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ACUTE HYPOXEMIC RESPIRATORY FAILURE WITH SEVERE HYPERLACTATEMIA AND MIXED ACID–BASE DISORDER IN A PATIENT WITH PREVIOUS MYOCARDIAL INFARCTION: A CASE REPORT

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ABSTRACT

Introduction: Acute heart failure (AHF) presenting as acute pulmonary edema is a leading cause of acute hypoxemic respiratory failure (AHRF). Elevated lactate identifies organ injury and a worse prognosis in AHF even without overt hypoperfusion, and lactate combined with acidemia strongly modifies mortality in cardiogenic shock; when severe hyperlactatemia coexists with a primary respiratory alkalosis, a near-normal pH may conceal the true metabolic severity.[1–3]

Case illustration: A 48-year-old man with a previous ST-elevation myocardial infarction (STEMI) treated with percutaneous coronary intervention (PCI) and a prior episode of AHF presented with dyspnea since the previous night, respiratory rate 41 breaths/min, SpO_2 78% on room air (87% on a non-rebreathing mask), blood pressure 148/103 mmHg, heart rate 120 beats/min, warm extremities and bilateral crackles with wheezing.

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Arterial blood gas (reported FiO_2 0.81) showed pH 7.35, $PaCO_2$ 21 mmHg, PaO_2 52 mmHg, HCO_3^- 11.8 mmol/L, base excess −14 mmol/L and lactate 11.20 mmol/L; $PaCO_2$ below the Winter's range (23.7–27.7 mmHg) indicated a concurrent primary respiratory alkalosis. High-sensitivity troponin I was 65.2 pg/mL (≈1.7 × upper reference limit), creatinine 1.84 mg/dL, potassium 2.8 mmol/L and glucose 300 mg/dL. Despite nitroglycerin, furosemide and mask oxygen he required intubation, after which high respiratory drive (total rate 43 breaths/min; tidal volume ≈10.6 mL/kg predicted body weight), severe hypoxemia (SpO_2 93% on FiO_2 0.85) and a fall in blood pressure to 101/55 mmHg were documented.[4–6]

Discussion: The ≈13% reduction in arterial oxygen content could not by itself explain a lactate of 11.2 mmol/L, implying superimposed normotensive low-output hypoperfusion, catecholamine-driven aerobic glycolysis—mirrored by hypokalemia and hyperglycemia—alkalosis-enhanced glycolysis, increased work of breathing and reduced clearance. The near-normal pH concealed a profound base deficit, a metabolic–hemodynamic discordance poorly captured by blood pressure-based staging, while vigorous post-intubation effort carried a risk of patient self-inflicted lung injury.[7–10]

Conclusion: In AHF complicated by AHRF, severe hyperlactatemia with a mixed acid–base disorder is a high-risk phenotype even when blood pressure is preserved. Systematic acid–base analysis, serial lactate, early echocardiography and lung ultrasound, timely evaluation for acute coronary syndrome, potassium repletion before or with insulin and diuretics, and lung-protective ventilation that controls respiratory drive are essential.[11,12]

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Keywords: Acute hypoxemic respiratory failure; Hyperlactatemia; Mixed acid–base disorder; Acute heart failure; Myocardial infarction

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INTRODUCTION

Background

The 2021 European Society of Cardiology (ESC) guidelines describe four presentations—acute decompensated heart failure, acute pulmonary edema, isolated right ventricular failure and cardiogenic shock—and respiratory failure and peripheral hypoperfusion cut across these phenotypes and largely determine early management in the emergency department.[1,13]

In survivors of myocardial infarction, a new AHF episode obliges clinicians to exclude recurrent acute coronary syndrome (ACS), stent-related events and mechanical complications, because AHF presumed secondary to ongoing ischemia warrants an immediate invasive strategy; conversely, high-sensitivity troponin elevation must be interpreted in context to distinguish type 1 from type 2 infarction and non-ischemic injury.[12,14]

Acute pulmonary edema produces AHRF through alveolar flooding, intrapulmonary shunt, ventilation–perfusion mismatch and reduced compliance, and must be distinguished from—or recognized alongside—acute respiratory distress syndrome (ARDS) and pneumonia; point-of-care lung ultrasound is more sensitive than chest radiography for cardiogenic edema.[15–17]

Blood lactate is a key prognostic biomarker across AHF. Among 237 patients with a "warm" profile, 43% had lactate ≥2 mmol/L, which was associated with organ injury and higher one-year mortality (36% vs. 21%); in cardiogenic shock, lactate ≥5 mmol/L or pH <7.2 increases 30-day mortality within every shock stage, lactate is part of the CardShock score, and early clearance strongly predicts survival.[2,3,18–20]

Hyperlactatemia, however, is not synonymous with tissue hypoxia: type A results from inadequate oxygen delivery, whereas type B arises from adrenergic aerobic glycolysis coupled to skeletal-muscle Na^+/K^+-ATPase, mitochondrial dysfunction, drugs or impaired clearance, so that an identical value may reflect very different physiological states.[7,8,21]

Acid–base disturbances in AHF are often interpreted superficially. In the Korean Heart Failure (KorHF) registry, respiratory alkalosis was the commonest disorder at admission, whereas

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acidosis—most often mixed—affected about one-fifth of patients and independently predicted 12-month mortality; because opposing primary processes can neutralize each other, formal compensation analysis is essential.[5,22–24]

The Society for Cardiovascular Angiography and Interventions (SCAI) SHOCK classification grades cardiogenic shock from stage A to E, yet staging remains anchored to blood pressure, leaving normotensive or hypertensive patients with marked biochemical hypoperfusion in an uncertain position.[9,25–27]

Non-invasive ventilation reduces intubation in cardiogenic pulmonary edema, but emergency intubation is complicated by major adverse events in almost half of critically ill patients, and high respiratory drive after intubation may generate injurious transpulmonary pressure swings—patient self-inflicted lung injury (P-SILI).[10,28–30]

Prepared according to the CARE (CAse REport) guidelines, this report describes such a patient in a regional public hospital in Indonesia, interpreted with quantitative bedside physiology.[4,13]

Objectives

The general objective is to describe and critically analyze the presentation, diagnostic reasoning and early management of this patient in relation to current guidelines and evidence.[4,13]

The specific objectives are to (i) perform a stepwise acid–base analysis showing how a primary respiratory alkalosis concealed profound metabolic acidosis; (ii) dissect the mechanisms of hyperlactatemia using oxygen-content calculations and coexisting electrolyte and glycemic abnormalities; (iii) interpret myocardial injury, electrocardiographic and radiographic findings after prior infarction; (iv) analyze ventilator data in terms of tidal volume per predicted body weight, driving pressure and respiratory drive; and (v) derive practical, evidence-based recommendations.[5–7,14]

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Benefits of the Report

Clinical benefit. A reproducible bedside framework—Winter's formula, oxygen-content estimation, glucose-corrected sodium, rate-corrected QT and predicted-body-weight normalization of tidal volume—for settings without advanced hemodynamic monitoring.[5,6,31,32]

Educational benefit. Illustration of common pitfalls: reliance on pH alone, Bazett overestimation of QTc at high heart rates, attribution of lactate solely to hypoperfusion, and insulin and loop diuretics given during severe hypokalemia without documented repletion.[8,23,33,34]

Institutional benefit. Support for protocolized AHF pathways in regional hospitals, including early non-invasive ventilation, vasodilator titration, diuretic-response assessment, electrolyte-repletion protocols and point-of-care ultrasound training.[13,28,34,35]

Scientific benefit. Highlighting an under-characterized phenotype—hypertensive acute pulmonary edema with severe hyperlactatemia—and the need for prospective data on metabolic–hemodynamic discordance in Indonesian and Southeast Asian populations.[2,3,9]

Research Gap

Evidence linking lactate to outcome derives mainly from cardiogenic shock and sepsis cohorts, and although the SCAI classification now includes risk modifiers and a three-axis model, guidance on classifying patients whose biochemistry suggests advanced shock while blood pressure is preserved is lacking.[2,3,8,9,26]

Literature on sympathetic crashing acute pulmonary edema (SCAPE) focuses on vasodilators and non-invasive ventilation, with little attention to hyperlactatemia, hypokalemia and stress hyperglycemia and scarce Indonesian contributions, and few case reports integrate ventilator-derived physiology with the metabolic profile.[10,16,36–39]

Novelty

This report (i) integrates quantitative interpretation of routine bedside data, including Winter's formula, the alveolar–arterial gradient, oxygen content, glucose-corrected sodium, multiple QT corrections and normalized ventilator volumes; (ii) describes a metabolic–hemodynamic discordance with lactate and base deficit typical of advanced shock despite hypertension; (iii)

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identifies the hyperlactatemia–hypokalemia–hyperglycemia triad as a unifying adrenergic signature; and (iv) quantifies the negligible contribution of a low-rate lactated Ringer's infusion to the measured lactate.[5,8,9,32,40]

CASE PRESENTATION

Patient Information

A 48-year-old Indonesian man presented to the emergency department of a regional public hospital with dyspnea that began the previous night and worsened the following morning, without chest pain. His history included a previous AHF episode and a STEMI treated with PCI. In accordance with CARE recommendations, all identifiers were removed from the text and masked on every reproduced image.[4]

Clinical Findings

On arrival the patient was conscious and in severe respiratory distress (Table 1); weight was 75 kg and height 170 cm (predicted body weight ≈66.0 kg). The presentation corresponded to the acute pulmonary edema phenotype with a "warm–wet" profile, and its abrupt, hypertensive character is consistent with SCAPE, in which a catecholamine surge redistributes fluid into the lungs rather than producing gross volume overload.[1,13,36]

Table 1. Vital signs and physical findings at presentation

Parameter	Finding	Interpretation
Consciousness	Conscious, alert	Preserved cerebral perfusion
Blood pressure (MAP)	148/103 mmHg (≈118 mmHg)	Hypertensive presentation
Heart rate; shock index	120 beats/min; 0.81	Sinus tachycardia; mildly elevated index
Respiratory rate; temperature	41 breaths/min; 36.8 °C	Severe tachypnea; afebrile
SpO_2	78% (room air) → 87% (NRM 15 L/min)	Severe hypoxemia refractory to conventional oxygen
Lungs; heart	Bilateral crackles and wheezing; regular rhythm, no murmur	Congestion; absent murmur does not exclude acute mitral regurgitation
Peripheries	Warm extremities; palpable pulses; no edema; soft abdomen	"Warm–wet" clinical profile

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Notes: MAP = DBP + (SBP − DBP)/3; PBW (men) = 50 + 0.91 × (height in cm − 152.4); body mass index ≈26.0 kg/m^{2}.[6] MAP, mean arterial pressure; NRM, non-rebreathing mask; PBW, predicted body weight.

Timeline

Events reconstructed from time-stamped records are presented in Table 2; unrecorded items are flagged for completion from the original charts.[4]

Table 2. Timeline of clinical events on the day of presentation

Time	Event / key findings
Previous night	Onset of dyspnea, progressive worsening by the morning
Arrival	BP 148/103 mmHg, HR 120/min, RR 41/min, T 36.8 °C; SpO_2 78% → 87% (NRM 15 L/min); crackles and wheezing
09:39	12-lead ECG: sinus tachycardia ≈119/min, QRS 124 ms, QT/QTc (Bazett) 340/480 ms
10:19–10:54	Blood count, renal function, electrolytes and hs-troponin I sampled (10:19) and reported (10:54)
10:41–10:49	ABG (FiO_2 0.81): pH 7.35, $PaCO_2$ 21, PaO_2 52 mmHg, HCO_3^- 11.8, BE −14, lactate 11.20 mmol/L
Emergency department	NRM 15 L/min; furosemide 40 mg IV then 5 mg/h; nitroglycerin 50 µg/min; insulin aspart 8 U; ranitidine 50 mg IV; lactated Ringer's 8 drops/min; urinary catheter (no initial output)
13:14–13:20	BP 117/76 mmHg (MAP 87), HR 121/min, SpO_2 81%, RR 43/min on NRM
13:20–15:11	Intubation, SIMV-PC; first display: total rate 43/min, VTe 702 mL (≈10.6 mL/kg PBW), VE 20.7 L/min *[exact time, induction agents and peri-intubation events to be confirmed]*
15:11–16:11	BP 101/55 mmHg (MAP 73), HR 94/min; second display: VT 344 mL (≈5.2 mL/kg), total rate 38/min, $EtCO_2$ 22 mmHg, SpO_2 93% on FiO_2 0.85
17:43	Portable supine chest radiograph; <100 mL of concentrated urine (collection interval not recorded)

Notes: ABG, arterial blood gas; BE, base excess; $EtCO_2$, end-tidal CO_2; SIMV-PC, synchronized intermittent mandatory ventilation–pressure control; VE, minute ventilation; VT/VTe, (exhaled) tidal volume.

Laboratory Investigations

Results are shown in Table 3, with original de-identified reports in Appendix A. The blood count showed mild neutrophilic leukocytosis, relative lymphopenia, monocytosis and marked eosinopenia with normal hemoglobin and platelets. Creatinine was 1.84 mg/dL (eGFR 46 mL/min/1.73 m^{2}) with urea only 17 mg/dL, potassium 2.8 mmol/L, glucose 300 mg/dL and hs-troponin I 65.2 pg/mL; glucose-corrected sodium (136.2–137.8 mmol/L) indicated largely translocational hyponatremia. Baseline creatinine, serial troponin, natriuretic peptide, chloride, magnesium, albumin, liver tests, ketones and glycated hemoglobin were unavailable.[14,31,41,42]

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Table 3. Laboratory results at presentation with derived indices

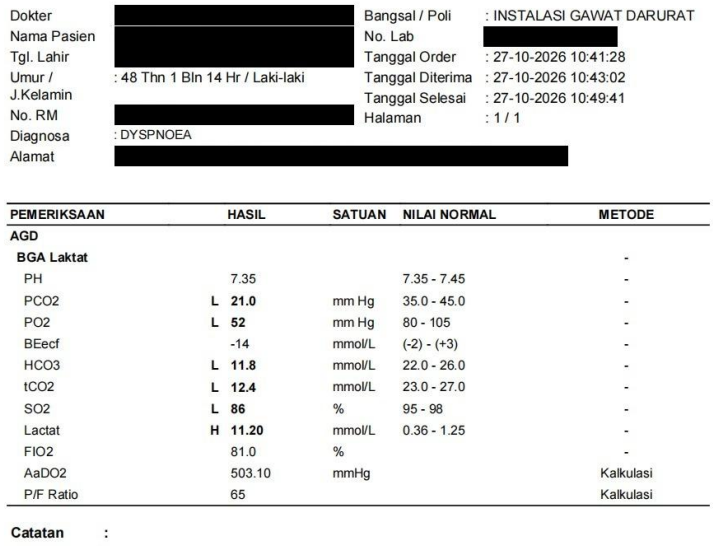
Parameter	Result	Reference†	Interpretation
Hematology			
Leukocytes; neutrophils	11.71 × 10^3/μL; 8.31 × 10^3/μL (70.9%)	3.8–10.6; 1.8–8	Mild neutrophilic leukocytosis
Lymphocytes; monocytes	2.12 (18.1%); 1.23 × 10^3/μL (10.5%)	0.9–5.2; 0.16–1	Relative lymphopenia; monocytosis
Eosinophils; NLR	0.02 × 10^3/μL (0.2%); 3.92	0.045–0.44; <3.13	Eosinopenia; elevated NLR (stress)
Hemoglobin / hematocrit; MCV	14.6 g/dL / 45.3%; 87.8 fL	13.2–17.3 / 40–52; 82–92	Normal, normocytic
Platelets; RDW	184 × 10^3/μL; 12.4%	150–440; 11.5–14.5	Normal
Clinical chemistry			
Urea (BUN)	17 mg/dL (≈7.9 mg/dL)	10–50	Normal; BUN:creatinine ≈4.3
Creatinine; eGFR	1.84 mg/dL; 46 mL/min/1.73 m^2	0.70–1.10; ≥90	Reduced (acute vs. chronic undetermined)
Sodium (glucose-corrected)	133 (136.2–137.8) mmol/L	135–147	Largely translocational
Potassium	2.8 mmol/L	3.5–5.0	Severe hypokalemia
Glucose; osmolality	300 mg/dL; ≈285 mOsm/kg	—; ≈275–295	Marked hyperglycemia; normal osmolality
hs-Troponin I (CMIA)	65.2 pg/mL	URL 39.2	≈1.7 × URL: myocardial injury

Notes: †Hospital laboratory ranges. BUN = urea/2.14; corrected Na = Na + factor × (glucose − 100)/100 (factor 1.6 or 2.4); osmolality = 2 × Na + glucose/18 + BUN/2.8.[31,42] CMIA, chemiluminescent microparticle immunoassay; NLR, neutrophil-to-lymphocyte ratio; URL, upper reference limit.

Arterial Blood Gas and Acid–Base Analysis

Arterial blood gas on high-concentration oxygen (Figure 1; Table 4) reported FiO_2 0.81, PaO_2/FiO_2 65 mmHg and an alveolar–arterial (A–a) gradient of 503.1 mmHg, consistent with the alveolar gas equation (≈499 mmHg). Mask FiO_2 varies with inspiratory flow, so the ratio is approximate, but shunt physiology is unequivocal.[15,16]

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Dokter	[redacted]	Bangsal / Poli	: INSTALASI GAWAT DARURAT
Nama Pasien	[redacted]	No. Lab	[redacted]
Tgl. Lahir	[redacted]	Tanggal Order	: 27-10-2026 10:41:28
Umur / J.Kelamin	: 48 Thn 1 Bln 14 Hr / Laki-laki	Tanggal Diterima	: 27-10-2026 10:43:02
		Tanggal Selesai	: 27-10-2026 10:49:41
No. RM	[redacted]	Halaman	: 1 / 1
Diagnosa	: DYSPNOEA		
Alamat	[redacted]		

PEMERIKSAAN		HASIL	SATUAN	NILAI NORMAL	METODE
AGD					
BGA Laktat					-
PH		7.35		7.35 - 7.45	-
PCO2	L	**21.0**	mm Hg	35.0 - 45.0	-
PO2	L	**52**	mm Hg	80 - 105	-
BEecf		-14	mmol/L	(-2) - (+3)	-
HCO3	L	**11.8**	mmol/L	22.0 - 26.0	-
tCO2	L	**12.4**	mmol/L	23.0 - 27.0	-
SO2	L	**86**	%	95 - 98	-
Lactat	H	**11.20**	mmol/L	0.36 - 1.25	-
FIO2		81.0	%		-
AaDO2		503.10	mmHg		Kalkulasi
P/F Ratio		65			Kalkulasi

Catatan :

Figure 1. Arterial blood gas and lactate report at 10:41 (identifiers masked): near-normal pH (7.35) despite HCO_3^- 11.8 mmol/L, base excess −14 mmol/L and lactate 11.20 mmol/L, with marked hypocapnia and severe hypoxemia on a reported FiO_2 of 0.81.

Arterial oxygen content (CaO_2) was ≈17.0 mL/dL versus ≈19.5 mL/dL expected—a reduction of only ≈13%, insufficient on its own to explain a lactate of 11.2 mmol/L unless delivery was further compromised by low cardiac output or regional hypoperfusion, or production and clearance were altered independently of oxygen delivery.[7,21]

Table 4. Arterial blood gas results and derived oxygenation indices

Parameter	Result	Reference	Interpretation
pH; $PaCO_2$	7.35; 21 mmHg	7.35–7.45; 35–45	"Pseudo-normal" pH; marked hypocapnia
PaO_2; SaO_2	52 mmHg; 86%	80–105; 95–98	Severe hypoxemia despite high FiO_2
HCO_3^-; base excess	11.8 mmol/L; −14 mmol/L	22–26; −2 to +3	Severe base deficit
Lactate	11.20 mmol/L	0.36–1.25	Severe hyperlactatemia
PaO_2/FiO_2; A–a gradient	≈64 mmHg; 503.1 mmHg	>400; —	Shunt / V/Q mismatch
Expected $PaCO_2$ (Winter's)	23.7–27.7 mmHg	—	Measured 21 mmHg → additional respiratory alkalosis
CaO_2 (estimated)	≈17.0 mL/dL	≈19.5 expected	≈13% reduction

Notes: $PAO_2 = FiO_2 \times (760 - 47) - PaCO_2/0.8 \approx 551$ mmHg; Winter's formula: expected $PaCO_2 = 1.5 \times HCO_3^- + 8 \pm 2$;[5] $CaO_2 = 1.34 \times Hb \times SaO_2 + 0.003 \times PaO_2$. V/Q, ventilation–perfusion.

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The stepwise analysis (Table 5) establishes a mixed disorder: metabolic acidosis with a $PaCO_2$ below the Winter's range, signifying an additional primary respiratory alkalosis, so that pH 7.35 is "pseudo-normal". The lactate rise explains most of the base deficit; the residual ≈3–4 mmol/L could not be characterized without chloride, albumin and ketones.[5,23,24]

Table 5. Stepwise physiological (bicarbonate-based) acid–base analysis

Step	Analysis	Result in this patient
1. Internal consistency	$[H^+] = 24 \times 21/11.8 \approx 42.7$ nmol/L (pH ≈7.37)	Consistent with measured pH
2. Primary process	pH 7.35; HCO_3^- 11.8 mmol/L; BE −14 mmol/L	Metabolic acidosis
3. Respiratory compensation	Winter's 23.7–27.7 mmHg vs. measured 21 mmHg	Concurrent primary respiratory alkalosis
4. Anion gap / delta ratio	$Na^+ - (Cl^- + HCO_3^-)$; albumin correction	Not calculable *[chloride, albumin to obtain]*
5. Lactate contribution	ΔLactate ≈10.2 vs. ΔHCO_3^- 12.2 mmol/L	Lactate explains ≈70–85% of the deficit
6. Residual deficit	≈3–4 mmol/L	Ketoacids, uremic anions or hyperchloremia possible
7. Conclusion	—	High-lactate metabolic acidosis + primary respiratory alkalosis

Notes: Physiological approach with validated compensation formulas.[5,23] BE, base excess.

Electrocardiography

The 09:39 electrocardiogram (Figure 2) showed sinus tachycardia ≈119 beats/min, PR 140 ms, QRS 124 ms (nonspecific intraventricular conduction delay), low-amplitude limb leads, poor R-wave progression and nonspecific ST–T changes, without confirmable new ST elevation meeting STEMI criteria. The machine QTc of 480 ms (Bazett) fell to 417–444 ms with alternative formulas (Table 6), so prolongation was probably overestimated, although severe hypokalemia still required continuous monitoring.[12,14,32,33,43]

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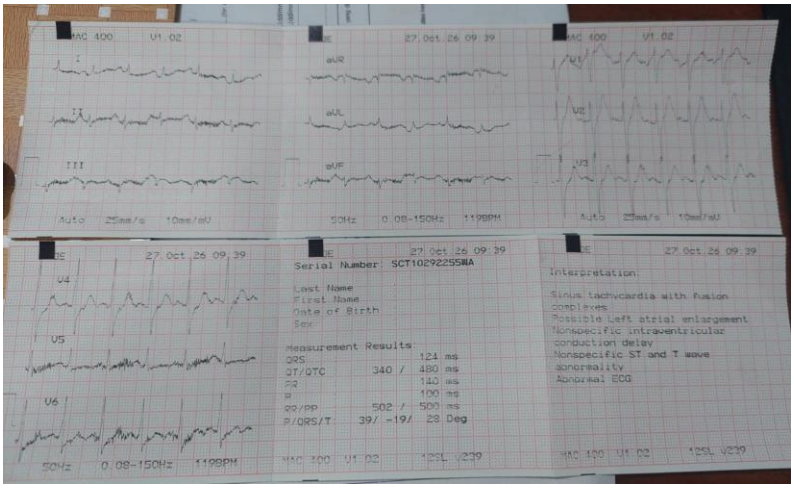


Figure 2. Twelve-lead electrocardiogram at 09:39 (25 mm/s, 10 mm/mV): sinus tachycardia ≈119 beats/min, QRS 124 ms, poor R-wave progression in V1–V3, low-amplitude limb leads and nonspecific ST–T changes; machine QT/QTc (Bazett) 340/480 ms.

Table 6. Electrocardiographic measurements and heart-rate correction of the QT interval

Parameter	Value	Comment
Rhythm / rate; axes P/QRS/T	Sinus tachycardia ≈119/min (RR 502 ms); 39°/−19°/28°	Catecholamine-driven; leftward but normal QRS axis
PR / QRS duration	140 ms / 124 ms	Possible LAE (machine); nonspecific IVCD
QTc Bazett ($QT/RR^{1/2}$)	480 ms (QT 340 ms)	Machine value; overcorrects at high rate
QTc Fridericia ($QT/RR^{1/3}$)	428 ms	Within normal limits
QTc Framingham / Hodges	417 ms / 444 ms	Normal / upper-normal

Notes: RR in seconds.[32,33] IVCD, intraventricular conduction delay; LAE, left atrial enlargement.

Chest Radiography

A portable supine anteroposterior radiograph after intubation (17:43; Figure 3) showed bilateral perihilar interstitial–alveolar opacities with right-sided predominance and an apparently enlarged cardiac silhouette. The asymmetric distribution broadens the differential to pneumonia, aspiration and right-sided edema from an eccentric mitral regurgitant jet, and radiography is less sensitive than lung ultrasound for cardiogenic edema.[17,44,45]

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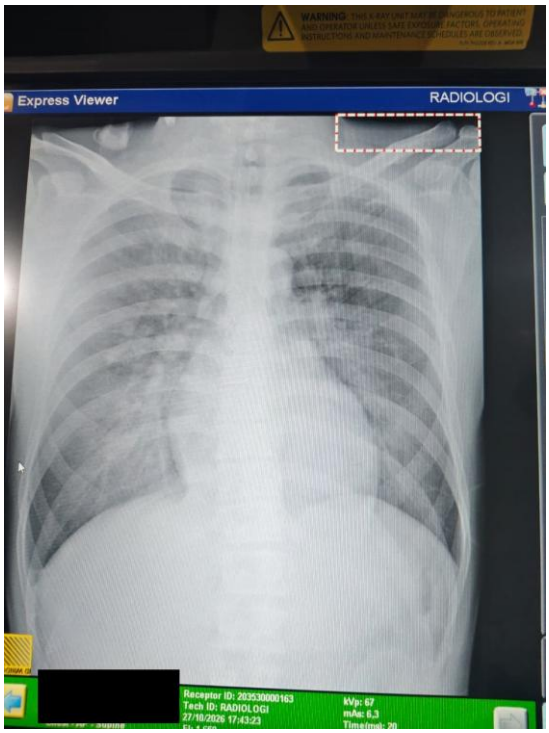


Figure 3. Portable anteroposterior supine chest radiograph at 17:43 (identifiers masked): bilateral perihilar opacities, more conspicuous on the right, with an apparently enlarged cardiac silhouette (limited assessment in the supine projection); official report pending.

Therapeutic Interventions

Initial management (Table 7) comprised mask oxygen, intravenous furosemide 40 mg followed by 5 mg/h, nitroglycerin 50 µg/min, insulin aspart 8 units, ranitidine 50 mg, lactated Ringer's at 8 drops/min and urinary catheterization. Oxygenation remained critically impaired (SpO_2 81%, respiratory rate 43/min at 13:20; Figure 4a), and after endotracheal intubation blood pressure fell to 101/55 mmHg (Figure 4b).[13,30,34,46]

Table 7. Treatment administered and evidence-based benchmark

Intervention	Dose / details	Evidence-based comment
Oxygen via NRM	15 L/min	Delivered FiO_2 falls at high flow; consider early CPAP/NIV or HFNC when RR >25/min and SpO_2 <90%[13,28,47]
Nitroglycerin IV	50 µg/min	In SCAPE, ≥100 µg/min ± boluses gives faster afterload reduction; down-titrate as SBP approaches 100 mmHg[36,37]
Furosemide IV	40 mg + 5 mg/h	1–2 × home dose if on oral diuretics; urinary Na^+ at 2 h, urine output

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		at 6 h[34,48]
Insulin aspart	8 U *[route to confirm]*	Shifts K^+ into cells; with K^+ 2.8 mmol/L concurrent IV repletion required[34,49]
Ranitidine IV	50 mg	Stress-ulcer prophylaxis once ventilated
Lactated Ringer's	8 drops/min (≈24–32 mL/h)	Lactate load <1 mmol/h; cannot explain measured lactate[40,50]
Urinary catheter	—	Timed output needed for diuretic response and KDIGO criteria[41]
Potassium / magnesium	Not documented *[to confirm]*	High-normal K^+ (≈4.0–5.0 mmol/L) lowers arrhythmic risk[51,52]
Intubation + IMV	SIMV-PC (Table 8)	Peri-intubation instability common; lung-protective settings[6,30]

Notes: Drop rate assumes a 15–20 drops/mL set; lactated Ringer's contains 28 mmol/L lactate. HFNC, high-flow nasal cannula; IMV, invasive mechanical ventilation; KDIGO, Kidney Disease: Improving Global Outcomes; NIV, non-invasive ventilation.



(a) 13:20, before intubation *(b) 15:12, after intubation*

Figure 4. Bedside monitor. (a) On a non-rebreathing mask: heart rate 121/min, SpO_2 81%, respiratory rate 43/min, blood pressure 117/76 mmHg (MAP 87). (b) After intubation: heart rate 94/min, SpO_2 98%, respiratory rate 26/min, blood pressure 101/55 mmHg (MAP 73).

Mechanical Ventilation Data

Two ventilator displays were photographed (Figure 5; Table 8). On SIMV-PC with only 10 cmH_2O above PEEP, the first showed a total rate of 43/min, exhaled tidal volume 702 mL (≈10.6 mL/kg predicted body weight) and minute ventilation 20.7 L/min, generated largely by vigorous patient effort. The second (16:06–16:11) showed a total rate of 38/min, tidal volume 344 mL (≈5.2 mL/kg), estimated driving pressure 12.4 cmH_2O, end-tidal CO_2 22 mmHg and SpO_2 93% on FiO_2 0.85 (SpO_2/FiO_2 ≈109).[6,10,15,16,39]

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(a) First display (time not recorded)



(b) Second display, 16:06–16:11

Figure 5. Ventilator displays after intubation. (a) SIMV-PC (rate 12, Pi 10, PS 8, PEEP 6 cmH_2O, O_2 80%): total rate 43/min, VTe 702 mL, VE 20.7 L/min. (b) SIMV-PC (ΔPC 10, PEEP 5, PS 8 cmH_2O, FiO_2 0.85): total rate 38/min, VT 344 mL, VE 14.5 L/min, $EtCO_2$ 22 mmHg, SpO_2 93%.

Table 8. Ventilator settings and measured parameters after intubation

Parameter	Display 1	Display 2 (16:06–16:11)	Comment
ΔP above PEEP / PS; FiO_2	10 / 8 cmH_2O; 0.80	10 / 8 cmH_2O; 0.85	Low set pressures; high O_2 requirement
PEEP set / measured	6.0 / 5.3 cmH_2O	5.0 / 6.4 cmH_2O	Low for FiO_2 ≥0.80
Total respiratory rate	43/min	38/min	High respiratory drive
Tidal volume (per kg PBW)	VTe 702 mL (≈10.6 mL/kg)	VT 344 mL (≈5.2 mL/kg)	Protective target 4–8 mL/kg
Minute ventilation	20.7 L/min	14.5 L/min	Compensatory hyperventilation
Peak / plateau; driving pressure	18 / — ; ≈12.7 (dynamic) cmH_2O	15.6 / 15.3; 12.4 (est.) cmH_2O	Plateau ≤30; driving <15 cmH_2O
Compliance (estimated)	≈55 mL/cmH_2O	≈28 mL/cmH_2O	Effort confounds estimates
$EtCO_2$ / RSBI / SpO_2	—	22 mmHg / 72 / 93%	Persistent hypocapnia and hypoxemia

Notes: PBW = 50 + 0.91 × (170 − 152.4) = 66.0 kg (65 and 70 kg were entered).[6] Driving pressure = plateau − PEEP. PS, pressure support; RSBI, rapid shallow breathing index.

Hemodynamic Trajectory

Mean arterial pressure fell from ≈118 mmHg at presentation to 87 mmHg before and 73 mmHg after intubation (Table 9). This may reflect vasodilators, sedation, positive pressure and

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relief of sympathetic drive, but may also herald evolving cardiogenic shock, mandating repeated perfusion assessment and SCAI restaging.[9,46]

Table 9. Serial vital signs and respiratory support

Time	BP (mmHg)	MAP	HR (/min)	Shock index	RR (/min)	SpO_2	Support
Arrival	148/103	≈118	120	0.81	41	78% → 87%	Room air → NRM
13:14–13:20	117/76	87	121	1.03	43	81%	NRM (pre-intubation)
15:11–15:12	101/55	73	94	0.93	26	98%	IMV, FiO_2 ≈0.80
16:06–16:11	*[n/a]*	—	—	—	38 (total)	93%	IMV, FiO_2 0.85

Notes: Shock index = heart rate/systolic blood pressure.

Urine Output

The collection bag contained <100 mL of concentrated urine (Figure 6), but without a recorded interval oliguria cannot be graded by KDIGO criteria; absent initial output after a loop diuretic with reduced eGFR raises the possibility of type 1 cardiorenal syndrome and inadequate diuretic response.[34,41,53]



Figure 6. Urine-collection bag with <100 mL of dark, concentrated urine after catheterization; collection interval not recorded.

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Diagnostic Assessment and Working Diagnoses

Working diagnoses are summarized in Table 10. Diagnostic challenges included the absence of echocardiography, natriuretic peptide and serial troponin, reliance on photographed displays, and uncertain baseline renal function and medications.[13,14]

Table 10. Problem list, supporting findings and data required for confirmation

No.	Working diagnosis	Supporting findings	Data required
1	AHRF requiring invasive ventilation	PaO_2/FiO_2 ≈64; A–a ≈500 mmHg	Post-intubation ABG; lung ultrasound
2	AHF, acute pulmonary edema ("warm–wet", hypertensive), presumed ischemic	Prior STEMI/PCI and AHF; crackles; BP 148/103 mmHg; perihilar opacities	Echocardiography; NT-proBNP
3	Severe hyperlactatemia, probable mixed type A/B	Lactate 11.20 mmol/L; BE −14 mmol/L	Serial lactate; $ScvO_2$; CO_2 gap
4	Metabolic acidosis + primary respiratory alkalosis	$PaCO_2$ below Winter's range; pH 7.35	Chloride, albumin, ketones
5	Myocardial injury: type 1 vs. type 2 MI vs. non-ischemic	hs-TnI ≈1.7 × URL; ST–T changes	Serial hs-TnI; angiography
6	AKI (cardiorenal type 1) vs. CKD; possible oliguria	Creatinine 1.84 mg/dL; eGFR 46	Baseline creatinine; timed output
7	Severe hypokalemia	K^+ 2.8 mmol/L	Repeat K^+; magnesium
8	Marked hyperglycemia (stress vs. diabetes)	Glucose 300 mg/dL	HbA1c; ketones
9	Mild translocational hyponatremia	Corrected Na^+ 136–138 mmol/L	Repeat sodium
10	Leukocytosis with eosinopenia: stress vs. infection	WBC 11.71 × 10^3/μL; afebrile	CRP/procalcitonin; cultures

Notes: AKI, acute kidney injury; CKD, chronic kidney disease; NT-proBNP, N-terminal pro-B-type natriuretic peptide; $ScvO_2$, central venous oxygen saturation.

DISCUSSION

Clinical Synthesis

The unifying diagnosis is AHF of the acute pulmonary edema phenotype on a presumed ischemic substrate, with an abrupt, hypertensive and sympathetically driven presentation complicated by severe AHRF, hyperlactatemia, a mixed acid–base disorder, myocardial injury, renal dysfunction, hypokalemia and hyperglycemia. Its most instructive feature is the discordance between reassuring blood pressure and pH and the extreme lactate; precipitants—recurrent ACS or

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stent thrombosis, ischemic mitral regurgitation, arrhythmia, hypertension, infection and non-adherence—should be sought in parallel with stabilization.[1,12,13,36]

Acute Hypoxemic Respiratory Failure: Cardiogenic Edema, ARDS or Both?

A PaO_2/FiO_2 of ≈64 mmHg with an A–a gradient of ≈500 mmHg indicates a large shunt. With bilateral opacities these values meet ARDS oxygenation and imaging criteria, but the definition requires that failure is not fully explained by cardiac failure; the abrupt onset, hypertension, prior AHF and congestive signs make hydrostatic edema the likely primary mechanism, although a superimposed permeability component cannot be excluded. Although mask FiO_2 is overestimated at >40 breaths/min, the post-intubation SpO_2/FiO_2 of ≈109 confirms persistent severe hypoxemia.[15,16]

Lung ultrasound has a pooled sensitivity of 0.88 versus 0.73 for radiography in cardiogenic edema, and its integration with clinical assessment raised AHF diagnostic accuracy to an area under the curve of 0.95. The right-sided predominance deserves attention: right upper lobe edema occurs with eccentric mitral regurgitant jets, and in acute regurgitation from chordal rupture unilateral, always right-sided edema occurred in half of cases and was often mistaken for pneumonia. Papillary-muscle dysfunction must therefore be excluded by urgent echocardiography, because the murmur may be inaudible (Table 11).[17,35,44,45,54]

Table 11. Differential diagnosis of AHRF with bilateral opacities in this patient

Condition	Supporting features	Against / uncertainty	Key discriminating test
Hydrostatic cardiogenic edema (APE/SCAPE)	Abrupt onset; hypertension; prior MI and AHF	Right-sided asymmetry	Echocardiography; lung ultrasound; NT-proBNP[17,35]
Acute ischemic mitral regurgitation	Prior MI; flash edema; right-predominant opacities	No audible murmur (does not exclude)	Urgent TTE/TEE[44,45]
ACS / stent thrombosis	Prior STEMI/PCI; hs-TnI elevation; ST–T changes	No chest pain; single troponin	Serial hs-TnI; angiography[12,14]
Pneumonia / aspiration	Asymmetric opacities; neutrophilia	Afebrile; stress eosinopenia	CRP/procalcitonin; cultures[15]
ARDS (permeability edema)	PaO_2/FiO_2 ≈64; bilateral opacities	Cardiac cause likely explains findings	Echocardiography; response to decongestion[15,16]
Pulmonary embolism	Tachycardia; hypoxemia; hypocapnia	Opacities and hypertension atypical	Echocardiography (RV); CT angiography

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Notes: APE, acute pulmonary edema; TTE/TEE, transthoracic/transesophageal echocardiography; RV, right ventricle.

Severe Hyperlactatemia: Dissecting the Mechanisms

A lactate of 11.2 mmol/L is extreme: in AHF without hypoperfusion even ≥2 mmol/L independently predicts death (hazard ratio 1.24), in cardiogenic shock ≥5 mmol/L raises 30-day mortality within each SCAI stage, and lactate, previous infarction, ACS etiology, ejection fraction and eGFR form the CardShock score—several elements of which this patient displayed.[2,3,18]

With oxygen content reduced by only ≈13%, hypoxemia could cause dysoxia only if cardiac output or regional flow were also compromised (Table 12). Warm, hypertensive features do not exclude low output, because vasoconstriction can preserve pressure while splanchnic and muscle perfusion fall—the SCAI pattern of normotensive hypoperfusion—so stroke volume, $ScvO_2$, the venous-to-arterial CO_2 gap and capillary refill should be prioritized.[3,7,9]

Much of the lactate was probably aerobic. Catecholamines acting through β_2-receptors accelerate glycolysis and Na^+/K^+-ATPase, so pyruvate production exceeds oxidation; the same mechanism drives potassium into cells and raises glucose, so the triad of hyperlactatemia, hypokalemia and hyperglycemia forms a coherent catecholamine signature consistent with SCAPE. Alkalemia (stimulating phosphofructokinase), respiratory-muscle work at >40 breaths/min, and reduced hepatic and renal clearance probably contributed, and metformin exposure should be verified; pulmonary lactate release has been shown in acute lung injury rather than cardiogenic edema.[7,8,21,36,55–57]

The lactated Ringer's infusion (<1 mmol lactate/h) cannot explain the value, as even 1 L over one hour does not meaningfully raise lactate. The residual base deficit may reflect ketoacids, uremic anions or hyperchloremia, and euglycemic ketoacidosis should be excluded if a sodium–glucose co-transporter 2 (SGLT2) inhibitor was used. Because a single value cannot discriminate these mechanisms, the trajectory is decisive: rapid clearance favors adrenergic and respiratory causes, whereas persistent lactate indicates hypoperfusion; complete clearance by 8 hours predicts survival in cardiogenic shock.[11,19,20,40,50,58]

Table 12. Probable mechanisms contributing to hyperlactatemia in this patient

Mechanism	Type	Supporting features	Uncertainty / data required

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Reduced arterial O_2 content	A	SaO_2 86%; CaO_2 ≈17.0 mL/dL	Only ≈13% reduction—insufficient alone[7]
Low output / normotensive hypoperfusion	A	Prior MI and AHF; tachycardia; later BP fall	Echo (LVEF, LVOT VTI), $ScvO_2$, CO_2 gap[3,9]
Catecholamine-driven aerobic glycolysis	B	Hypertension; hypokalemia; hyperglycemia	Inferred; catecholamines not measured[8]
Alkalosis-stimulated glycolysis	B	$PaCO_2$ 21 mmHg	Magnitude uncertain[7,21]
Respiratory-muscle work	A/B	RR 41–43/min; VE 20.7 L/min	Should fall with ventilatory support[21]
Pulmonary lactate production	B	Severe lung edema	Shown mainly in acute lung injury[56]
Reduced hepatic/renal clearance	B	eGFR 46; possible hepatic congestion	Liver tests, INR[7]
Drugs (metformin, β-agonists)	B	Medication history unknown	Verify medication history[21,57]
Lactated Ringer's infusion	—	8 drops/min	Negligible (<1 mmol/h)[40,50]

Notes: Type A, impaired oxygen delivery; type B, without overt circulatory failure. LVOT VTI, left ventricular outflow tract velocity–time integral.

Mixed Acid–Base Disorder: the Pseudo-normal pH

A pH of 7.35 superficially suggests a mild disorder, yet a base excess of −14 mmol/L reveals severe metabolic acidosis and a $PaCO_2$ of 21 mmHg lies below the compensatory range, identifying two opposing primary disorders. The respiratory alkalosis is driven by pulmonary vagal receptor stimulation, hypoxemia, dyspnea and the acidosis itself; in heart failure, $PaCO_2$ correlates inversely with wedge pressure ($r = -0.80$), and respiratory alkalosis was the commonest disorder in high-risk KorHF patients.[5,22,23,59]

Because lactate ≥5 mmol/L with pH <7.2 carries the highest mortality in cardiogenic shock, the protective alkalosis is fragile: a ventilator delivering only 12 breaths/min at 6 mL/kg (≈4.8 L/min) in a deeply sedated patient would raise $PaCO_2$ steeply. Minute ventilation should therefore initially match the pre-intubation level, guided by early blood gas, since end-tidal CO_2 may underestimate $PaCO_2$; chloride and albumin are needed to calculate the anion gap and delta ratio.[3,6,24]

Myocardial Injury, Electrocardiogram and the Post-infarction Substrate

Troponin ≈1.7-fold above the upper reference limit establishes injury, but infarction requires a rise and/or fall with evidence of ischemia; tachycardia, hypoxemia, wall stress and renal dysfunction can cause type 2 infarction or non-ischemic injury, whereas type 1 infarction or stent thrombosis remains possible. Because the 2023 ESC guidelines classify AHF due to ongoing

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ischemia as a very-high-risk feature warranting immediate invasive strategy, serial troponin, echocardiographic wall-motion assessment and early angiography are required if ischemia is supported.[12,14,60]

Poor right precordial R-wave progression may represent prior anterior scar, and nonspecific ST–T changes may reflect strain, ischemia or hypokalemia, which produces electrocardiographic abnormalities in about 40% of emergency patients with potassium <3.5 mmol/L. Fridericia and Framingham formulas are more rate-independent than Bazett, so the recalculated QTc suggests no marked prolongation.[32,33,43]

Electrolyte and Metabolic Derangements

Hypokalemia was probably multifactorial—catecholamine- and alkalosis-induced shift, prior diuretic losses and secondary hyperaldosteronism—and furosemide with insulin could lower potassium further, so repletion should precede or accompany these interventions. After myocardial infarction, potassium <3.5 mmol/L is associated with ventricular arrhythmias and mortality, the association is U-shaped in heart failure, and the POTCAST trial showed that targeting 4.5–5.0 mmol/L reduced arrhythmic events, hospitalization and death (hazard ratio 0.76).[34,48,49,51,52]

Glucose of 300 mg/dL may represent stress hyperglycemia or diabetes; the stress hyperglycemia ratio discriminates better than absolute glucose, and its highest category carried a relative mortality risk of 1.67 among 80,010 cardiovascular patients, so glycated hemoglobin should be measured. Sodium was largely translocational, though hyponatremia in AHF predicts worsening renal function, and leukocytosis with eosinopenia suggests a stress leukogram without excluding infection.[15,31,38,42,55,61,62]

Renal Dysfunction, Oliguria and Decongestion

Creatinine 1.84 mg/dL may represent type 1 cardiorenal syndrome, chronic kidney disease or acute-on-chronic dysfunction; the low urea nitrogen-to-creatinine ratio argues against a prerenal mechanism. The Heart Failure Association recommends assessing response by spot urinary sodium ≥50–70 mmol/L at 2 hours and/or urine output ≥100–150 mL/h during 6 hours, doubling the dose

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and adding sequential nephron blockade if inadequate; bolus and infusion are comparable, and modest creatinine rises during effective decongestion should not prompt de-escalation.[13,34,41,48,53]

Respiratory Support Strategy

Non-invasive ventilation should be considered when respiratory rate exceeds 25/min and SpO_2 is <90%: a Cochrane review of 24 trials found that it may reduce mortality (risk ratio 0.65) and probably reduces intubation (risk ratio 0.49), while 3CPO confirmed faster physiological improvement; high-flow nasal cannula also improved lactate clearance and blood gases in AHF. Here, the patient remained on a mask with SpO_2 81% almost three hours after the blood gas, and when non-invasive support is unavailable or fails, timely intubation is preferable to delayed intubation.[10,13,28,29,47,63]

In the INTUBE study, 45.2% of critically ill patients had a major peri-intubation event, chiefly cardiovascular instability, predicted by tachycardia, lower systolic pressure, lower SpO_2/FiO_2 and propofol; the post-intubation pressure fall here underlines the need for positive-pressure preoxygenation, cautious induction, ready vasopressors and temporary vasodilator reduction.[30,46]

After intubation, tidal volumes of ≈10.6 mL/kg generated with only 10 cmH_2O imply large negative pleural swings that increase transpulmonary pressure, overdistension, pendelluft and capillary transmural pressure, underpinning P-SILI. Lower tidal volumes (6 mL/kg; plateau ≤30 cmH_2O) reduced ARDS mortality from 39.8% to 31.0% and, in patients without ARDS, lung injury (risk ratio 0.33) and mortality (risk ratio 0.64); options included volume-targeted ventilation at 6–8 mL/kg, drive-titrated sedation and, if needed, short-term neuromuscular blockade. PEEP of 5–6 cmH_2O was low for FiO_2 0.80–0.85 (the ARDS Network table pairs FiO_2 0.8 with PEEP 14 cmH_2O), so stepwise titration would have been reasonable.[6,10,16,39,64]

Pharmacological Hemodynamic Management

High-dose nitroglycerin (boluses or ≥100 μg/min) with non-invasive ventilation is advocated in SCAPE, and a 2025 Indonesian meta-analysis found that it reduced the need for mechanical ventilation (risk ratio 0.31) and increased 6-hour symptom resolution (risk ratio 3.88)

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without more hypotension; 50 µg/min was therefore conservative. As pressure fell, dosing required reassessment, and persistent hypoperfusion should be managed within the SCAI framework with norepinephrine, dobutamine for documented low output and consideration of mechanical support; once stable, SGLT2 inhibitors and rapid up-titration of guideline-directed therapy are recommended.[9,11,13,36,37]

SCAI Staging and Metabolic–Hemodynamic Discordance

Hypertensive and warm at presentation, the patient would ordinarily be stage A or B (Table 13), yet SCAI also defines hypoperfusion biochemically, and a lactate of 11.2 mmol/L with a base deficit of 14 mmol/L is typical of advanced stages. Because the respiratory alkalosis kept pH above 7.2, pH-based modifiers would have underestimated severity. We propose that such patients be regarded as having a metabolic–hemodynamic discordance phenotype, investigated as intensively as overt shock until hypoperfusion is excluded.[3,9,25–27]

Table 13. Application of SCAI SHOCK classification parameters to this patient

Domain	Finding	Interpretation
Blood pressure; heart rate	148/103 mmHg (MAP ≈118); 120/min	Stage A/B by pressure alone; tachycardia
Clinical perfusion	Warm extremities; conscious	No clinical hypoperfusion
Lactate; base excess / pH	11.20 mmol/L; −14 mmol/L / 7.35	Biochemical hypoperfusion of advanced-stage magnitude; pH masked
Renal markers	Creatinine 1.84 mg/dL; low urine output	Possible end-organ dysfunction
Early trajectory	BP 117/76 → 101/55 mmHg after intubation	Requires repeated restaging
Overall	—	Metabolic–hemodynamic discordance: manage as at least stage C until hypoperfusion is excluded

Notes: Based on the SCAI consensus statement, its 2022 update and published risk modifiers.[3,9,25]

Literature Review Summary

Table 14 summarizes the principal evidence underpinning this analysis.[2,22,28,37]

Table 14. Summary of key evidence relevant to this case

Study (design)	Population	Key findings	Relevance
Zymliński 2018 (cohort)[2]	237 AHF, "warm"	Lactate ≥2 mmol/L in 43%; 1-year mortality 36% vs. 21%	Prognostic lactate
Jentzer 2022 (cohort)[3]	1814 CS	Lactate ≥5 or pH <7.2 raised mortality	Risk modifiers

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		within SCAI stages	
Marbach 2022 (DOREMI)[19]	CS on inotropes	Complete lactate clearance predicted survival from 8 h	Serial lactate
Harjola 2015 (CardShock)[18]	219 CS	Lactate, prior MI, ACS, LVEF, eGFR in score (AUC 0.85)	Risk elements
Park 2015 (KorHF)[22]	1982 AHF	Respiratory alkalosis commonest; acidosis HR 1.93	Mixed disorders
Lorenzi-Filho 2002[59]	Chronic HF	$PaCO_2$ vs. wedge pressure r = −0.80	Hypocapnia
Berbenetz 2019 (Cochrane)[28]	24 RCTs; 2664	NIV: mortality RR 0.65; intubation RR 0.49	Early NIV
Gray 2008 (3CPO)[63]	1069 APE	No mortality difference; faster improvement	NIV benefit
Pramudyo 2025 (SR/MA)[37]	185 SCAPE	High-dose NTG: ventilation RR 0.31; resolution RR 3.88	Vasodilator dose
Serpa Neto 2012 (MA)[64]	2822 without ARDS	Lower VT: lung injury RR 0.33; mortality RR 0.64	Protective VT
Russotto 2021 (INTUBE)[30]	2964	Major peri-intubation events 45.2%	Intubation risk
Jøns 2025 (POTCAST)[51]	1200 with ICD	K^+ 4.5–5.0 mmol/L: HR 0.76	Potassium target
Esdaile 2024 (MA)[38]	80,010	Highest stress hyperglycemia ratio: RR 1.67	Hyperglycemia
Maw 2019 (MA)[17]	1827	Lung ultrasound sensitivity 0.88 vs. 0.73	Ultrasound
Nishimura 2022 (cohort)[45]	38 acute MR	Right-sided unilateral edema in 50%	Asymmetry
Vandenberk 2016[32]	6609 ECGs	Fridericia/Framingham best; Bazett worst	QTc

Notes: AUC, area under the curve; CS, cardiogenic shock; HR, hazard ratio; MA, meta-analysis; MR, mitral regurgitation; NTG, nitroglycerin; RCT, randomized controlled trial; RR, risk ratio; VT, tidal volume.

Practical Bedside Approach

We propose a pragmatic checklist (Table 15) for settings without immediate invasive monitoring, relying on routinely obtainable data.[5,9,13]

Table 15. Proposed bedside checklist for AHF with AHRF and hyperlactatemia

Step	Action and rationale
1. Phenotype and perfusion	BP, MAP, mentation, capillary refill, urine output; SCAI stage including lactate[9]
2. Blood gas analysis	Compensation formulas, base excess and lactate; chloride and albumin for the anion gap[5,23]
3. Oxygen delivery	CaO_2; echocardiography (LVEF, LVOT VTI); $ScvO_2$ and CO_2 gap if a central line exists[7]
4. Serial lactate	Repeat within the first hours and track clearance[19]
5. Respiratory support	CPAP/NIV or HFNC when RR >25/min and SpO_2 <90%; avoid prolonged mask failure[28,47]
6. Vasodilators	High-dose nitroglycerin in hypertensive presentations; reduce as SBP approaches 100 mmHg[36,37]
7. Electrolytes first	Replete K^+ to high-normal and check Mg^{2+} before or with insulin and diuretics[51,52]
8. Diuretic response	Spot urinary sodium at 2 h and urine output at 6 h; escalate if inadequate[34]
9. Coronary evaluation	Serial hs-troponin, ECG comparison, wall motion; immediate angiography if ongoing

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	ischemia[12,14]
10. Imaging	Lung ultrasound plus focused echocardiography (mitral valve, RV)[17,45]
11. Ventilation	VT 6–8 mL/kg PBW; monitor drive; match minute ventilation to prior compensation; titrate PEEP[6,10]
12. Metabolic interpretation	HbA1c, ketones if on SGLT2 inhibitor, glucose-corrected sodium, Fridericia QTc[31,32,38,58]

Notes: CPAP, continuous positive airway pressure; PBW, predicted body weight.

Strengths and Limitations

The strengths of this report include near-real-time documentation of vital signs, blood gases, electrocardiography and ventilator displays across the first hours of care, enabling quantitative physiological reconstruction, and the systematic application of current guidelines to each clinical problem.[4]

CONCLUSIONS AND RECOMMENDATIONS

Conclusions

AHF of the acute pulmonary edema phenotype after myocardial infarction can present with extreme hypoxemia, severe hyperlactatemia and a mixed metabolic acidosis–respiratory alkalosis while blood pressure is elevated and pH near normal. Systematic analysis showed that the pH concealed a base deficit of 14 mmol/L, that hypoxemia alone could not account for a lactate of 11.2 mmol/L, and that coexisting hypokalemia and hyperglycemia pointed to a catecholamine-driven component.[5,7,8]

Severe hyperlactatemia with preserved blood pressure should be regarded as a high-risk, potentially pre-shock metabolic–hemodynamic discordance, requiring prompt clarification of cardiac output and perfusion, serial lactate, evaluation for ACS and mechanical complications, careful electrolyte management and lung-protective ventilation that controls excessive drive.[3,9,10,12]

Recommendations

For clinicians. Interpret every blood gas in AHF with compensation formulas, base excess, lactate, chloride and albumin; repeat lactate within hours; correct potassium toward the high-normal range before or with insulin and loop diuretics; recalculate QTc with Fridericia's formula; and

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normalize tidal volume to predicted body weight, matching minute ventilation to pre-intubation compensation.[5,6,19,32,51]

For institutions. Establish AHF pathways with early CPAP/NIV or high-flow nasal cannula, high-dose nitrate titration, response-guided diuretic algorithms, electrolyte-repletion protocols, a peri-intubation safety bundle, point-of-care ultrasound training and rapid access to echocardiography and coronary angiography.[28,30,34,35,37]

For post-discharge care. Initiate and rapidly up-titrate guideline-directed therapy, including SGLT2 inhibitors, with early follow-up.[11] *For research.* Prospective Indonesian and regional registries should characterize lactate trajectories in acute pulmonary edema and test lactate and base excess as explicit SCAI modifiers.[2,3,9] *For this manuscript.* Echocardiography, serial biochemistry, coronary evaluation and outcome data should be completed before submission.[4]

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APPENDIX A. CLINICAL PHOTOGRAPHIC DOCUMENTATION

Original laboratory reports (identifiers masked) supporting Table 3.

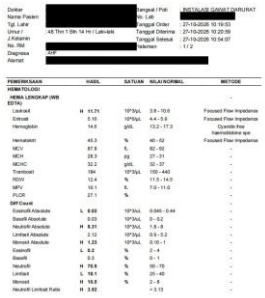


Figure A1. Complete blood count with differential (sampled 10:19, reported 10:54).



Figure A2. Renal function and electrolytes: creatinine 1.84 mg/dL, eGFR 46, Na^+ 133, K^+ 2.8 mmol/L.

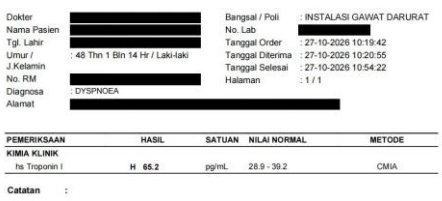


Figure A3. High-sensitivity troponin I (CMIA) 65.2 pg/mL (reference 28.9–39.2 pg/mL).

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