent values immediately following transfusion.
Bottom panel: Changes in serum lactate (Lac) (solid line, primary axis) and albumin (Alb) (dashed line, secondary axis). Note the rapid clearance of lactate within 24 hours (Day 2) following the initiation of high-dose Thiamine.

Thiamine produced a fast reaction (**Fig. 1**). In a week, her symptoms of difficulty in breathing and swelling were almost entirely gone; the fluid that had been seen surrounding her lungs (which had previously been identified on a chest x-ray) was no longer visible on day 7, and she no longer required the use of supplementary oxygen on day 8. One important finding was the complete disappearance of swelling, while her blood albumin remained at 2.4 grams per deciliter and her anemia (6.2 g/dL hemoglobin) was only partially resolved. To clarify, her cardiovascular system began to stabilize only a day after the thiamine treatment started, even though the protein/hemoglobin levels in the body were still low. The patient, who had received parenteral thiamine therapy for 2 weeks, had already started eating and completed the rehabilitation process, and was discharged on Day 24, having regained the ability to ambulate independently.

DISCUSSION

Our experience with this patient highlights the clinical importance of combined Thiamine (vitamin B1) and vitamin B12 deficiency in a patient with acute heart failure and severe metabolic derangement [1, 2]. On admission, she presented with marked macrocytic anemia (hemoglobin 2.1 g/dL), mildly elevated blood lactate (2.5 mmol/L), generalized edema, and circulatory instability, findings consistent with severe "wet beriberi" [3]. Because both vitamins are involved in energy metabolism and cardiovascular function, the coexistence of these deficiencies may have contributed to the rapid progression of heart failure in this patient [6]. In our case, the patient's edema and hyperlactatemia improved promptly after thiamine administration, despite persistent anemia and hypoalbuminemia. This clinical course suggested that thiamine deficiency played a major role in her acute hemodynamic deterioration [3, 6]. However, it is also probable that immediate blood transfusions played a supportive yet significant role in stabilizing her acute cardiovascular status by improving her intravascular volume and systemic oxygen-carrying capacity. Ultimately, we believe that both prompt thiamine replenishment and blood transfusion synergistically drove her rapid recovery.

Beriberi has long been associated with the consumption of polished white rice in Japan, particularly in relation to the historical experience of the Imperial Japanese Navy during the Meiji era [7]. Although severe nutritional deficiency is less common today, thiamine deficiency is still encountered in clinical practice. It is often associated with alcoholism or socially marginalized people, but our patient did not fit these backgrounds. She lived independently as a homemaker and appeared socially stable. Further history taking revealed markedly restricted dietary intake related to long-standing personal food preferences and routines ("kodawari"). In this case, she regularly prepared meals for her family despite having poor nutritional intake herself [4].

A practical limitation in such cases is the delay in obtaining serum vitamin assays. In our case, waiting for laboratory confirmation before initiating treatment would likely have resulted in clinical deterioration. Vitamin B12 deficiency is often considered in patients with a history of gastric surgery or pernicious anemia; however, it may also be present in individuals with psychological distress or highly selective eating patterns [5]. In primary care settings, it is crucial to recognize that "selective malnutrition" can be insidious; rigid personality traits, underlying psychiatric disorders (such as panic disorder in this patient), or unexpressed idiosyncratic beliefs can drive strict, self-imposed dietary restrictions. In this patient, the combination of macrocytosis (MCV 152.4 fL) and elevated LD raised strong suspicion of megaloblastic anemia at an early stage, even in the absence of relevant surgical history. In primary care settings, it is often insufficient to ask broadly about household diet; more detailed inquiry into the patient's individual dietary habits and the psychological motivations behind them is necessary.

A key consideration in emergency practice is the timing of thiamine administration. In patients with suspected or subclinical deficiency, initiating glucose-containing fluids without concurrent thiamine supplementation may pose a significant risk. This approach has been associated with complications such as Wernicke's encephalopathy and, in severe cases, cardiovascular collapse [6]. Because glucose metabolism rapidly exhausts remaining thiamine stores, we maintain that empirical intravenous Thiamine is mandatory before or alongside glucose in any patient where nutritional risk is even a remote possibility [3, 6]. In both emergency and primary care, the decision to intervene must be born from clinical suspicion. While macrocytic anemia strongly points to vitamin B12 deficiency, the critical decision to initiate immediate thiamine rescue should be guided by the co-occurrence of dietary imbalance and even mild hyperlactatemia, rather than waiting for overt, textbook-defined lactic acidosis to develop [8]. Ultimately, because thiamine deficiency can drive rapid, life-threatening hemodynamic deterioration, clinicians must initiate empirical vitamin supplementation based on these subtle clinical clues immediately, rather than waiting for delayed laboratory results.

CONCLUSION

In conclusion, nutritional deficiencies remain a relevant cause of high-output heart failure in the modern era, even among individuals who appear socially integrated. Clinicians should maintain a high index of suspicion for vitamin B1 and B12 deficiencies when a patient presents with heart failure accompanied by macrocytic anemia and even mild hyperlactatemia, without waiting for the development of severe lactic acidosis. Scrutinizing personal dietary habits, beyond general household nutrition, is essential. Early empirical supplementation is a safe and life-saving intervention that can reverse

severe multi-organ failure and prevent irreversible neurological damage.

CONSENT FOR PUBLICATION

Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

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GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this work, the authors used ChatGPT (GPT-4, OpenAI) only to obtain language suggestions and to perform minor proofreading in some parts of the manuscript. After using this tool/service, the authors reviewed and edited the content as needed and took full responsibility for the published article.

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