DOI: 10.66069/ojspub.27450702

A Case Report of a Diabetic Foot Ulcer Complicated by Dry Beriberi and a Literature Review

Yuanyuan Yu, Jiamin Zeng, Zhen Li, Jingwen Cao, Haili Zhang, Tianying Yu, Xia Liu*

Department of Endocrinology I, Yulin Hospital of Traditional Chinese Medicine, Yulin 719000, Shaanxi, China
**Correspondence Author*

Abstract: ***Diabetic foot refers to foot ulcers, deep tissue infections, and even gangrene associated with distal lower limb neuropathy and varying degrees of peripheral vascular disease. It is characterized by high rates of disability and mortality and is one of the most severe chronic complications of diabetes. Dry beriberi is a nutritional metabolic disorder caused by vitamin B1 (thiamine) deficiency. It primarily affects the sensory and motor nerves of the limbs and can manifest as symmetrical peripheral neuropathy. Early symptoms include numbness, tingling, and a burning sensation in the extremities, which are clinically similar to those of diabetic peripheral neuropathy. Both conditions can occur simultaneously in the same patient; however, this clinical scenario is relatively rare and prone to misdiagnosis or missed diagnosis. This article reports the diagnosis and treatment process of a patient with diabetic foot ulcers complicated by dry beriberi and discusses the case in light of the literature to enhance clinicians' awareness of this complication.***

Keywords: Diabetic foot ulcer, Dry beriberi, Diabetic peripheral neuropathy, Thiamine.

1. Introduction

Diabetic foot refers to foot ulcers, deep tissue infections, and even gangrene associated with distal lower limb neuropathy and varying degrees of peripheral vascular disease. It is characterized by high rates of disability and mortality and is one of the most severe chronic complications of diabetes. Dry beriberi is a nutritional metabolic disorder caused by vitamin B1 (thiamine) deficiency. It primarily affects the sensory and motor nerves of the limbs and can manifest as symmetrical peripheral neuropathy. Early symptoms include numbness, tingling, and a burning sensation in the extremities, which are clinically similar to those of diabetic peripheral neuropathy. Both conditions can occur simultaneously in the same patient; however, this clinical scenario is relatively rare and prone to misdiagnosis or missed diagnosis. This article reports the diagnosis and treatment process of a patient with diabetic foot ulcers complicated by dry beriberi and discusses the case in light of the literature to enhance clinicians' awareness of this complication.

2. Case Details

A 54-year-old male patient was admitted on June 9, 2025, presenting with "elevated blood glucose levels for over 3 years and ulcerations on both feet for half a month." Three years ago, a routine physical examination revealed high blood glucose levels, leading to a diagnosis of "type 2 diabetes." He began treatment with oral "metformin hydrochloride tablets 0.5 g twice a day" but did not monitor his blood glucose levels. Half a month prior to admission, due to cold, painful, and numb feet, the patient self-administered a foot soak and accidentally scalded the tops of his feet. Subsequently, both feet gradually swelled and developed blisters, accompanied by significant pain, prompting him to seek medical attention. He had a history of cerebral infarction and cerebral atrophy. He had been drinking alcohol for over 10 years and had frequently engaged in heavy drinking for the past 3 years, consuming approximately half a jin of baijiu daily. Physical examination: T 36.5°C, P 95 bpm, R 20 bpm, BP 108/80 mmHg; height 163 cm, weight 45 kg, BMI: 16.93 kg/m². Alert but listless; no significant abnormal findings on cardiac, pulmonary, or abdominal examination. Both feet were swollen and exhibited skin hyperpigmentation. The skin on the anterior third of the dorsum of both feet was eroded, and scattered vesicles were visible on the dorsum of the toes and between the toes, accompanied by the discharge of pale yellow fluid and marked tenderness (Figure 1). Supporting tests: HbA1c 6.7%; complete blood count: white blood cell count 11.64×10^9/L, neutrophil percentage 83.5%, hemoglobin 132 g/L, platelets 416×10^9/L. C-reactive protein 30.3 mg/L. Erythrocyte sedimentation rate (ESR): 28 mm/h. No abnormalities were observed in liver function, kidney function, lipid profile, coagulation function, infectious disease screening, thyroid function, or immunological tests. Echocardiogram: reduced left ventricular diastolic function; normal systolic function. Color Doppler flow imaging showed no pathological regurgitation at any valves. Upper abdominal ultrasound: No abnormalities were observed in the liver, gallbladder, pancreas, spleen, or both kidneys. Lower extremity arterial ultrasound: Rough endothelial surfaces in both lower extremity arteries; plaque formation on the posterior walls of both common femoral arteries. Lower extremity venous ultrasound showed no abnormalities. Electromyography (EMG) showed: Slowed conduction velocity of sensory potentials in the right superficial peroneal nerve.

After admission, the patient received treatment to lower blood sugar, nourish nerves, improve circulation, and combat infection. Debridement and dressing changes were performed on the wounds on both feet. Following comprehensive treatment, the wounds on the patient's feet gradually healed, but symptoms of cold sensitivity, pain, and numbness in both lower limbs remained pronounced. Even with an electric blanket and thick blankets provided during hospitalization, the patient's lower limbs remained cold, which could not be alleviated, affecting sleep and causing restlessness. Pregabalin capsules 75 mg were prescribed orally twice daily

for pain relief, and amitriptyline hydrochloride tablets 25 mg were prescribed orally twice daily to alleviate anxiety symptoms. However, there was still no significant improvement in symptoms. Considering the patient's history of alcohol abuse, a provisional diagnosis of dry beriberi was made. Vitamin B1 injection 50 mg was immediately administered intramuscularly three times daily. Three days later, the patient's coldness and pain in the lower extremities had significantly improved, and the wound was gradually healing. After discharge, the patient continued taking 10 mg of vitamin B1 tablets orally three times daily. During an outpatient follow-up two weeks later, the wound on the patient's foot had largely healed, and symptoms had significantly improved (Figure 2).

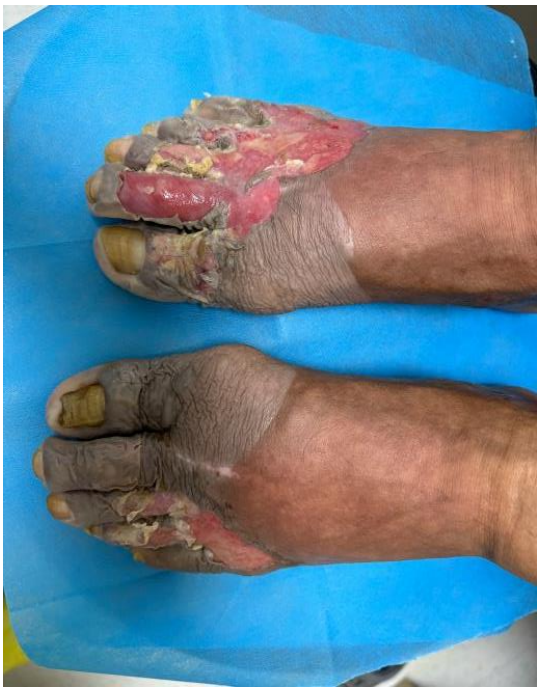

Figure 1: Foot wound at admission

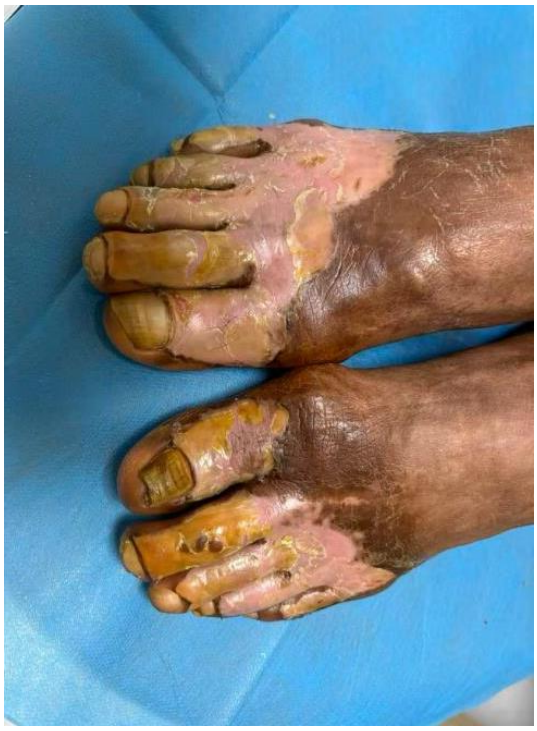

Figure 2: Foot wound at discharge

3. Discussion

Beriberi caused by vitamin B1 deficiency is now rare in China, but it still occurs from time to time in specific high-risk populations. Because the onset of this disease can be subacute and clinical manifestations are diverse—especially when peripheral nerve damage is the initial presentation—it is easily confused with diabetic peripheral neuropathy, alcoholic neuropathy, or other metabolic neuropathies, leading to missed or misdiagnoses. This report presents a case of diabetic foot ulcer complicated by dry beriberi and includes a literature review. It enhances the ability to recognize dry beriberi, particularly in patients with comorbidities, and is of significant clinical importance.

The body's stores of vitamin B1 are extremely low [1–4]; in the event of persistent dietary insufficiency or absorption disorders, these reserves can be depleted in as little as 2–3 weeks [5], leading to thiamine deficiency, also known as beriberi. Based on differences in the affected systems and clinical manifestations, the disease is primarily classified into four types: dry beriberi, wet beriberi, Wernicke's encephalopathy, and severe Shoshin beriberi. Among these, dry beriberi—characterized by peripheral nerve damage—is the most commonly misdiagnosed or overlooked clinically.

Epidemiological data show that chronic alcohol abuse is the primary risk factor for thiamine deficiency in developed countries and among clinically evaluated adult populations [6–7], with the prevalence of thiamine deficiency among heavy drinkers ranging from 25% to 80% [8].From a pathophysiological perspective, alcohol can disrupt thiamine metabolism in multiple ways: on the one hand, chronic alcohol consumption downregulates the expression of intestinal thiamine transporter 1, inhibiting the uptake and absorption of thiamine by intestinal epithelial cells [9]; on the other hand, alcohol hinders the phosphorylation of thiamine into its active form, TPP, significantly reducing the concentration of active thiamine in the body. At the same time, individuals with alcohol abuse often suffer from issues such as a monotonous diet, inadequate nutrient intake, and chronic gastrointestinal dysfunction, which further exacerbate thiamine deficiency and ultimately lead to impaired glucose metabolism. When vitamin B1 is deficient, glucose metabolism is disrupted: aerobic oxidation is inhibited, while anaerobic fermentation is enhanced, leading to the accumulation of pyruvate and lactate and a reduction in ATP synthesis. Due to their high energy metabolism, the nervous system and cardiac muscle are the primary target organs affected. In this case, the diabetic patient's long-term alcohol abuse triggered dry beriberi, presenting with cold, painful, and numb feet; soaking the feet in hot water resulted in burns to the feet.

The clinical manifestations of dry beriberi overlap significantly with those of diabetic peripheral neuropathy; both are characterized by distal, symmetrical peripheral nerve damage. This overlap presents a key challenge in clinical differential diagnosis and is the primary reason why diabetes complicated by beriberi is frequently misdiagnosed. Clinically, peripheral neuropathy is often attributed primarily to hyperglycemia-mediated microvascular damage and metabolic disorders, while the possibility of thiamine

deficiency is overlooked. However, there are significant differences between the two conditions in terms of etiology, onset characteristics, clinical features, laboratory indicators, and response to treatment: diabetic peripheral neuropathy has a chronic, insidious onset and a protracted course; it is often accompanied by other microvascular complications such as diabetic retinopathy and nephropathy; patients' blood lactate and pyruvate levels are typically within the normal range; and supplementation with vitamin B1 only partially alleviates symptoms; In contrast, dry beriberi has a relatively acute onset, with marked neurological deficits appearing within weeks to months. Patients often have concomitant malnutrition, and some may present with edema and serous effusions characteristic of wet beriberi. Laboratory findings show decreased serum vitamin B1 levels and significantly elevated blood lactate and pyruvate levels; clinical symptoms can be rapidly and significantly alleviated with targeted vitamin B1 supplementation. Therefore, in diabetic patients with peripheral neuropathy, if the condition progresses rapidly and cannot be explained solely by hyperglycemia and metabolic disturbances, or if it is accompanied by symptoms such as weakness, loss of appetite, or unexplained edema, clinicians should be highly vigilant for the possibility of concurrent dry beriberi.

The causes of thiamine deficiency are diverse and include not only alcohol abuse but also multiple factors such as inadequate intake, absorption disorders, the effects of medications, and increased metabolic demand. Improper food storage, repeated thawing, and prolonged cooking at high temperatures (above 120°C) result in significant loss of thiamine from food [10]; a long-term diet characterized by refined foods, a heavy reliance on polished white rice, and insufficient intake of fruits, vegetables, and meat is the primary cause of thiamine deficiency in the general population. At the same time, many commonly used clinical medications can interfere with thiamine absorption and metabolism. For example, acid-suppressing drugs such as omeprazole, as well as metformin, diuretics, antibiotics, antipsychotics, and anticancer drugs, can all inhibit intestinal thiamine absorption or accelerate its metabolic excretion; patients on long-term medication are at high risk for thiamine deficiency [11–12]. Furthermore, chronic wasting diseases, aging, gastrointestinal disorders, and the postoperative state following bariatric surgery increase the body's nutrient consumption and impair intestinal absorption, thereby further elevating the risk of beriberi.

Currently, the diagnosis of beriberi primarily relies on a comprehensive assessment of medical history, typical clinical manifestations, and the response to vitamin B1 treatment. Routine clinical measurement of serum thiamine concentrations is difficult to implement in most settings with limited medical resources due to its technical complexity, time-consuming nature, and the need for a cold chain and specialized laboratory support. Compared to complex laboratory testing, diagnostic treatment with vitamin B1 offers significant advantages in terms of safety, efficiency, and accessibility. Vitamin B1 is highly water-soluble; excess amounts ingested by the body are rapidly metabolized and excreted. To date, there have been no reports of thiamine toxicity from extremely high doses, making this treatment extremely safe. This patient developed neuropathy following long-term alcohol abuse, raising a strong suspicion of thiamine deficiency. Empirical supplementation therapy was promptly initiated, leading to rapid improvement in symptoms; the diagnosis was therefore dry beriberi due to thiamine deficiency. Empirical treatment is particularly suitable for patients with severe or rapidly progressing disease, as it prevents treatment delays caused by waiting for test results.

Regarding the treatment regimen, the core principles for this condition are eliminating the underlying cause, thiamine supplementation, and nutritional support. For patients with mild beriberi, oral vitamin B1 at 10 mg three times daily is sufficient to effectively correct the deficiency; for moderate-to-severe cases, priority should be given to rapidly increasing blood levels via intramuscular or intravenous administration, followed by a switch to oral sequential therapy once symptoms have subsided. Patients with alcohol-related beriberi require higher doses of thiamine [2]; the standard recommendation is 100–200 mg administered intravenously daily, with further dose increases necessary for critically ill patients. This is because alcohol exerts a dual inhibitory effect on both the absorption and activation of thiamine, making it difficult to achieve effective therapeutic concentrations with standard doses. In this case, a stepwise treatment regimen was adopted, involving rapid symptom control via intramuscular injection followed by oral maintenance therapy, which ultimately resulted in significant improvement in neurological function, further validating the clinical efficacy of this treatment strategy.

Vitamin B1 supplementation in patients with diabetes not only treats beriberi but may also provide additional benefits for glycemic control and neuropathy, thereby indirectly promoting the healing of diabetic foot ulcers. Vitamin B1 inhibits acetylcholinesterase activity, promotes acetylcholine synthesis, and improves nerve conduction; simultaneously, it reduces insulin resistance, exerting a dual positive effect on glycemic control and slowing the progression of diabetic peripheral neuropathy. Following supplementation, the patient's neurological symptoms improved significantly. After debridement and dressing changes, the diabetic foot ulcer healed completely, with a favorable prognosis, further supporting the importance of early identification and timely treatment.

4. Summary

In summary, dry beriberi and diabetic peripheral neuropathy show a high degree of overlap in clinical presentation, but there are clear differences in their etiology, onset patterns, accompanying manifestations, and response to vitamin B1. For patients with a history of chronic alcohol abuse, malnutrition, a monotonous diet, or long-term use of specific medications—especially those with diabetes—there should be a high degree of vigilance regarding the possibility of beriberi. In the absence of reliable laboratory tests, prompt empirical vitamin B1 supplementation serves not only as a safe and effective diagnostic tool but also as a key measure for improving patient prognosis.

Fund Project

Yulin Municipal Association for Science and Technology

Young Talent Support Program (20230538); Science and Education Project of the Yulin Municipal Science and Technology Bureau (2023-SF-58).

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