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CASE REPORT

Pulmonary artery catheter usage in diagnosis of Shoshin beriberi presented with unexplained lactic acidosis

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Abstract

Wet beriberi is a rare but fatal disease in modern society. The nonspecific clinical manifestations, including symptoms of heart failure and recalcitrant lactic acidosis, can prevent timely diagnosis. The use of a pulmonary artery catheter can promptly confirm a high cardiac output state and plays a crucial role in rapidly deteriorating cases. Appropriate treatment with intravenous administration of thiamine leads to dramatic recovery within hours. We present two cases of Shoshin beriberi, a fulminant variant of wet beriberi, diagnosed in 2016 and 2022 at our institute. The patients experienced haemodynamic collapse and refractory lactic acidosis, which were successfully diagnosed with the use of a pulmonary artery catheter and reversed by thiamine supplementation. We also reviewed 19 cases of wet beriberi reported between 2010 and 2022.

Keywords High-output heart failure; Wet beriberi; Shoshin beriberi; Lactic acidosis; Pulmonary artery catheter

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Introduction

Thiamine (vitamin B1) deficiency, also known as beriberi disease, has two major types: dry and wet. The former presents with peripheral neuropathy, whereas the latter presents with high-output heart failure. Shoshin beriberi is a fulminant variant of wet beriberi, described as a rapid haemodynamic disaster with multi-organ failure. It should be suspected in patients with causal factors, including alcoholism, chronic malnutrition, or long-term use of total parenteral nutrition. However, nonspecific clinical manifestations and rapid deterioration can prevent the timely diagnosis of thiamine deficiency. Consequently, the use of invasive haemodynamic monitoring with pulmonary artery catheter plays a crucial role in these cases. Clinical conditions reverse dramatically within hours after thiamine administration, whereas the untreated disease could be fatal. We present two cases of Shoshin beriberi with a history of alcoholism and chronic malnutrition. Both patients experienced haemodynamic collapse and refractory lactic acidosis.

Case Report

Case 1

A 47-year-old man presented to the emergency department with a 2-week history of progressive shortness of breath and general weakness. The patient had a history of hypertension and alcohol use disorder. His diet was monotonous with carbohydrates for the last 6 months. In addition, he engaged in binge drinking comprising >6 standard drinks.

Upon arrival, the patient was afebrile, tachycardiac (101 bpm), and hypotensive (73/38 mmHg). He showed tachypnoea (30/min) and oxygen desaturation (88% under ambient air) with accessory muscles in use. The patient had an engorged jugular vein and accentuated first heart sound. Auscultation revealed bilateral basal crackles. Pitting oedema was prominent in both legs. The extremities were warm, and his capillary refilling time was less than 2 s.

Electrocardiography revealed sinus tachycardia without ST-T abnormalities. Chest radiography revealed pulmonary congestion. Venous blood gas analysis suggested severe metabolic acidosis (pH, 6.55; $PaCO_2$, 62.2 mmHg; and HCO_3, 4.1 mmol/L). The serum lactate level was markedly elevated at 16.9 mmol/L. Acute kidney injury was observed, with creatinine levels rising from 0.9 to 11.6 mg/dL. Liver function was abnormal (alanine aminotransferase level, 114 U/L). Echocardiography revealed a hyperkinetic left ventricular ejection fraction (LVEF) of 70%, without regional wall motion abnormalities or valvular heart disease. The chamber size was within the normal range. Continuous-wave Doppler echocardiography revealed a systolic pulmonary artery pressure of 50 mmHg.

The patient received piperacillin/tazobactam after excluding hypovolemic, cardiogenic, and obstructive shocks. Continuous renal replacement therapy was initiated to treat severe metabolic acidosis. The patient's condition deteriorated; consequently, vasopressors (norepinephrine 0.30 μg/kg/min and vasopressin 0.06 U/min) were administered at high doses. A pulmonary artery catheter was used to tailor the therapy. The patient's cardiac output was 11.5 L/min, cardiac index was 5.4 L/min/m^2, pulmonary artery wedge pressure was 21.0 mmHg, and systemic venous resistance index was 1013 dynes/s/cm^{-5}/m^2.

High-output heart failure was suspected. The patient's history of alcohol use disorder and malnutrition (0.75 mg thiamine/day) prompted doctors to suspect Shoshin beriberi disease. Thiamine (200 mg) was administered twice daily. The lactic acidosis resolved dramatically, and the vasopressors were discontinued within 2 days of the first thiamine dose administration. Renal replacement therapy was discontinued subsequently. Creatinine levels returned to the baseline levels on day 3 post thiamine administration (*Figures 1* and *2*). On days 6 and 7, the patient was switched to oral thiamine therapy and transferred to the general ward, respectively.

Case 2

A 20-year-old man visited the emergency department with progressive exertional dyspnoea for 1 week. He was diagnosed with porencephaly and colpocephaly associated with mental retardation.

The patient was agitated, tachycardiac (120 b.p.m.), and hypotensive (87/47 mmHg) shortly after arrival. The oxygen saturation was 99% under ambient air. The jugular vein was engorged, and cardiac examination revealed regular heart rate without murmurs or extra heart sounds. Mild crackles

Figure 1 Liver and renal function significantly improved after thiamine administration in case 1. (ALT, alanine aminotransferase; vasoactive inotropic score, dopamine dose [μg/kg/min] + dobutamine dose [μg/kg/min] + 100 × epinephrine dose [μg/kg/min] + 10 × milrinone dose [μg/kg/min] + 10 000 × vasopressin dose [U/kg/min] + 100 × norepinephrine dose [μg/kg/min]).

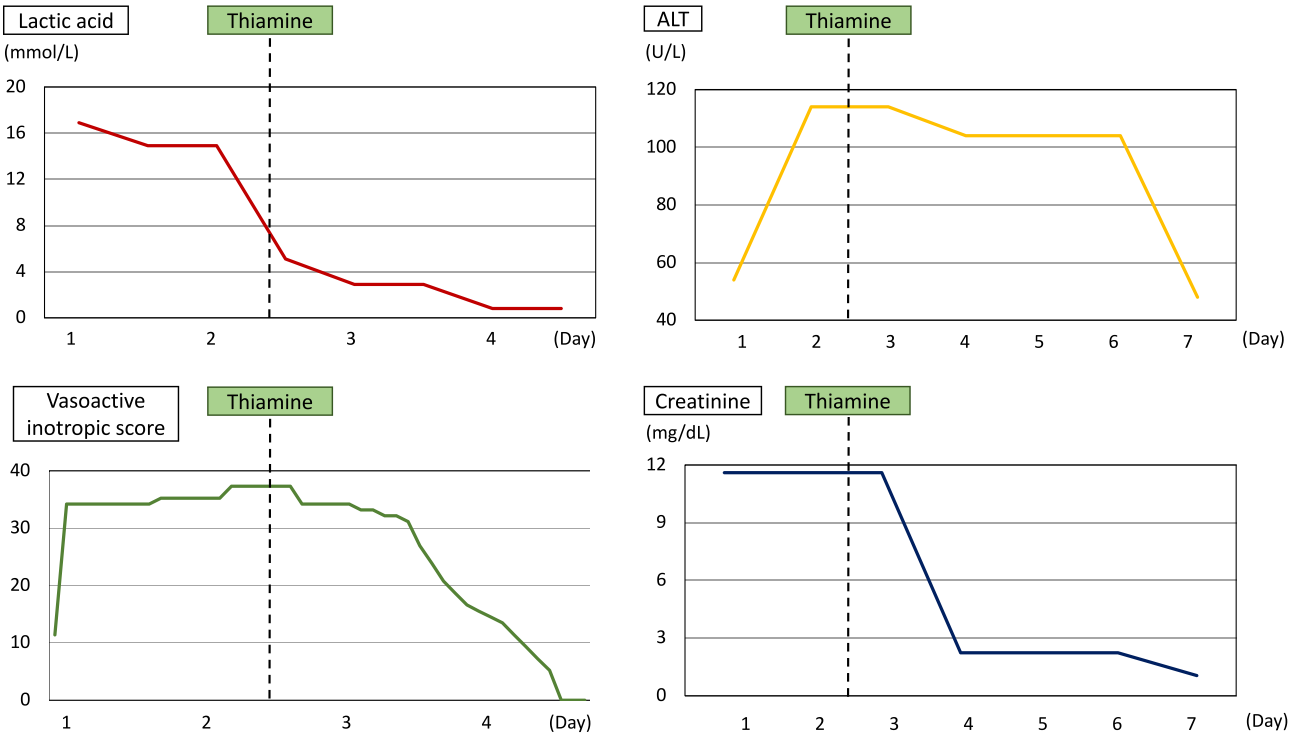

Figure 2 Serial haemodynamic monitoring with pulmonary artery catheter before and after thiamine administration in case 1 and 2. CO, cardiac output; CI, cardiac index; PAWP, pulmonary artery wedge pressure; CVP, central venous pressure; PAPm, mean pulmonary arterial pressure; SVRI, systemic venous resistance index; PVRI, pulmonary venous resistance index.

Case 1			
Time	Before thiamine	24 hrs after thiamine	48 hrs after thiamine
Norepinephrine (μg/kg/min)	0.30	0.12	0
Vasopressin (U/min)	0.06	0	0
CO (L/min)	11.00	9.56	6.38
CI (L/min/m^2)	5.37	4.64	3.14
PAWP (mmHg)	21	15	21
CVP (mmHg)	8	8	11
PAPm (mmHg)	36	36	22
SVRI (dynes-sec-cm^{-5}/m^2)	1013	1099	1577
PVRI (dynes-sec-cm^{-5}/m^2)	358	461	280

Case 2			
Time	Before thiamine	6 hrs after thiamine	18 hrs after thiamine
Norepinephrine (μg /kg/min)	0.41	0.41	0
Vasopressin (U/min)	0.02	0	0
Dopamine (μg /kg/min)	5.30	0	0
CO (L/min)	9.90	6.48	6.74
CI (L/min/m^2)	6.90	4.52	4.70
PAWP (mmHg)	15	16	15
CVP (mmHg)	16	20	15
SVRI (dynes-sec-cm^{-5}/m^2)	564	1257	1089
PVRI (dynes-sec-cm^{-5}/m^2)	357	407	238

were heard bilaterally in the basilar lungs. Bilateral lower-extremity oedema was observed. No focal deficits were noted on neurological examination. Venous blood gas analysis revealed pronounced lactic acidosis (13.5 mmol/L; pH, 7.34; $PaCO_2$, 16.0 mmHg; HCO_3, 8.9 mmol/L). Laboratory investigations showed abnormal liver (aspartate aminotransferase, 258 U/L; γ-glutamyl transferase, 29.0 U/L; total bilirubin, 5.0 mg/dL; direct bilirubin, 1.0 mg/dL) and renal (blood urea nitrogen, 25.3 mg/dL; creatinine 1.8 mg/dL) functions. The C-reactive protein level was low (0.9 mg/dL). The N-terminal pro-B-type natriuretic peptide level was elevated at 25 881 pg/mL. The haemogram and coagulation profile implied disseminated intravascular coagulopathy with intravascular haemolysis (haemoglobin, 12.8 mg/dL; platelet, 91.0 k/μL; international normalized ratio, 1.3, D-dimer, 7.1 μg/mL; haptoglobin, <30 mg/dL; lactate dehydrogenase 595.0 U/L). Echocardiography revealed preserved LVEF (50%) with 40 mmHg systolic pulmonary artery pressure. Of note, paradoxical interventricular septal motion was observed along with a D-shaped left ventricle and dilated right heart chambers. Chest computerized tomography revealed engorged pulmonary trunk without evidence of pulmonary embolism.

Broad-spectrum antibiotics were administered empirically; however, the patient's condition did not improve. Lactic acidosis progressed despite the administration of continuous renal replacement therapy. Haemodynamic evaluation using a pulmonary artery catheter revealed high output heart failure with low peripheral vascular resistance, 9.9 L/min cardiac output, 6.9 L/min/m^2 cardiac index, 15.0 mmHg pulmonary artery wedge pressure, and 564.0 dynes/s/cm^{-5}/m^2 systemic venous resistance index under 5.30 μg/kg/min dopamine, 0.41 μg/kg/min norepinephrine, and 0.02 U/min vasopressin.

The family reported that the patient had a soft diet of only rice and fish. Unusual causes of vasodilatory shock and high-output heart failure were considered, and Shoshin beriberi disease was highly suspected. A therapeutic trial of 100 mg thiamine was administered intravenously. Subsequently, increased blood pressure and systemic vascular resistance index were noted. Moreover, the lactic acidosis resolved and the cardiac index decreased (*Figure 2*). The three-line vasopressors were discontinued within 8 hours. Next, the liver and renal functions and disseminated intravascular coagulopathy recovered. The patient received intravenous 100 mg thiamine every 8 h. Complete recovery from multiple organ failures was noted within 2 days.

Discussion

Thiamine pyrophosphate is a cofactor for pyruvate dehydrogenase and α-ketoglutarate dehydrogenase, which are essential components of the Krebs cycle.[1] Thiamine depletion leads to lactic acid accumulation, which decreases systemic vascular resistance and activates the renin-angiotensin-aldosterone system, thereby increasing the cardiac preload. High cardiac output, defined as a resting cardiac output of >8.0 L/min or cardiac index of >4.0 L/min/m^2, ultimately leads to cardiac remodelling and congestive heart failure.[2]

Diagnosis of Shoshin beriberi is often difficult due to its vague and nonspecific symptoms, including general weakness, shortness of breath, haemodynamic instability, lactic acidosis, unexplained high-output heart failure, and multi-organ failure with rapid clinical deterioration. Beriberi disease rarely occurs in developed countries. However, there are populations that remain at risk, including those with a history of heavy alcohol consumption, malnutrition, gastric bypass surgery, or long-term use of total parenteral nutrition. *Table 1* lists 19 reported cases of wet beriberi disease over the past

Table 1 Reported cases of wet beriberi disease between 2010 and 2022

	Case 1 (our case)	Case 2 (our case)	Case 3[5]	Case 4[5]
Reported year	2022	2022	2022	2022
Age and gender	47M	20M	54F	42M
Aetiology	Alcoholism	Malnutrition (unbalanced diet)	Alcoholism	Alcoholism
Co-morbidity	Hypertension	Porencephaly, colpocephaly, mental retardation	Nil	Human immunodeficiency virus
Presentation	Dyspnoea, generalized weakness, haemodynamic instability	Dyspnoea, haemodynamic instability	Dyspnoea, nausea, vomiting, haemodynamic instability	Dyspnoea, altered mental status, haemodynamic instability
Physical examination	Jugular vein distension, bilateral crackles, pitting oedema	Jugular vein distension, bilateral crackles, pitting oedema	Parotidomegaly, right lower abdominal quadrant tenderness	Bilateral crackles
Laboratory test	• Lactic acidosis (arterial pH 6.55, pCO_2 62.2 mmHg, HCO_3 4.1 mmol/L, lactate 16.9 mmol/L) • AKI on CKRT • Abnormal liver function	• Lactic acidosis (arterial pH 7.34, pCO_2 16.0 mmHg, HCO_3 8.9 mmol/L, lactate 13.5 mmol/L) • AKI on CKRT • Abnormal liver function • Disseminated intravascular coagulopathy	• Lactic acidosis (arterial pH 7.12, HCO_3 3.0 mmol/L, lactate 21.0 mmol/L) • AKI • Abnormal liver function	• Lactic acidosis (arterial pH 7.04, pCO_2 12.7 mmHg, HCO_3 7.2 mmol/L, lactate 20.0 mmol/L) • Abnormal liver function
ECG and echocardiogram	• LVEF of 70% • Hyperkinetic status	• LVEF of 50% • Dilated right heart chambers with D-shape left ventricle	• ST-segment elevation in V3-V6 • Fair contractility	• ST-segment depression in V4-V6 • Fair contractility
Invasive haemodynamic monitoring	• High cardiac output (11.5 L/min) • Low systemic vascular resistance (1013 dynes/s/cm^{-5}) • High pulmonary capillary wedge pressure (21 mmHg)	• High cardiac output (9.9 L/min) • Low systemic vascular resistance (564 dynes/s/cm^{-5}) • High pulmonary capillary wedge pressure (15 mmHg)	Not documented	Not documented
Initial treatment	Thiamine 200 mg IV	Thiamine 100 mg IV	Thiamine 300 mg IV	Thiamine 300 mg IV
Outcome	• Lactic acidosis resolved in 24 h • Haemodynamic indices improved in 24 h • Urine output increased in 48 h	• Off vasopressor in 8 h • Completely recovered from multiple organ failures in 2 days	• Lactic acidosis resolved in 27 h • Liver function improved in 72 h • ST-segment resolution	• Lactic acidosis improved in 3 h • Haemodynamic indices improved in 3 h • Widespread T-wave inversion

	Case 5[6]	Case 6[7]	Case 7[8]	Case 8[9]
Reported year	2022	2021	2020	2019
Age and gender	40M	74M	69F	63M
Aetiology	Alcoholism, malnutrition	Alcoholism	Malnutrition (TPN use)	Alcoholism, malnutrition (unbalanced diet)
Co-morbidity	Nil	Nil	Previous Whipple procedure	Diabetes mellitus, hypertension
Presentation	Dyspnoea, chest tightness, altered mental status, haemodynamic instability on ECMO	Dyspnoea, weight gain	Chest discomfort on exertion, oliguria	Dyspnoea, altered mental status
Physical examination	Pitting oedema	Wide pulse pressure, jugular vein distension, Quincke's pulse (alternating flushing of nail beds in concert with cardiac cycle)	Jugular vein distension	Jugular vein distension, bilateral rales, pitting oedema

Table 1 (continued)

	Case 5[6]	Case 6[7]	Case 7[8]	Case 8[9]
Laboratory test	• Lactic acidosis (arterial pH 7.47, pCO_2 12.7 mmHg, HCO_3 7.2 mmol/L, lactate 20.0 mmol/L) • AKI on CKRT • Abnormal liver function	Low thiamine diphosphate concentration (39 nmol/L, reference 70–180 nmol/L)	Low thiamine concentration (<10 ng/mL, reference 20-60 ng/mL)	Undetectable thiamine concentration
Echocardiogram and coronary angiography	• LVEF of 50% • Patent coronary arteries	Not documented	Not documented	• LVEF of 20% • Patent coronary arteries
Invasive haemodynamic monitoring	• Measured by pulse indicator continuous cardiac output • Normal cardiac index (3.5 L/min/m^2) • Low systemic vascular resistance (463 dynes/s/cm^{-5})	• High cardiac output (12.3 L/min) • Low systemic vascular resistance (587 dynes/s/cm^{-5})	High cardiac index (5.85 L/min/m^2)	Not documented
Initial treatment Outcome	Thiamine 200 mg IM • Narrowing of pulse pressure by 40 mmHg in 28 weeks • Symptoms completely resolved	Thiamine 100 mg IV Haemodynamic indices improved in 12 h	Thiamine 100 mg IV Symptoms and lactic acidosis improved in 24 h	Thiamine 100 mg IV • Lactic acidosis resolved in 12 h • ECMO removed on day 7 • LV function normalized in few days

	Case 9[10]	Case 10[11]	Case 11[12]	Case 12[13]
Reported year	2018	2017	2016	2016
Age and gender	39M	39M	17F	61M
Aetiology	Alcoholism	Malnutrition (Hikikomori syndrome)	Malnutrition (TPN use)	Alcoholism, malnutrition (unbalanced diet)
Co-morbidity	Bilateral congenital foot deformity	Amnesia	Congenital jejunal atresia with gut malrotation	Nil
Presentation	Dyspnoea, palpitations, vomiting, haemodynamic instability	Generalized weakness	Haemodynamic instability on ECMO, respiratory failure	Dyspnoea, generalized weakness, anuria, haemodynamic instability
Physical examination	Jugular vein distension, pitting oedema	Jugular vein distension, pitting oedema	Not documented	Pitting oedema
Laboratory test	• Lactic acidosis (arterial pH 7.41, pCO_2 17.4 mmHg, HCO_3 10.3 mmol/L, lactate 5 mmol/L) • AKI on CKRT • Acute liver injury	Lactic acidosis (HCO_3 6.8 mmol/L, lactate 7.1 mmol/L)	• Lactic acidosis (arterial pH 7.26, pCO_2 42 mmHg, lactate 28.6 mmol/L) • AKI on CKRT	• Lactic acidosis (lactate 6.2 mmol/L) • Low thiamine concentration (13 ng/mL)
Echocardiogram and coronary angiography	• LVEF of 77% • Dilatation of left atrium	Not documented	• LVEF of 25% • Patent coronary arteries	LVEF of 85%
Invasive haemodynamic monitoring	• High cardiac output (9.06 L/min) • Low systemic vascular resistance (397 dynes/s/cm^{-5}) • High pulmonary capillary wedge pressure (13 mmHg)	High cardiac output (15 L/min)	Not documented	• High cardiac index (5.35 L/min/m^2) • Low systemic vascular resistance (400 dynes/s/cm^{-5}) • High pulmonary capillary wedge pressure (18 mmHg)

Table 1 (continued)

	Case 9[10]	Case 10[11]	Case 11[12]	Case 12[13]
Initial treatment	Thiamine 100 mg IV	Thiamine 100 mg IV	Thiamine 100 mg IV	Thiamine 100 mg IV
Outcome	• Anuria resolved in 7 h • Lactic acidosis resolved in 47 h • Cardiac index of 3.93 L/min/m^2 20 days after thiamine administration	Off vasopressor in 48 h	• Off vasopressor in 6 h • Lactic acidosis resolved in 24 h	• Lactic acidosis resolved in 12 h • Increased urine output and off vasopressors in 24 h • Completely recovered from multiple organ failures in 2 days

	Case 13[14]	Case 14[14]	Case 15[14]	Case 16[14]
Reported year	2015	2015	2015	2015
Age and gender	45M	60F	23M	48F
Aetiology	Malnutrition (TPN use for 15 days)	Malnutrition (TPN use for 29 days)	Malnutrition	Malnutrition (TPN use with for 26 days)
Co-morbidity	Roux-en-Y anastomosis due to peptic ulcer disease	Pancreatic adenocarcinoma with peritoneal metastases	Von Gierke's disease, supplemented by starches	End stage renal disease on PD, PD catheter-related peritonitis
Presentation	Acute abdominal pain, haemodynamic instability	Haemodynamic instability	Nausea, vomiting, generalized weakness, severe loss of appetite and paraparesis, haemodynamic instability	Altered mental status, respiratory failure, haemodynamic instability
Physical examination	Not documented	Not documented	Not documented	Not documented
Laboratory test	Lactic acidosis (arterial pH 7.26, pCO_2 19 mmHg, HCO_3 9.0 mmol/L, lactate 23 mmol/L)	Lactic acidosis (arterial pH 7.38, HCO_3 22.0 mmol/L, lactate 13.6 mmol/L)	• Lactic acidosis (arterial pH 7.19, HCO_3 7.7 mmol/L, lactate 31 mmol/L) • AKI on CKRT • Abnormal liver function	Lactic acidosis (arterial pH 7.27, pCO_2 21 mmHg, HCO_3 9.0 mmol/L, lactate 34 mmol/L)
Echocardiogram	Not documented	Not documented	Not documented	Not documented
Invasive haemodynamic monitoring	Not documented	Not documented	Not documented	Not documented
Initial treatment	Thiamine 100 mg IV	Thiamine 100 mg IV	Thiamine 100 mg IV	Thiamine 200 mg IV
Outcome	Off vasopressor in 48 h	• Off vasopressor in 6 h • Lactic acidosis resolved in 24 h	• Lactic acidosis resolved in 12 h • Increased urine output and off vasopressors in 24 h • Completely recovered from multiple organ failures in 2 days	• Off vasopressors in 12 h, later death due to small bowel ischaemia

	Case 17[15]	Case 18[16]	Case 19[17]
Reported year	2014	2013	2010
Age and gender	61M	42F	25M
Aetiology	Diuretics (furosemide 40 mg/day, trichlormethiazide 1 mg/day for 6 months)	Alcoholism,malnutrition (anorexia nervosa)	Malnutrition (vegetable refuser)
Co-morbidity	Diabetes mellitus, chronic kidney disease stage 3	Nil	Nil
Presentation	Orthopnoea, haemodynamic instability		

(*Continues*)

Table 1 (continued)

	Case 17[15]	Case 18[16]	Case 19[17]
		Altered mental status, haemodynamic instability	Dyspnoea, altered mental status, oliguroa, haemodynamic instability
Physical examination	Not documented	Not documented	Jugular vein distension, pitting oedema
Laboratory test	• Lactic acidosis (arterial pH 7.39, pCO_2 12.8 mmHg, HCO_3 7.7 mmol/L, lactate 8.6 mmol/L) • AKI on CKRT	Lactic acidosis (lactate 9.3 mmol/L)	• Lactic acidosis (arterial pH 6.905, pCO_2 12.6 mmHg, HCO_3 2.8 mmol/L, lactate 27 mmol/L) • Abnormal liver function
Echocardiogram	Not documented	Not documented	Not documented
Invasive haemodynamic monitoring	• High cardiac index (8.0 L/min/m^2) • Low systemic vascular resistance (728 dynes/s/cm^{-5}) • High pulmonary capillary wedge pressure (18 mmHg)	Not documented	Not documented
Initial treatment	Thiamine 100 mg IV	Thiamine 100 mg IV	Thiamine 100 mg IV
Outcome	• Haemodynamic indices improved in 6 h • Cardiac index of 4.3 L/min/m^2 in 60 h	Haemodynamic indices improved in 45 min	• Lactic acidosis resolved in 12 h • Haemodynamic indices and urine output improved in 24 h • Completely recovered from multiple organ failure in 2 days

AKI, acute kidney injury; CKRT, continuous kidney replacement therapy; IV, intravenous; LVEF, left ventricular ejection fraction; ECMO, extra corporeal membrane oxygenation; PD, peritoneal dialysis; TPN, total parenteral nutrition.

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decade. Like the present cases, comprehensive history and physical examination are important in the diagnosis of patients, which is subsequently confirmed by echocardiography and haemodynamic monitoring. Although previous studies suggested that the routine use of pulmonary artery catheters does not improve survival,[3–4] it can facilitate the prompt confirmation of a high-output state.

Timely diagnosis of Shoshin beriberi is crucial. Laboratory tests, including plasma thiamine concentration or erythrocyte transketolase activity measurement, have limited sensitivity and specificity. The only definitive diagnostic method is the intravenous administration of thiamine (100–500 mg), which serves as a therapeutic trial and dramatically reverses the profound cardiovascular collapse and lactic acidosis. A maintenance dosage of 100 mg/day, either intravenously or orally, should be administered for at least 2 weeks.[2]

In the present cases, thiamine supplementation promptly reversed both haemodynamic disasters and metabolic disturbances. The diagnosis relied on a thorough patient history: the first case of alcoholism and malnutrition, and the second of chronic malnutrition. Subsequently, the timely use of a pulmonary artery catheter confirmed the high cardiac output state, thereby playing an essential role in these rapidly deteriorating cases. Accordingly, whenever the patient presents with high-output heart failure associated with unexplained lactic acidosis in the setting of the relevant medical histories, the diagnosis of Shoshin beriberi should always be considered.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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