



The Critical Role of Insulin Therapy in the Management of Stress Hyperglycaemia in Sepsis Secondary to Community-Acquired Pneumonia with Impending Respiratory Failure and Acute Severe Asthma Exacerbation: A Case Report from a Secondary-Level Indonesian Hospital

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ABSTRACT

Introduction. Stress hyperglycaemia is a common but frequently overlooked metabolic derangement in acutely ill patients without diabetes, arising from catecholamine and glucocorticoid surges, cytokine-mediated insulin resistance, and accelerated hepatic gluconeogenesis. It independently predicts adverse outcomes in both sepsis and community-acquired pneumonia (CAP). Recognition becomes particularly challenging when severe dyspnoea dominates, leucocyte count is normal, and chest radiography is non-specific—a combination common when acute asthma exacerbation and lower respiratory tract infection coexist.

Case Illustration. A 58-year-old Indonesian woman with lifelong asthma, hypertension, and remote cardiovascular and cerebrovascular history, but no known diabetes, presented with three days of progressive dyspnoea, productive cough, fever,

nausea, and epigastric pain. She was orthopnoeic, agitated, speaking in single words, with respiratory rate 62/min, heart rate 116/min, oxygen saturation 87% on room air, and a normal leucocyte count of $5.1 \times 10^3/\mu\text{L}$. Random blood glucose was markedly elevated at 248 mg/dL. Chest radiography showed a bronchitic pattern, cardiomegaly without congestion, and aortic atherosclerosis. She was diagnosed with sepsis secondary to CAP with impending respiratory failure, acute severe asthma with life-threatening features, and newly detected stress hyperglycaemia. Management comprised high-flow oxygen, empirical ceftriaxone and levofloxacin, systemic corticosteroid, nebulised bronchodilator and corticosteroid, intravenous magnesium sulphate, and basal insulin glargine 8 units subcutaneously at night. Fasting plasma glucose the following morning was 150 mg/dL. She improved steadily without intubation and was discharged on day four speaking in full sentences, saturating 95% on room air.

Discussion. This case demonstrates the convergence of three hyperglycaemic mechanisms: endogenous stress response, high-dose corticosteroid, and β_2 -agonist therapy. Hyperglycaemia impairs neutrophil function, elevates airway surface glucose promoting bacterial proliferation, amplifies cytokine release, and contributes to respiratory muscle dysfunction—each aggravating dyspnoea. Insulin was the only rational glucose-lowering agent: rapidly titratable, anti-inflammatory, and avoiding organ-specific hazards in sepsis.

Conclusion. Random blood glucose should be measured in every patient presenting with severe dyspnoea, regardless of diabetes history. Early recognition and prompt basal insulin, delivered alongside guideline-directed therapy in a secondary-level hospital

without intensive care facilities, accompanied rapid clinical recovery and avoided invasive ventilation. Glycaemic assessment deserves integration into emergency evaluation of the severely breathless patient.

Keywords: stress hyperglycaemia; insulin therapy; sepsis; community-acquired pneumonia; acute severe asthma

INTRODUCTION

Background

Acute dyspnoea is among the most frequent and most time-critical presentations to the emergency department, and in adult patients it is dominated by three overlapping aetiologies: lower respiratory tract infection, obstructive airway disease and cardiac decompensation. When more than one of these processes coexists in the same patient, the clinical picture becomes considerably more difficult to disentangle, and the physician is required to make simultaneous decisions about airway support, antimicrobial therapy and bronchodilation on the basis of incomplete data. Community-acquired pneumonia (CAP) remains a leading cause of hospitalisation and death worldwide, and current guidance from the American Thoracic Society and the Infectious Diseases Society of America emphasises early severity stratification, prompt empirical antimicrobial therapy and reassessment as microbiological information becomes available.^{3,4}

Sepsis, redefined by the Third International Consensus Definitions (Sepsis-3) as life-threatening organ dysfunction caused by a dysregulated host response to infection, is operationalised by an acute rise of at least two points in the Sequential Organ Failure Assessment (SOFA) score, with the bedside quick SOFA (qSOFA) offered as a prompt for closer evaluation outside the intensive care unit. A crucial and frequently misunderstood consequence of this framework is that the systemic inflammatory response syndrome criteria — including leucocytosis — are no longer required to establish the diagnosis. A patient with a normal white cell count may nevertheless be septic, and the Surviving Sepsis Campaign accordingly recommends immediate administration of empirical antimicrobials when the probability of sepsis is high.^{1,2}

Asthma adds a further layer of complexity. The Global Initiative for Asthma (GINA) classifies an exacerbation as severe when the patient speaks in words rather than sentences, sits hunched forward, is agitated, has a respiratory rate above 30 breaths per minute, uses accessory muscles and desaturates below 90% on room air; a silent chest, drowsiness or confusion signal a life-threatening exacerbation requiring immediate escalation. In such patients the physical signs of

pneumonia may be entirely masked by bronchospasm, and the chest radiograph — which in uncomplicated asthma is typically normal or shows only hyperinflation and coarse peribronchial markings — rarely resolves the diagnostic uncertainty.⁶

Against this background, one metabolic variable is routinely available, inexpensive and yet commonly overlooked: blood glucose. Stress hyperglycaemia — an acute elevation of blood glucose in a patient without established diabetes mellitus — arises from the interaction of catecholamines, cortisol, growth hormone and glucagon with inflammatory cytokines such as interleukin-6 and tumour necrosis factor- α , producing accelerated hepatic gluconeogenesis and glycogenolysis together with peripheral insulin resistance. While a modest rise in glucose may be an adaptive response that supplies substrate to immune and neural tissue, sustained or excessive hyperglycaemia becomes maladaptive, amplifying oxidative stress and inflammation.¹⁵

The prognostic weight of this observation is substantial. In a prospective cohort of 2,471 patients hospitalised with CAP, an admission glucose above 11 mmol/L was associated with a 73% higher risk of death and a 52% higher risk of in-hospital complications compared with patients whose glucose was 6.1 mmol/L or below, and each 1 mmol/L increment raised complication risk by approximately 3% even after adjustment for the Pneumonia Severity Index.¹⁷

These findings were subsequently reinforced by the German CAPNETZ cohort of 6,891 patients, in which acute hyperglycaemia at admission independently predicted death at 28 and 90 days among patients without pre-existing diabetes, with the hazard rising progressively across glucose strata. A Danish population-based cohort of 29,900 patients similarly showed that a glucose of 14 mmol/L or above on admission predicted 30-day mortality more strongly in patients without diagnosed diabetes than in those with established disease, and observational data from non-critical care wards demonstrated that hyperglycaemia in non-diabetic patients with CAP was accompanied by higher C-reactive protein, leucocyte and pro-adrenomedullin concentrations over seven days as well as an adverse clinical course.^{18,19,20}

In the critical care setting the stress hyperglycaemia ratio — admission glucose divided by the estimated average glucose derived from glycated haemoglobin — has emerged as a more discriminating index than absolute glucose, because it corrects for background glycaemia. In 2,312

1075

critically ill patients with sepsis extracted from the MIMIC-IV database, this ratio showed a U-shaped association with 28-day and in-hospital mortality, and analyses across broader critical care populations have confirmed that relative rather than absolute hyperglycaemia independently predicts in-hospital death across the glycaemic spectrum.^{13,14,16}

The therapeutic corollary is that glucose should be measured and, when persistently elevated, treated. The American Diabetes Association recommends initiating or intensifying insulin therapy when blood glucose persistently exceeds 180 mg/dL, targeting 140–180 mg/dL in critically ill and 100–180 mg/dL in non-critically ill hospitalised individuals, and the Endocrine Society advises scheduled basal insulin with correction rather than sliding-scale monotherapy. The upper boundary of these recommendations was set by the NICE-SUGAR trial, in which intensive control to 81–108 mg/dL increased 90-day mortality and severe hypoglycaemia relative to a conventional target of 180 mg/dL or below, tempering the earlier enthusiasm generated by the Leuven surgical intensive care trial.^{7,8,9,10}

Yet in acute severe asthma the problem is compounded rather than simplified, because the treatment itself is diabetogenic. A systematic review and meta-analysis of 118 randomised trials found a pooled prevalence of hyperglycaemia of 10% in systemic corticosteroid arms, rising to 22% in respiratory illness, with intravenous administration conferring greater risk than oral therapy; and paediatric data have shown that oral corticosteroids, inhaled corticosteroids and inhaled β_2 -agonists each independently increase the odds of hyperglycaemia, with children who have asthma several times more likely to develop it than other acutely ill children.^{25,26,27}

Research Gap

Despite this considerable body of evidence, three gaps remain conspicuous. First, the literature on stress hyperglycaemia has developed along parallel and largely non-intersecting tracks: one examining dysglycaemia in sepsis and critical illness, a second examining admission glucose in CAP, and a third examining corticosteroid-induced hyperglycaemia in obstructive airway disease. Reports describing the simultaneous convergence of all three processes in a single non-diabetic patient — sepsis due to CAP, acute severe asthma requiring high-dose corticosteroid and repeated

β_2 -agonist nebulisation, and impending respiratory failure — are scarce, and the interaction between endogenous and iatrogenic hyperglycaemia in that specific context is poorly characterised.^{13,17,25}

Second, no current guideline specifies glycaemic targets or insulin regimens for the patient in whom sepsis-related and glucocorticoid-related hyperglycaemia coexist. The Endocrine Society systematic review identified only very low certainty evidence that combining neutral protamine Hagedorn insulin with a basal–bolus regimen achieves lower mean glucose than basal–bolus therapy alone in patients receiving glucocorticoids, and a 2025 systematic review of ten clinical practice guidelines found agreement on monitoring and on basal–bolus insulin but persistent disagreement on treatment targets and on the transition of therapy at discharge.^{10,11,12}

Third, almost all of the supporting evidence derives from tertiary and intensive care environments in high-resource settings. There is very little published experience describing how stress hyperglycaemia is detected and managed in a secondary-level district hospital that lacks an intensive care unit, arterial blood gas analysis, glycated haemoglobin testing and microbiological culture — the circumstances under which a large proportion of the world’s severely breathless patients are actually treated.¹²

Novelty of the Case Report

This report contributes four elements that, to the best of the authors’ knowledge, have not previously been presented together. First, it documents the detection and treatment of stress hyperglycaemia at the precise clinical moment of impending respiratory failure in a patient with no prior history of diabetes mellitus, thereby linking a metabolic observation to an airway decision point rather than to a retrospective outcome measure.^{15,17}

Second, it dissects the tripartite origin of the hyperglycaemia — endogenous stress response, high-dose intravenous methylprednisolone and repeated nebulised β_2 -agonist — and uses the resulting analysis to argue that in acute severe asthma the glycaemic consequence of therapy should be anticipated prospectively rather than discovered incidentally. Third, it demonstrates that a simple, low-cost basal insulin strategy can be delivered safely outside an intensive care unit in a resource-constrained Indonesian district hospital, with no hypoglycaemic event and no requirement for invasive ventilation.^{25,26}

Fourth, it provides an explicit, teachable reconstruction of the diagnostic reasoning required when two conventional anchors fail simultaneously: a normal leucocyte count in a septic patient and a non-specific chest radiograph in a patient with pneumonia complicating asthma. The report therefore functions both as a clinical narrative and as an instructional framework for physicians and residents working in comparable settings.^{1,3,6}

Objectives

General objective

To describe, analyse and critically discuss the clinical course, diagnostic reasoning and therapeutic decision-making — with particular emphasis on the recognition and insulin-based management of stress hyperglycaemia — in an adult non-diabetic patient presenting with sepsis secondary to community-acquired pneumonia with impending respiratory failure and a concurrent acute severe asthma exacerbation.^{1,3,6}

Specific objectives

1. To describe the clinical, laboratory, electrocardiographic and radiological profile of the patient at presentation and throughout the four-day admission.
2. To apply and interpret validated severity instruments — qSOFA, SOFA, CURB-65 and the GINA exacerbation severity classification — and to identify the limitations imposed by the absence of certain laboratory parameters.^{1,5,6}
3. To analyse the pathophysiological mechanisms by which stress hyperglycaemia arises in sepsis and acute severe asthma, and by which it may aggravate dyspnoea, hypoxaemia and infection.^{15,20}
4. To justify, on pharmacological and evidence-based grounds, the selection of insulin rather than non-insulin agents, the choice of a basal insulin analogue, the initial dose and the glycaemic target adopted.^{7,8,10,36}
5. To explain why empirical antimicrobial therapy was appropriate despite a normal leucocyte count, with reference to the Sepsis-3 definition and the Surviving Sepsis Campaign.^{1,2}
6. To explain why chest radiography is characteristically non-specific in asthma and why the radiological absence of consolidation does not exclude pneumonia.^{3,6}

7. To identify the deficiencies of the diagnostic work-up in this case and to translate them into practical recommendations for clinical practice, institutional policy and future research.^{11,12}

Benefits of the Case Report

Theoretical benefit

The report extends the conceptual understanding of dysglycaemia at the interface between infection-driven and treatment-driven metabolic derangement, a territory that has been examined separately in sepsis and in obstructive airway disease but seldom at their intersection. It provides a mechanistic account of the bidirectional relationship between hyperglycaemia and respiratory compromise that may inform subsequent hypothesis-generating research.^{15,20,25}

Clinical and practical benefit

For emergency physicians, internists and residents, the report offers a reproducible sequence of actions: measure capillary or random blood glucose in every severely dyspnoeic patient; interpret the result against the American Diabetes Association threshold of 180 mg/dL; anticipate the glycaemic impact of systemic corticosteroid and β_2 -agonist therapy; and select a scheduled basal insulin regimen with correction rather than relying on sliding-scale insulin alone.^{7,10}

Educational benefit

The case constitutes a compact teaching vehicle for three high-yield concepts that are commonly misunderstood at postgraduate level: that sepsis can occur without leucocytosis, that a normal or non-specific chest radiograph does not exclude pneumonia, and that hyperglycaemia in an acutely ill patient without diabetes is a marker of illness severity that requires action rather than observation.^{1,3,17}

Institutional and policy benefit

The report supports the case for embedding point-of-care glucose measurement into emergency department triage protocols and for developing standardised, nurse-driven insulin algorithms in district hospitals. A multidisciplinary quality-improvement initiative in an intensive care setting reduced the incidence of corticosteroid-induced hyperglycaemia from 48% to 26% and shortened length of stay by 0.8 days without increasing hypoglycaemia, indicating that protocolised glycaemic care is both feasible and beneficial.³⁷

Benefit to patients and families

Recognition and correction of hyperglycaemia contributed to a course that avoided endotracheal intubation, limited hospitalisation to four days and identified a previously unsuspected metabolic abnormality requiring outpatient follow-up. In a cohort of nearly 2,000 patients with CAP, 5.0% had undiagnosed diabetes and 37.5% had prediabetes, and those with undiagnosed diabetes had markedly higher 180-day mortality than those without — underlining the long-term value to the patient of pursuing the abnormal glucose after discharge.²¹

Working Hypotheses

A single case report cannot test a hypothesis in the inferential sense, and the propositions below are therefore presented as working explanatory statements that structure the analysis and that require confirmation in adequately powered prospective studies.

1. H₁. In a patient without pre-existing diabetes mellitus, sepsis secondary to community-acquired pneumonia accompanied by acute severe asthma and impending respiratory failure provokes clinically significant stress hyperglycaemia, the magnitude of which reflects the intensity of the neuroendocrine and inflammatory response.^{13,15}
2. H₂. Hyperglycaemia in this setting is not merely an epiphenomenon but participates in a self-reinforcing loop with hypoxaemia, increased work of breathing and catecholamine release, and thereby contributes to the severity and persistence of dyspnoea.^{15,20}
3. H₃. High-dose systemic corticosteroid therapy and repeated nebulised β_2 -agonist administration, both of which are mandatory in acute severe asthma, materially amplify the underlying stress hyperglycaemia and account for its persistence beyond the first 24 hours.^{25,26,31}
4. H₄. Early initiation of a scheduled basal insulin regimen, titrated to the American Diabetes Association inpatient target, achieves adequate glycaemic control without hypoglycaemia and is compatible with rapid clinical recovery in a secondary-level hospital lacking intensive care facilities.^{7,10,33}

CASE ILLUSTRATION

Patient Identity and Mode of Presentation

A 58-year-old Indonesian woman, a self-employed trader working from home and covered by the national health insurance scheme (BPJS Kesehatan), was referred by a primary clinic to the emergency department of RSUD Tani dan Nelayan, Tasikmalaya, West Java, in August 2026 with severe dyspnoea. She arrived in the late evening, seated upright, visibly distressed and unable to lie flat. Written informed consent for the publication of this anonymised case report and its accompanying images was obtained from the patient.

History of Present Illness

The patient reported long-standing exertional breathlessness that had become markedly worse over the preceding three days. She had used a home nebuliser without benefit before attending the primary clinic, where initial treatment produced a brief and partial improvement. Shortly afterwards the dyspnoea intensified until she was able to tolerate only the sitting position, could speak in single words only, and became restless and agitated. The breathlessness was accompanied by a productive cough of three days' duration in which the sputum was thick and difficult to expectorate, by an intermittent fever over the same period, and by nausea with epigastric pain. After a short period of observation at the clinic without clinical response, she was referred to hospital.

Past Medical, Family and Social History

Asthma had been present since childhood and had required recurrent treatment; she had been managed at a clinic one month previously and admitted to hospital four months previously with a similar presentation. Approximately twenty years earlier she had been told she had a cardiac valvular abnormality, described to her as a "weak valve"; she had remained asymptomatic since and had discontinued cardiology follow-up. In 2021 she sustained a stroke with right-sided weakness and slurred speech, from which she had recovered substantially, and she had likewise discontinued neurology follow-up. Hypertension was known but labile and irregularly monitored. She specifically denied any history of diabetes mellitus and any history of pulmonary tuberculosis, and she had never undergone glucose testing to her knowledge. Her father had hypertension; there was

no family history of similar respiratory illness. She had never smoked and reported no occupational or domestic exposure to smoke or biomass fuel.

Table 1. Baseline demographic and clinical characteristics at emergency department presentation

Domain	Finding
Age / sex	58 years / female
Occupation	Self-employed trader (works from home)
Insurance	BPJS Kesehatan (national health insurance)
Referral source	Primary clinic (klinik pratama), after failed initial treatment
Chief complaint	Severe dyspnoea
Duration of acute deterioration	3 days
Associated symptoms	Productive cough with tenacious sputum; intermittent fever; nausea; epigastric pain
Asthma	Since childhood; recurrent treatment; clinic attendance 1 month ago; hospital admission 4 months ago
Cardiac history	Valvular abnormality diagnosed ~20 years ago; asymptomatic; no follow-up
Cerebrovascular history	Ischaemic stroke 2021 with right hemiparesis and dysarthria; substantially recovered; no follow-up
Hypertension	Present, labile, irregularly monitored
Diabetes mellitus	Denied; no previous glucose testing recalled
Pulmonary tuberculosis	Denied
Family history	Father with hypertension; no similar respiratory illness
Smoking / smoke exposure	Never smoked; no significant environmental smoke exposure

Physical Examination

On arrival the patient appeared severely ill with marked respiratory distress. She was conscious but agitated, with a Glasgow Coma Scale of 14 (E3 V5 M6). Body weight was 57 kg and height 159 cm as reported by the family, giving a body mass index of 22.6 kg/m². Blood pressure was 121/87 mmHg, heart rate 116 beats per minute, temperature 37.1°C and respiratory rate 62 breaths per minute. Peripheral oxygen saturation was 87% on room air, rising to 99% with oxygen at 10 L/min delivered by non-rebreather mask.

Table 2. Vital signs and anthropometry on admission

Parameter	Value on admission	Reference / comment
General appearance	Severe respiratory distress; tripod / sitting position only	Unable to lie supine
Consciousness	Compos mentis but agitated; GCS 14 (E3 V5 M6)	GCS < 15 scores 1 point on qSOFA and SOFA CNS domain
Speech	Single words only	GINA criterion for severe exacerbation
Body weight	57 kg	Family report
Height	159 cm	Family report
Body mass index	22.6 kg/m ²	Normal (18.5–24.9 kg/m ²)
Blood pressure	121/87 mmHg	MAP ≈ 98 mmHg; no hypotension
Heart rate	116 beats/min	Sinus tachycardia
Respiratory rate	62 breaths/min	Markedly elevated (normal 12–20)
Temperature	37.1 °C (axillary)	Afebrile at presentation despite reported fever
SpO ₂ (room air)	87 %	Hypoxaemic; GINA life-threatening threshold < 90 %
SpO ₂ (O ₂ 10 L/min NRM)	99 %	Rapid response to high-flow oxygen

GCS = Glasgow Coma Scale; GINA = Global Initiative for Asthma; MAP = mean arterial pressure; NRM = non-rebreather mask; qSOFA = quick Sequential Organ Failure Assessment; SpO₂ = peripheral oxygen saturation.

Table 3. Systematic physical examination findings

System	Findings
Head and face	Normocephalic; conjunctivae not pale; sclerae not icteric; pupils isochoric 3 mm / 3 mm with brisk direct light reflex bilaterally; external ears normal; nasal cavities patent; no nasal flaring; lips dry; no central cyanosis
Neck	No cervical lymphadenopathy; jugular venous pressure not elevated
Thorax (general)	Rapid respiration; symmetrical chest wall movement; visible intercostal retraction
Lungs	Vesicular breath sounds present and equal bilaterally; no crackles; bilateral wheeze present but barely audible (silent chest); intercostal retraction present
Heart	First and second heart sounds irregular; no murmur; no gallop
Abdomen	Distended and hypertympanic; soft on palpation; epigastric tenderness present; bowel sounds normal
Extremities	Warm peripheries; capillary refill time < 2 seconds; no pallor; no pitting oedema

Investigations

Complete blood count and random blood glucose

The complete blood count was unremarkable. Haemoglobin, haematocrit, erythrocyte and platelet counts were within the reference range, and — importantly — the leucocyte count was $5.1 \times 10^3/\mu\text{L}$, that is, entirely normal and towards the lower half of the reference interval. Mean corpuscular volume was marginally reduced at 79 fL and mean corpuscular haemoglobin concentration marginally raised at 36 g/dL, findings of limited clinical significance in isolation. The

single striking abnormality was the random blood glucose of 248 mg/dL (13.8 mmol/L) against a reference value below 140 mg/dL, in a patient who had consistently denied any history of diabetes mellitus.⁷

Table 4. Laboratory results on admission

Investigation	Result	Reference range	Interpretation
Haemoglobin	15.4 g/dL	13.2–17.3	Normal
Haematocrit	43 %	40–52	Normal
Leucocytes	$5.1 \times 10^3/\mu\text{L}$	3.8–10.6	Normal — no leucocytosis
Erythrocytes	$5.42 \times 10^6/\mu\text{L}$	4.4–5.9	Normal
Platelets	$257 \times 10^3/\mu\text{L}$	150–450	Normal (SOFA coagulation = 0)
MCV	79 fL	80–96	Marginally low
MCH	29 pg	27.5–33.2	Normal
MCHC	36 g/dL	33.4–35.5	Marginally high
Random blood glucose	248 mg/dL (13.8 mmol/L)	< 140	Markedly elevated — above ADA treatment threshold of 180 mg/dL
Fasting plasma glucose (day 2)	150 mg/dL (8.3 mmol/L)	70–100	Persistently elevated
Urea / creatinine	Not performed	—	Limits CURB-65 and SOFA renal scoring
Arterial blood gas / lactate	Not performed	—	Not available at this facility
HbA _{1c}	Not performed	—	Prevents calculation of stress hyperglycaemia ratio

Blood and sputum culture	Not performed	—	Not available at this facility
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ADA = American Diabetes Association; HbA_{1c} = glycated haemoglobin; MCH = mean corpuscular haemoglobin; MCHC = mean corpuscular haemoglobin concentration; MCV = mean corpuscular volume; SOFA = Sequential Organ Failure Assessment.

Electrocardiography

A twelve-lead electrocardiogram recorded on the evening of admission demonstrated a ventricular rate of 116 beats per minute with a PR interval of 210 ms, QRS duration of 98 ms and QT/QTc intervals of 358/455 ms. The automated interpretation reported sinus tachycardia with borderline first-degree atrioventricular block, suggested consideration of left atrial abnormality, and described junctional ST depression as non-specific, concluding that the tracing was borderline. There was no evidence of acute ischaemic injury, of right heart strain or of an arrhythmia requiring immediate intervention; the tachycardia was consistent with the physiological response to hypoxaemia, increased work of breathing and repeated β_2 -agonist administration.⁶

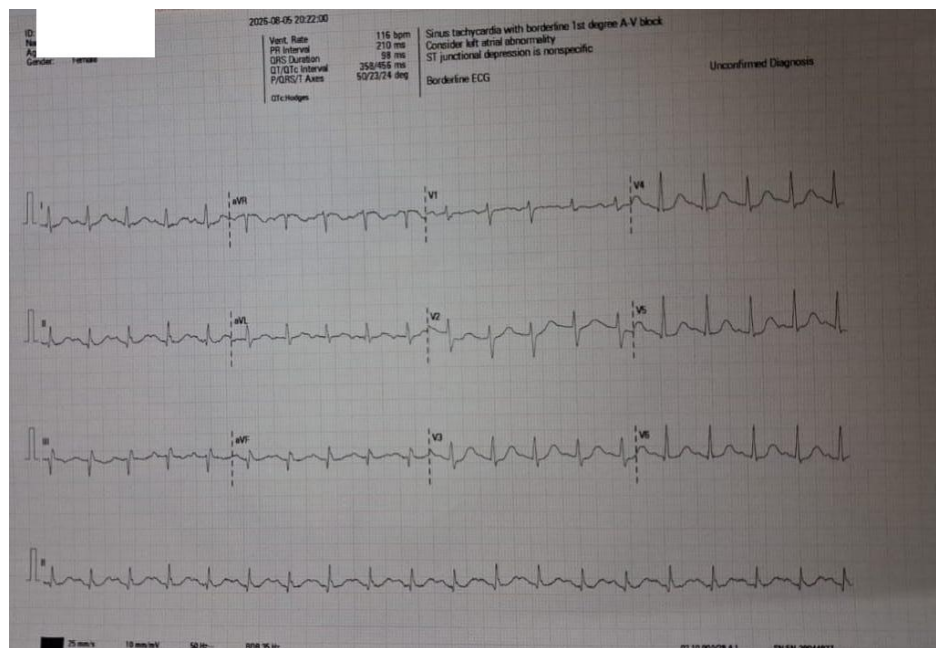


Figure 1. Twelve-lead electrocardiogram on admission showing sinus tachycardia at 116 beats/min with borderline first-degree atrioventricular block and non-specific junctional ST depression.

Patient identifiers have been redacted.

Table 5. Electrocardiographic parameters and interpretation

Parameter	Value	Comment
Rhythm	Sinus	Regular sinus mechanism
Ventricular rate	116 beats/min	Sinus tachycardia
PR interval	210 ms	Borderline first-degree AV block (normal 120–200 ms)
QRS duration	98 ms	Normal (< 120 ms)
QT / QTc (Hodges)	358 / 455 ms	QTc at upper limit; relevant to β_2 -agonist and electrolyte shifts
P / QRS / T axes	50° / 23° / 24°	Normal axis; no right axis deviation
ST segment	Junctional ST depression	Reported as non-specific
Atrial morphology	Possible left atrial abnormality	Consistent with reported remote valvular disease
Overall	Borderline electrocardiogram	No acute ischaemic pattern; no right heart strain

Chest radiography

A chest radiograph was obtained the following morning in the anteroposterior supine projection, symmetrical and with adequate inspiration and exposure. There was no widening of the superior mediastinum. The costophrenic angles and the diaphragmatic contours were normal. The cardiac silhouette was enlarged towards the left with the apex embedded in the diaphragm and obliteration of the cardiac waist, giving a cardiothoracic ratio exceeding 50%. The aorta showed no elongation but did show calcification. The hila were relatively coarse, and the bronchovascular markings were increased and coarsened throughout. There was no cranialisation of the pulmonary

vasculature, and no fracture of the ribs, clavicles or scapulae was identified. The reporting radiologist concluded that the appearances suggested a bronchitic pattern, cardiomegaly without pulmonary congestion, and aortic atherosclerosis.



Figure 2. Anteroposterior supine chest radiograph demonstrating increased and coarsened bronchovascular markings, relatively coarse hila, and an enlarged cardiac silhouette with obliteration of the cardiac waist. No lobar consolidation, pneumothorax or pleural effusion is seen. Patient identifiers have been redacted.

Fasting plasma glucose

In accordance with the instruction of the consulting internal medicine specialist, a fasting plasma glucose was obtained on the morning following admission, after the first dose of basal insulin and after the patient had received intravenous methylprednisolone and repeated nebulised bronchodilator therapy. The result was 150 mg/dL (8.3 mmol/L) against a reference interval of 70–100 mg/dL, confirming that the dysglycaemia was persistent rather than a transient single

measurement, while remaining below the 180 mg/dL threshold at which the American Diabetes Association advises intensification of therapy.⁷

Severity Assessment

Formal severity scoring was applied retrospectively using the data available at the bedside. The patient satisfied two of the three qSOFA criteria — a respiratory rate of at least 22 breaths per minute and altered mentation with a Glasgow Coma Scale below 15 — while systolic blood pressure remained above 100 mmHg. A qSOFA of 2 identifies a patient with suspected infection who is at substantially increased risk of a prolonged intensive care stay or death and mandates urgent assessment for organ dysfunction.¹

Table 6. qSOFA score at emergency department presentation

qSOFA criterion	Threshold	Patient value	Points
Respiratory rate	≥ 22 breaths/min	62 breaths/min	1
Altered mentation	GCS < 15	GCS 14 (E3 V5 M6), agitated	1
Systolic blood pressure	≤ 100 mmHg	121 mmHg	0
Total	—	—	2 of 3 (positive)

A qSOFA score of 2 or more in a patient with suspected infection indicates a markedly increased risk of adverse outcome and prompts formal assessment for organ dysfunction using the full SOFA score.¹

The full SOFA score could only be estimated, because serum bilirubin, creatinine and arterial blood gas analysis were unavailable. Taking a peripheral oxygen saturation of 87% on room air as corresponding to an arterial oxygen tension of approximately 55 mmHg, the estimated $\text{PaO}_2/\text{FiO}_2$ ratio was approximately 262 mmHg, scoring two points in the respiratory domain; the Glasgow Coma Scale of 14 scored one point in the central nervous system domain; mean arterial pressure of approximately 98 mmHg and a platelet count of $257 \times 10^3/\mu\text{L}$ scored zero. The resulting minimum estimated SOFA of 3, arising acutely in a patient with no documented pre-existing organ

dysfunction, satisfies the Sepsis-3 requirement of an acute increase of two points or more and therefore establishes sepsis.¹

Table 7. Estimated SOFA score at presentation

SOFA domain	Variable used	Patient value	Score
Respiration	PaO ₂ /FiO ₂ (estimated from SpO ₂ 87 % on room air)	≈ 262 mmHg	2
Coagulation	Platelet count	257 × 10 ³ /μL	0
Liver	Serum bilirubin	Not measured	0 (assumed)
Cardiovascular	Mean arterial pressure	≈ 98 mmHg, no vasopressor	0
Central nervous system	Glasgow Coma Scale	14	1
Renal	Creatinine / urine output	Not measured	0 (assumed)
Estimated total	—	—	≥ 3

Values marked “assumed” reflect the absence of the corresponding laboratory measurement at this facility; the true SOFA score may therefore have been higher than the estimate presented. FiO₂ = fraction of inspired oxygen; PaO₂ = arterial partial pressure of oxygen.

For severity stratification of the pneumonia, the CURB-65 instrument was applied. New-onset confusion was accepted on the basis of agitation with a Glasgow Coma Scale of 14, and the respiratory rate of 62 breaths per minute clearly exceeded the threshold of 30; blood pressure and age did not contribute points, and serum urea was unavailable. The resulting score of 2 corresponded, in the original derivation cohort of 1,068 patients, to a 30-day mortality of approximately 3%, rising steeply to 17% at a score of 3. Because urea was not measured, the score obtained should be regarded as a lower bound.⁵

Table 8. CURB-65 score for community-acquired pneumonia severity

Component	Definition	Patient status	Points
C — Confusion	New-onset disorientation or reduced consciousness	Agitated, GCS 14 — present	1
U — Urea	> 7 mmol/L (> 19 mg/dL)	Not measured	Not scored
R — Respiratory rate	≥ 30 breaths/min	62 breaths/min — present	1
B — Blood pressure	Systolic < 90 or diastolic ≤ 60 mmHg	121/87 mmHg — absent	0
65 — Age	≥ 65 years	58 years — absent	0
Total (minimum)	—	—	2 (urea not available)

In the original international derivation and validation study, reported 30-day mortality was 0.7 % for a score of 0, 3.2 % for 1, 3 % for 2, 17 % for 3, 41.5 % for 4 and 57 % for 5.⁵

The asthma exacerbation was classified according to the GINA framework. The patient satisfied every criterion for a severe exacerbation — speech limited to single words, inability to adopt any posture other than sitting, agitation, respiratory rate far above 30 breaths per minute, accessory muscle use with intercostal retraction and oxygen saturation below 90% on room air — and in addition demonstrated two features that define a life-threatening exacerbation, namely a silent chest and drowsiness or agitation. Peak expiratory flow could not be measured because the patient was too breathless to perform the manoeuvre, a limitation explicitly acknowledged in the guideline for patients of this severity.⁶

Table 9. GINA classification of acute asthma exacerbation severity

GINA feature	Severe exacerbation criterion	Patient finding	Present
Speech	Talks in words	Single words only	Yes
Posture	Sits hunched forward	Can tolerate sitting position only	Yes
Mental state	Agitated	Agitated, GCS 14	Yes
Respiratory rate	> 30 breaths/min	62 breaths/min	Yes
Accessory muscles	In use	Intercostal retraction present	Yes
Pulse rate	> 120 beats/min	116 beats/min	Near threshold
SpO ₂ on room air	< 90 %	87 %	Yes
PEF	≤ 50 % predicted or best	Not measurable — too breathless	Not assessed
Silent chest	Life-threatening feature	Wheeze barely audible bilaterally	Yes
Drowsiness / confusion	Life-threatening feature	Agitation with GCS 14	Yes
Classification	—	Severe exacerbation with life-threatening features	—

*GINA = Global Initiative for Asthma; PEF = peak expiratory flow; SpO₂ = peripheral oxygen saturation.*⁶

Finally, the constellation of findings satisfied the clinical definition of impending respiratory failure: inability to speak more than single words, exhaustion with agitation and a falling level of consciousness, a silent chest indicating airflow so limited that turbulent sound could no longer be generated, extreme tachypnoea with accessory muscle recruitment, and hypoxaemia on room air.

Arterial blood gas analysis, which would conventionally demonstrate a normal or rising arterial carbon dioxide tension in this situation, was not available.⁶

Table 10. Clinical criteria for impending respiratory failure identified in this patient

Criterion	Patient finding	Present
Inability to speak more than single words	Single-word speech	Yes
Exhaustion, agitation or deteriorating consciousness	Agitated, GCS 14 (E3 V5 M6)	Yes
Silent chest or markedly diminished breath sounds	Wheeze barely audible bilaterally	Yes
Extreme tachypnoea with accessory muscle use	RR 62/min with intercostal retraction	Yes
Hypoxaemia despite spontaneous ventilation	SpO ₂ 87 % on room air	Yes
Normal or rising PaCO ₂ despite tachypnoea	Arterial blood gas not available	Not assessed
Haemodynamic instability	Blood pressure maintained at 121/87 mmHg	No

Diagnosis

Integrating the history, examination, laboratory, electrocardiographic and radiological findings, the following working diagnoses were established in the emergency department and endorsed by the consulting internal medicine specialist.

1. Sepsis secondary to community-acquired pneumonia with impending respiratory failure (qSOFA 2 of 3; estimated SOFA $\geq$ 3; CURB-65 $\geq$ 2).^{1,3,5}
2. Acute severe asthma exacerbation with life-threatening features (silent chest, agitation, SpO₂ 87 % on room air).⁶

3. Stress hyperglycaemia, newly detected, in a patient with no known diabetes mellitus (random blood glucose 248 mg/dL).^{7,15}
4. Dyspepsia (nausea and epigastric tenderness).
5. Additional findings requiring outpatient evaluation: radiographic cardiomegaly without pulmonary congestion, aortic atherosclerosis, irregular heart sounds with a history of valvular disease, hypertension, and previous ischaemic stroke.

Management in the Emergency Department

Treatment was initiated immediately and in parallel rather than sequentially, reflecting the concurrent threats to oxygenation, airway calibre, infection control and metabolic homeostasis. Oxygen was delivered at 10 L/min through a non-rebreather mask, restoring saturation to 99%. Intravenous crystalloid was given as 0.9% sodium chloride 1,000 mL over 24 hours, a deliberately conservative volume given the presence of radiographic cardiomegaly and the absence of hypotension. Empirical antimicrobial therapy comprised ceftriaxone 2 g intravenously once daily together with levofloxacin 750 mg intravenously once daily. Airway inflammation was addressed with intravenous methylprednisolone 125 mg three times daily, and bronchospasm with nebulised salbutamol–ipratropium combined with budesonide every 20 minutes for the first hour and subsequently every six hours. A single additional dose of magnesium sulphate 20%, 2 g in 100 mL of 5% dextrose over two hours, was administered as adjunctive bronchodilator therapy for the severe exacerbation. N-acetylcysteine 200 mg three times daily was prescribed as a mucolytic for the tenacious sputum, and omeprazole 20 mg intravenously once daily for the dyspeptic symptoms and for gastroprotection during high-dose corticosteroid therapy.^{2,3,6}

For the hyperglycaemia, insulin glargine 8 units was prescribed subcutaneously at night, equivalent to approximately 0.14 units per kilogram per day, and a fasting plasma glucose was requested for the following morning. No oral glucose-lowering agent was used. This regimen represented a deliberately conservative basal-only strategy in a patient with no previous exposure to insulin, no established diagnosis of diabetes mellitus and an uncertain oral intake.^{7,10}

Table 11. Emergency department therapy with pharmacological rationale

Therapy	Dose and route	Indication and rationale
Oxygen	10 L/min via non-rebreather mask	Correction of hypoxaemia (SpO ₂ 87 % → 99 %); GINA target saturation ≥ 94 %
Sodium chloride 0.9 %	1,000 mL intravenously over 24 h	Restoration of intravascular volume and correction of osmotic losses from hyperglycaemia; volume restricted in view of radiographic cardiomegaly
Ceftriaxone	2 g intravenously once daily	Empirical β-lactam coverage of <i>Streptococcus pneumoniae</i> and <i>Haemophilus influenzae</i> in CAP
Levofloxacin	750 mg intravenously once daily	Respiratory fluoroquinolone providing atypical pathogen coverage
Methylprednisolone	125 mg intravenously three times daily	Suppression of airway inflammation in severe asthma exacerbation
Salbutamol–ipratropium nebulisation	Every 20 min for 1 h, then every 6 h	Short-acting β ₂ -agonist plus antimuscarinic bronchodilation
Budesonide nebulisation	With each bronchodilator nebulisation	Inhaled corticosteroid adjunct to systemic therapy
Magnesium sulphate 20 %	2 g in 100 mL dextrose 5 % over 2 h (single dose)	Adjunctive smooth-muscle relaxation in severe exacerbation unresponsive to initial therapy
N-acetylcysteine	200 mg orally three times daily	Mucolysis of tenacious sputum difficult to expectorate
Omeprazole	20 mg intravenously	Dyspepsia; gastroprotection during high-

	once daily	dose corticosteroid therapy
Insulin glargine	8 units subcutaneously at night (0–0–8)	Control of stress hyperglycaemia (RBG 248 mg/dL) above the ADA treatment threshold of 180 mg/dL
Fasting plasma glucose	Next morning	Assessment of response to basal insulin and persistence of dysglycaemia

ADA = American Diabetes Association; CAP = community-acquired pneumonia; GINA = Global Initiative for Asthma; RBG = random blood glucose.

Clinical Course

No procedural intervention was required. The patient was not intubated, did not receive non-invasive ventilation and was not transferred to a higher level of care. She was managed on the general medical ward and improved progressively on each successive day, with reduction in respiratory rate, improvement in oxygen saturation on decreasing oxygen requirement, restoration of full sentence speech and resolution of fever, cough and epigastric discomfort.

Table 12. Clinical timeline over the four-day admission

Day	Clinical status and events	Key measurements
Day 0 (ED)	Referred with severe dyspnoea; single-word speech; agitation; silent chest; high-flow oxygen commenced; empirical antibiotics, systemic corticosteroid, nebulisation, magnesium sulphate and basal insulin started	RR 62/min; HR 116/min; BP 121/87 mmHg; SpO ₂ 87 % RA → 99 % on NRM; T 37.1 °C; GCS 14; WBC $5.1 \times 10^3/\mu\text{L}$; RBG 248 mg/dL
Day 1	Chest radiograph obtained; fasting glucose drawn; dyspnoea decreasing; wheeze becoming audible as airflow improved; continued nebulisation every 6 h	FPG 150 mg/dL; oxygen requirement decreasing

Day 2	Speech in short phrases; fever settled; sputum expectoration easier; oral intake resumed	Progressive fall in respiratory rate
Day 3	Ambulating with minimal dyspnoea; chest auscultation clearing	Oxygen weaned towards room air
Day 4 (discharge)	Full sentence speech; cough much reduced; afebrile; chest clear; discharged on oral therapy with home oxygen standby and internal medicine follow-up	RR 23/min; HR 76/min; BP 140/89 mmHg; SpO ₂ 95 % on room air; T 36.0 °C; GCS 15

BP = blood pressure; ED = emergency department; FPG = fasting plasma glucose; GCS = Glasgow Coma Scale; HR = heart rate; NRM = non-rebreather mask; RA = room air; RBG = random blood glucose; RR = respiratory rate; T = temperature; WBC = white blood cell count.

Table 13. Comparison of clinical parameters between admission and discharge

Parameter	Admission (day 0)	Discharge (day 4)	Direction of change
Level of consciousness	GCS 14, agitated	GCS 15, calm	Improved
Speech	Single words	Full sentences	Improved
Respiratory rate	62 breaths/min	23 breaths/min	Reduced by 63 %
Heart rate	116 beats/min	76 beats/min	Normalised
Blood pressure	121/87 mmHg	140/89 mmHg	Higher — reflects hypertension and corticosteroid effect
SpO ₂	87 % on room air	95 % on room air	Improved
Temperature	37.1 °C	36.0 °C	Normalised
Lung auscultation	Silent chest,	Vesicular, no wheeze	Resolved

	intercostal retraction	or crackles, no retraction	
Heart sounds	S1–S2 irregular, no murmur	S1–S2 irregular, no murmur	Unchanged — requires outpatient evaluation
Abdomen	Distended, epigastric tenderness	Flat, no tenderness	Resolved
Blood glucose	RBG 248 mg/dL	FPG 150 mg/dL (day 1)	Improved but still above target
Oxygen requirement	10 L/min NRM	Room air	Weaned completely

Condition at Discharge and Discharge Plan

On the fourth hospital day the patient reported that her breathlessness had improved greatly, that she could complete a full sentence without becoming breathless, that her cough had diminished substantially and that she had had no further fever. Examination showed mild residual dyspnoea only, a Glasgow Coma Scale of 15, blood pressure 140/89 mmHg, heart rate 76 beats per minute, oxygen saturation 95% on room air, temperature 36.0°C and a respiratory rate of 23 breaths per minute. The lips were no longer dry, there was no cyanosis and no jugular venous distension. Chest movement was symmetrical with normal vesicular breath sounds bilaterally and no wheeze or crackles. The first and second heart sounds remained irregular without murmur. The abdomen was flat, soft and non-tender with normal bowel sounds, and the extremities were warm with a capillary refill time under two seconds and no oedema.

Table 14. Discharge prescription and instructions

Medication / instruction	Dose and frequency	Purpose
Cefixime	200 mg orally twice daily	Oral step-down β -lactam therapy for CAP

Levofloxacin	750 mg orally once daily	Continuation of respiratory fluoroquinolone coverage
N-acetylcysteine	200 mg orally three times daily	Mucolytic
Methylprednisolone	32 mg orally once daily	Tapering systemic corticosteroid after severe exacerbation
Omeprazole	20 mg orally once daily	Dyspepsia and gastroprotection
Insulin glargine	8 units subcutaneously at night	Continued basal control of hyperglycaemia
Salmeterol–fluticasone 250	One inhalation twice daily	Inhaled corticosteroid–long-acting β_2 -agonist maintenance therapy
Salbutamol	4 mg orally twice daily as required	Reliever therapy for breakthrough dyspnoea
Home oxygen	To be available at home	Contingency for recurrent hypoxaemia
Follow-up	Internal medicine outpatient clinic; fasting plasma glucose to be repeated before medication is exhausted	Reassessment of glycaemic status and of asthma control

The patient was specifically instructed to attend the internal medicine outpatient clinic and to have a repeat fasting plasma glucose performed at that visit, before her supply of medication was exhausted, so that the persistence or resolution of the hyperglycaemia could be determined and the insulin either continued, converted or withdrawn accordingly.⁷

DISCUSSION

The value of this case lies less in the rarity of any single diagnosis than in the density of their intersection. Within a single patient, four distinct processes — an infection-driven systemic response, an obstructive airway crisis, an evolving failure of gas exchange and an acute metabolic

derangement — developed simultaneously, and each of them modified the presentation, the interpretation and the treatment of the others. The discussion that follows examines these processes in turn, then addresses the therapeutic decisions taken and the deficiencies of the work-up.

Establishing Sepsis under the Sepsis-3 Framework

The Third International Consensus Definitions replaced the earlier reliance on systemic inflammatory response syndrome criteria with a definition anchored in organ dysfunction: sepsis is life-threatening organ dysfunction caused by a dysregulated host response to infection, identified clinically by an acute rise of two or more points in the Sequential Organ Failure Assessment score in a patient with suspected infection. The quick SOFA was introduced not as a diagnostic instrument but as a bedside prompt: two or more of altered mentation, systolic blood pressure of 100 mmHg or below, and respiratory rate of 22 breaths per minute or above should trigger a search for organ dysfunction and for the source of infection.¹

Applied to this patient, the framework yields an unambiguous conclusion. Two qSOFA criteria were met on arrival, and the estimated SOFA of at least 3 — two points for the respiratory domain and one for the central nervous system domain — arose acutely in a woman with no documented pre-existing organ dysfunction. That the true score may have been higher, because bilirubin, creatinine and arterial blood gases were not obtained, only strengthens the conclusion. The suspected source was the respiratory tract, supported by three days of fever, productive cough with purulent tenacious sputum and the radiographic bronchitic pattern.^{1,3}

The Surviving Sepsis Campaign recommends that antimicrobials be administered immediately, ideally within one hour, for adults with possible septic shock or a high likelihood of sepsis, and within three hours where sepsis is possible but the likelihood lower and shock is absent. In this patient, in whom organ dysfunction was manifest and the respiratory source clinically evident, immediate empirical therapy was the correct course.²

Why Empirical Antibiotics Were Justified Despite a Normal Leucocyte Count

The normal leucocyte count of $5.1 \times 10^3/\mu\text{L}$ is the single most likely element of this case to generate diagnostic hesitation, and it deserves careful treatment because the reasoning generalises

well beyond this patient. Three arguments converge on the conclusion that antibiotics were indicated.

First, and most fundamentally, leucocytosis is no longer part of the definition of sepsis. The Sepsis-3 task force explicitly abandoned the systemic inflammatory response syndrome criteria on the grounds that they lacked specificity and, more importantly, sensitivity — a substantial proportion of patients with infection-related organ dysfunction and high mortality never satisfy two SIRS criteria. To require leucocytosis before treating sepsis is therefore to apply a superseded and demonstrably unsafe standard.¹

Second, the white cell count is a poor discriminator in respiratory infection for reasons that are well recognised at the bedside. Patients may present in the earliest phase of the leucocyte response, before demargination and bone marrow release have raised the circulating count; the count may be suppressed in overwhelming infection; and older adults frequently mount a blunted leucocyte response. Notably, the leucocyte count is not a component of either CURB-65 or the qSOFA, both of which stratify risk without reference to it, whereas the American Thoracic Society and Infectious Diseases Society of America base the decision to treat on the clinical and radiographic diagnosis of pneumonia together with severity assessment.^{3,5}

Third, the potential harm of withholding therapy vastly exceeds that of a short course of empirical antibiotics in a patient with organ dysfunction. Observational data from CAP cohorts consistently show that hyperglycaemia, tachypnoea, confusion and hypoxaemia — all present here — predict adverse outcome, and that the inflammatory response measured by C-reactive protein, leucocyte count and pro-adrenomedullin is heterogeneous rather than uniformly elevated. In a cohort of 880 patients with confirmed CAP, hyperglycaemia in non-diabetic patients was itself associated with higher inflammatory marker concentrations and an adverse clinical course, indicating that the metabolic signal may sometimes be more informative than the haematological one.²⁰

A separate and more critical point concerns the antibiotic regimen chosen. Current guidance for inpatients with non-severe CAP recommends either a β -lactam combined with a macrolide, or monotherapy with a respiratory fluoroquinolone; for severe CAP, a β -lactam plus either a macrolide

or a fluoroquinolone is recommended. The combination of ceftriaxone with levofloxacin used here is therefore defensible if the pneumonia is classified as severe — which, given the impending respiratory failure, is arguable — but it represents broader coverage than a strict reading of the guideline requires for non-severe disease, and it carries the recognised fluoroquinolone risks of tendinopathy, QT prolongation and dysglycaemia. The latter deserves particular emphasis in a patient already hyperglycaemic. De-escalation to a single agent once clinical stability was achieved, and the continuation of both cefixime and levofloxacin at discharge, would both merit reconsideration against contemporary stewardship principles and the 2026 American Thoracic Society update.^{3,4}

Acute Severe Asthma with Life-Threatening Features: The Significance of the Silent Chest

The GINA strategy report defines a severe exacerbation by the inability to speak in sentences, upright posture, agitation, respiratory rate above 30 breaths per minute, accessory muscle use, pulse above 120 beats per minute, oxygen saturation below 90% on room air and peak expiratory flow at or below 50% of predicted. Drowsiness, confusion or a silent chest indicate a life-threatening exacerbation. This patient satisfied all of the severe criteria that could be assessed and both of the life-threatening ones.⁶

The silent chest is frequently misinterpreted by inexperienced clinicians as evidence of improvement, because the wheeze that had previously dominated auscultation has diminished. In fact the opposite is true: wheeze is generated by turbulent flow through narrowed airways, and its disappearance in a patient who is more rather than less distressed signifies that airflow has fallen below the threshold at which sound can be produced. In this patient the sequence was documented in reverse during recovery — wheeze became audible again on day one as airflow improved, before disappearing entirely by discharge. This natural experiment within a single admission is one of the clearest illustrations of the principle that the clinical meaning of a physical sign depends entirely on its trajectory and on the accompanying respiratory effort.⁶

It is also worth noting that the heart rate of 116 beats per minute fell marginally below the GINA threshold of 120. Isolated adherence to a numerical cut-off would have understated the severity; the composite clinical picture, in which single-word speech, agitation, a respiratory rate of

1102

62 and a silent chest all pointed the same way, correctly overrode a single sub-threshold vital sign. Severity classification in asthma is a gestalt supported by criteria, not an arithmetic exercise.⁶

Recognition of Impending Respiratory Failure

Impending respiratory failure describes the interval during which a patient is still ventilating spontaneously but is doing so at a cost that cannot be sustained. The clinical markers are those of exhaustion and of failing compensation rather than of established failure: single-word speech, agitation progressing towards drowsiness, a silent or near-silent chest, respiratory rates so high that tidal volume must be falling, accessory muscle recruitment and hypoxaemia. Recognition matters because the therapeutic window during which aggressive medical therapy may still avert intubation is narrow.⁶

The absence of arterial blood gas analysis at this facility removed the most objective marker of this transition, namely a normal or rising arterial carbon dioxide tension in a patient breathing at 62 times per minute — a finding that indicates the respiratory pump is failing to keep pace with the load. The clinical team compensated by relying on the composite bedside picture and by escalating therapy immediately, an approach that in this instance proved sufficient. In facilities where blood gas analysis is unavailable, serial assessment of speech, mental state, respiratory rate and the audibility of wheeze constitutes the practical substitute, and each of these should be documented explicitly at short intervals.⁶

Why the Chest Radiograph Is Non-Specific in Asthma

Asthma is a disease of the small and medium airways in which the abnormality is functional — reversible bronchoconstriction, mucosal oedema and mucus plugging — rather than one of parenchymal density. Because plain radiography images differences in tissue attenuation, an airway that is narrowed but still patent produces no discernible radiographic signature. The chest radiograph in acute asthma is therefore typically normal, or shows only hyperinflation with flattened diaphragms, increased retrosternal air space and coarsened peribronchial markings. Its principal role is not to confirm asthma but to exclude complications and alternative diagnoses: pneumothorax, pneumomediastinum, lobar collapse, consolidation, foreign body and cardiac failure.⁶

The report in this case — increased and coarsened bronchovascular markings with relatively coarse hila, summarised as a bronchitic pattern — illustrates the difficulty precisely. That descriptor is compatible with asthma itself, with acute bronchitis, with atypical or viral pneumonia, and with chronic changes from years of recurrent airway inflammation in a patient with lifelong asthma. It neither confirms nor excludes bacterial pneumonia. Conversely, the absence of lobar consolidation does not exclude pneumonia: early infection may antedate radiographic change, dehydration reduces the conspicuity of infiltrates, and hyperinflated lungs in an asthmatic patient reduce contrast further. The supine anteroposterior technique used here compounds all of these effects, and interobserver agreement for the radiographic diagnosis of pneumonia is only moderate even among experienced readers.^{3,4}

The same technical caveat applies with even greater force to the cardiac silhouette. A cardiothoracic ratio exceeding 50% on a supine anteroposterior film obtained in a tachypnoeic patient is unreliable, because the short focus-to-film distance magnifies the heart, the supine position increases venous return and mediastinal width, and shallow inspiration elevates the diaphragm. The reported cardiomegaly, occurring in a woman with a remote diagnosis of valvular disease, an irregular first and second heart sound, hypertension and possible left atrial abnormality on electrocardiography, therefore cannot be dismissed but equally cannot be accepted at face value. Transthoracic echocardiography is the appropriate next investigation and its omission is discussed among the limitations below.

Table 15. Differential diagnosis of acute severe dyspnoea in this patient and the basis for retention or exclusion

Differential diagnosis	Supporting features	Opposing features	Conclusion
Acute severe asthma exacerbation	Lifelong asthma; recurrent admissions; silent chest with bilateral wheeze;	None	Retained as primary

	intercostal retraction; rapid response to bronchodilator and corticosteroid		
Community- acquired pneumonia with sepsis	Three days of fever, productive purulent sputum; qSOFA 2; estimated SOFA ≥ 3 ; coarse bronchovascular markings	Normal leucocyte count; no lobar consolidation	Retained — leucocytosis not required under Sepsis-3
Acute decompensated heart failure	Radiographic cardiomegaly; irregular heart sounds; remote valvular disease; hypertension	Jugular venous pressure not elevated; no crackles; no peripheral oedema; no pulmonary congestion or vascular cranialisation on radiograph	Considered unlikely acutely; requires outpatient echocardiography
Pulmonary embolism	Tachypnoea; tachycardia; hypoxaemia; previous stroke	No pleuritic pain, haemoptysis or unilateral leg swelling; normal electrocardiographic axis without right heart strain; rapid response to bronchodilator therapy	Considered unlikely; D- dimer and computed tomography unavailable
Pneumothorax	Sudden severe dyspnoea in an asthmatic patient	Symmetrical chest expansion; bilateral breath sounds; no	Excluded

		radiographic pneumothorax	
Pulmonary tuberculosis	Cough; intermittent fever; endemic setting	Three-day rather than chronic course; no weight loss or night sweats; no apical or cavitary radiographic change; personal history denied	Considered unlikely
Diabetic ketoacidosis	Hyperglycaemia; tachypnoea; nausea and epigastric pain	Tachypnoea explained by bronchospasm; no acetone odour or Kussmaul pattern; glucose 248 mg/dL only; rapid resolution of tachypnoea with bronchodilators	Excluded on clinical grounds; ketones and blood gases not measured
Metabolic acidosis of other cause	Extreme tachypnoea	Haemodynamically stable; no history of poisoning or renal failure; clinical course inconsistent	Considered unlikely

Stress Hyperglycaemia: Definition and Pathophysiology

Stress-induced hyperglycaemia is a physiological response to acute illness characterised by elevated blood glucose in patients with or without diabetes. It develops through complex interactions among catecholamines, cortisol and inflammatory mediators including cytokines, which together increase hepatic glucose production and induce peripheral insulin resistance. While

mild to moderate elevation may be protective during stress, prolonged or excessive hyperglycaemia exacerbates inflammation and oxidative stress and contributes to adverse outcomes.¹⁵

In this patient every element of that cascade was maximally activated. Severe hypoxaemia and an enormous work of breathing generated a sustained sympathoadrenal discharge; the infection provoked release of interleukin-6 and tumour necrosis factor- α ; and the endogenous cortisol response to critical illness added glucocorticoid-mediated gluconeogenesis and insulin antagonism. The resulting glucose of 248 mg/dL in a woman who had never been told she had diabetes is best interpreted as a quantitative index of the intensity of that response rather than as an incidental laboratory abnormality.¹⁵

The observation carries prognostic weight. In critically ill patients with sepsis, the stress hyperglycaemia ratio demonstrates a U-shaped relationship with 28-day and in-hospital mortality, with higher values significantly associated with increased risk of adverse events. Analyses of broader critical care populations have identified an inflection point at a ratio of approximately 0.96, and in a prospective study of 1,262 intensive care admissions relative hyperglycaemia — unlike absolute admission glucose — independently predicted in-hospital mortality after adjustment for a validated risk-of-death score, and did so across the whole glycaemic spectrum including patients with glycated haemoglobin below 6.5%.^{13,14,16}

It is precisely this literature that renders the absence of a glycated haemoglobin measurement in the present case a significant methodological deficiency, since without it the stress hyperglycaemia ratio cannot be computed and the patient's relative rather than absolute degree of hyperglycaemia cannot be established.^{15,16}

Iatrogenic Amplification: Corticosteroid and β_2 -Agonist Contributions

Two components of the mandatory treatment for acute severe asthma are themselves hyperglycaemic. A systematic review and meta-analysis of 118 randomised trials involving 17,113 participants, of whom 8,569 received systemic corticosteroid therapy, found a pooled prevalence of hyperglycaemia of 10% overall, rising to 22% in respiratory illness; the association with hyperglycaemia was significant in double-blind placebo-controlled parallel-arm studies with an

odds ratio of 2.13, and intravenous administration carried greater risk than oral therapy with an odds ratio of 2.39.²⁵

The dose administered in this case merits explicit scrutiny. Methylprednisolone 125 mg three times daily amounts to 375 mg per day, equivalent to approximately 470 mg of prednisone, whereas the GINA strategy report and the wider literature on acute asthma support doses in the region of 40–50 mg of prednisone equivalent per day for five to seven days, with no demonstrated advantage of higher doses. A Cochrane-based review of corticosteroids in obstructive airway exacerbations reported a number needed to harm of six for adverse drug effects and of seven for hyperglycaemia, with intravenous therapy conferring greater hyperglycaemic risk than oral, and a systematic review of short-course corticosteroids in children found moderate-certainty evidence of an increased risk of hyperglycaemia of 38 additional events per 1,000 patients treated.^{6,27,31}

The persistence of a fasting plasma glucose of 150 mg/dL on the morning after a single 8-unit dose of basal insulin is therefore entirely explicable: the insulin was competing against a suprathreshold and thrice-daily glucocorticoid load. This observation should not be read as an insulin failure but as evidence that the corticosteroid dose was the dominant hyperglycaemic driver, and that dose reduction towards guideline-recommended levels would have been the more physiological corrective.^{6,25}

The β_2 -agonist contribution is smaller but real. Salbutamol stimulates hepatic glycogenolysis and gluconeogenesis and drives potassium intracellularly, producing both hyperglycaemia and hypokalaemia; this patient received nebulised salbutamol every 20 minutes for the first hour and six-hourly thereafter. In a prospective study of 1,005 acutely ill children, admission hyperglycaemia was associated with oral corticosteroid therapy, inhaled corticosteroid therapy and inhaled β_2 -agonist therapy, and children with asthma were 5.58 times more likely to have admission hyperglycaemia and 7.86 times more likely to develop in-hospital hyperglycaemia than other acutely ill children — a striking demonstration that the asthma treatment package is intrinsically diabetogenic.²⁶

Table 16. Contributors to hyperglycaemia in this patient

Contributor	Mechanism	Present in this case
Catecholamine surge	Hepatic glycogenolysis and gluconeogenesis; inhibition of insulin secretion; peripheral insulin resistance	Severe hypoxaemia, extreme work of breathing, agitation, heart rate 116/min
Endogenous cortisol	Gluconeogenesis; insulin antagonism; increased proteolysis and lipolysis	Sepsis with organ dysfunction
Inflammatory cytokines	IL-6 and TNF- α impair insulin receptor signalling	Community-acquired pneumonia with sepsis
Exogenous corticosteroid	Potent, dose-dependent post-prandial hyperglycaemia; intravenous route higher risk than oral	Methylprednisolone 125 mg IV three times daily (375 mg/day)
β_2 -agonist therapy	Glycogenolysis and gluconeogenesis; intracellular potassium shift	Nebulised salbutamol every 20 min for 1 h, then every 6 h
Fluoroquinolone	Recognised association with dysglycaemia (both hyper- and hypoglycaemia)	Levofloxacin 750 mg daily
Possible undiagnosed diabetes	Pre-existing β -cell dysfunction unmasked by acute illness	Cannot be excluded — HbA _{1c} not measured

HbA_{1c} = glycated haemoglobin; IL-6 = interleukin-6; IV = intravenous; TNF- α = tumour necrosis factor-alpha.^{25,26}

How Hyperglycaemia Aggravates Dyspnoea and Infection

The clinical instruction to control glucose in this setting rests on the proposition that hyperglycaemia is causally harmful rather than merely correlated with severity. Several mechanisms, each supported by evidence in respiratory infection, converge on the respiratory system.²⁰

Impairment of innate immunity is the best characterised. Hyperglycaemia negatively affects both innate and adaptive immune responses, which may reduce bacterial clearance and increase mortality from infection; this effect extends to patients without diabetes, in whom admission hyperglycaemia is an independent risk factor for higher complication rates, longer length of stay and higher mortality in community-acquired pneumonia.²⁴

A second mechanism operates directly within the airway. When plasma glucose rises above the threshold at which glucose crosses the airway epithelium, glucose concentration in airway surface liquid increases, providing substrate for bacterial proliferation. Coupled with osmotic diuresis, relative hypovolaemia and consequent inspissation of secretions, this predicts precisely the clinical feature reported by this patient — sputum that was present but tenacious and difficult to expectorate — and provides the rationale for the mucolytic prescribed.^{20,24}

Third, hyperglycaemia amplifies rather than dampens the inflammatory response. In 724 non-diabetic patients with confirmed community-acquired pneumonia, moderate and severe hyperglycaemia were associated with an increased risk of an adverse clinical course, with an adjusted odds ratio of 3.0 for glucose above 11 mmol/L, and with higher C-reactive protein, leucocyte and pro-adrenomedullin concentrations across seven days of follow-up. Prolonged hyperglycaemia in diabetic and non-diabetic patients alike was similarly associated with an adverse course.²⁰

Fourth, the metabolic consequences reach the respiratory pump itself. Osmotic diuresis, together with the intracellular potassium shift produced by β_2 -agonists and insulin, promotes hypokalaemia, hypomagnesaemia and hypophosphataemia, each of which impairs diaphragmatic and intercostal muscle contractility in a patient whose respiratory muscles are already working at the limit of their endurance. Insulin resistance further favours a catabolic state unfavourable to the maintenance of respiratory muscle mass during a prolonged exacerbation.¹⁵

Fifth, at the level of the airway itself, metabolic dysfunction appears to influence asthma activity directly. In a five-year retrospective cohort of 108,219 patients with asthma, an elevated triglyceride–glucose index — a marker of insulin resistance — conferred a 6% higher hazard of severe asthma exacerbation independent of eosinophil count, smoking, obesity and treatment intensity; and in an obese asthma cohort of 5,722 adults, exacerbation rates were 27% higher in those with glycated haemoglobin in the prediabetes range and 33% higher in those in the diabetes range compared with normal values.^{28,29}

The corollary is a self-reinforcing loop. Hypoxaemia and the work of breathing drive catecholamine and cortisol release; these raise glucose; hyperglycaemia impairs bacterial clearance, thickens secretions, amplifies cytokine release and weakens respiratory muscles; and the resulting deterioration in gas exchange intensifies the stress response that began the cycle. Interrupting this loop pharmacologically, at the point where it is most readily accessible, is the conceptual justification for early insulin.^{15,20}

Table 17. Mechanisms linking hyperglycaemia to respiratory deterioration

Mechanism	Pathophysiological effect	Clinical correlate in this patient
Impaired innate immunity	Reduced neutrophil chemotaxis, phagocytosis and oxidative burst; impaired bacterial clearance	Sepsis despite a normal circulating leucocyte count
Elevated airway surface glucose	Substrate for bacterial proliferation in the airway lumen	Purulent sputum with a bronchitic radiographic pattern
Amplified cytokine response	Higher CRP, leucocyte and pro-adrenomedullin concentrations; adverse clinical course	Systemic illness with organ dysfunction
Osmotic diuresis and dehydration	Reduced airway surface liquid; increased sputum viscosity;	Tenacious sputum difficult to expectorate; dry lips

	impaired mucociliary clearance	
Electrolyte depletion	Hypokalaemia, hypomagnesaemia and hypophosphataemia impair respiratory muscle contractility	Respiratory rate 62/min with accessory muscle recruitment and exhaustion
Insulin resistance and catabolism	Loss of respiratory muscle mass and endurance during prolonged exacerbation	Impending respiratory failure
Metabolic effect on airway	Insulin resistance independently associated with severe exacerbation risk	Recurrent exacerbations with two treatment episodes in the preceding four months

CRP = C-reactive protein.^{20,24,28,29}

Rationale for Insulin as the Glucose-Lowering Agent of Choice

The American Diabetes Association recommends that insulin therapy be initiated for treatment of persistent hyperglycaemia starting at a threshold of 180 mg/dL, and that a glycaemic goal of 140–180 mg/dL be adopted for most critically ill and 100–180 mg/dL for most non-critically ill hospitalised individuals. The admission glucose of 248 mg/dL was substantially above that threshold, and the decision to treat rather than observe was therefore guideline-concordant.⁷

Insulin, rather than any oral agent, is the appropriate drug in this context for four reasons. It acts rapidly and can be titrated within hours, which no oral agent can match. It carries no organ-specific contraindication in sepsis, whereas metformin is hazardous where tissue hypoperfusion may occur, sulfonylureas produce unpredictable and prolonged hypoglycaemia in patients with variable oral intake, and sodium–glucose cotransporter-2 inhibitors may precipitate euglycaemic ketoacidosis in the setting of acute illness and reduced carbohydrate intake. It exerts anti-inflammatory actions independent of glucose lowering. And it is the only agent for which inpatient efficacy and safety are supported by an extensive guideline-endorsed evidence base. A review of international practice acknowledges that dipeptidyl peptidase-4 inhibitors and glucagon-like

peptide-1 receptor agonists have a growing role in selected stable non-critically ill patients with type 2 diabetes and mild hyperglycaemia, but that population is fundamentally different from the acutely septic, corticosteroid-loaded patient described here.^{7,36}

The specific regimen chosen — basal insulin glargine 8 units at night, approximately 0.14 units per kilogram per day — also merits examination. The Endocrine Society advises scheduled insulin rather than correctional (sliding-scale) insulin alone, since correctional insulin as sole therapy is associated with higher mean daily glucose. A randomised trial of 215 non-intensive-care patients with type 2 diabetes on basal–bolus regimens showed that less intensive supplemental sliding-scale correction was non-inferior for mean daily glucose where hyperglycaemia was moderate, supporting a strategy in which the scheduled component carries the therapeutic burden. Retrospective data from 25,186 admissions further indicate that dose increments of 44–100% of the total daily dose were significantly more likely to achieve a mean glucose of 160 mg/dL or below without subsequent hypoglycaemia than the conventional 10–20% increment, suggesting that inpatient insulin is frequently titrated too timidly.^{10,33,35}

A conservative starting dose was nevertheless defensible in a patient with no previous insulin exposure, no established diagnosis of diabetes mellitus and uncertain oral intake during severe dyspnoea, in whom the consequences of hypoglycaemia in the presence of agitation and altered consciousness would have been difficult to detect. The correct criticism is not of the initial dose but of the absence of a structured titration plan and of adequate glucose monitoring thereafter.^{7,10}

One further pharmacological refinement deserves comment. Where hyperglycaemia is predominantly glucocorticoid-driven and the glucocorticoid is administered in divided doses through the day, the glycaemic excursion is chiefly post-prandial and afternoon-predominant, and a once-daily long-acting basal analogue given at night is imperfectly matched to it. The Endocrine Society systematic review found, albeit with very low certainty, that in hospitalised adults receiving glucocorticoids a combination of neutral protamine Hagedorn insulin with a basal–bolus regimen achieved lower mean blood glucose than a basal–bolus regimen alone. In a patient receiving methylprednisolone three times daily, the addition of intermediate-acting insulin timed to the

corticosteroid, or of prandial and correction components, would have been mechanistically more appropriate than basal insulin alone.^{10,11}

Table 18. Comparison of glucose-lowering options in the acutely septic, corticosteroid-treated patient

Agent class	Suitability in this case	Basis
Insulin (basal ± correction)	Appropriate — agent of choice	Rapidly titratable; no organ-specific contraindication; guideline-endorsed for inpatient hyperglycaemia ≥ 180 mg/dL; anti-inflammatory properties
Sliding-scale insulin alone	Not recommended as sole therapy	Associated with higher mean daily glucose than scheduled regimens; advised against in guidelines
Metformin	Contraindicated acutely	Risk of lactic acidosis where tissue hypoperfusion, sepsis or unknown renal function is present
Sulfonylureas	Unsuitable	Prolonged unpredictable hypoglycaemia with variable oral intake during severe dyspnoea
SGLT2 inhibitors	Unsuitable	Risk of euglycaemic ketoacidosis in acute illness with reduced carbohydrate intake
DPP-4 inhibitors / GLP-1 receptor agonists	Not appropriate in this presentation	Emerging inpatient role limited to stable non-critically ill patients with type 2 diabetes and mild hyperglycaemia

DPP-4 = dipeptidyl peptidase-4; GLP-1 = glucagon-like peptide-1; SGLT2 = sodium–glucose cotransporter-2.^{7,10,36}

Glycaemic Targets: Why Not Tighter?

The history of inpatient glycaemic control is a cautionary one. The single-centre Leuven trial of intensive insulin therapy targeting 80–110 mg/dL in a predominantly surgical intensive care population reported a substantial mortality reduction and led to widespread adoption of tight control. That enthusiasm was decisively tempered by the NICE-SUGAR trial, in which 6,104 medical and surgical intensive care patients were randomised to intensive control targeting 81–108 mg/dL or to conventional control targeting 180 mg/dL or below: 90-day mortality was higher in the intensive arm and severe hypoglycaemia was far more frequent.^{8,9}

The contemporary consensus that emerged from this literature — target 140–180 mg/dL in critical illness and 100–180 mg/dL in non-critical illness — is precisely the range implied by the management of this patient, whose glucose fell from 248 mg/dL to 150 mg/dL without any hypoglycaemic episode. A 2025 systematic review of ten clinical practice guidelines found that eight recommended an upper glucose target of 180 mg/dL, five with a lower limit of 100 mg/dL and three of 140 mg/dL, with agreement on capillary glucose monitoring and on basal–bolus insulin, but with continuing disagreement on targets and on discharge transition. It should also be acknowledged that a large real-world analysis of 10,120 non-critically ill admissions found that implementing a basal–bolus protocol reduced both hyperglycaemia and hypoglycaemia without improving short-term clinical outcomes except in the small subgroup with newly diagnosed diabetes — a subgroup to which this patient may well belong.^{7,12}

Interpreting the Fasting Plasma Glucose of 150 mg/dL

The fasting value obtained on the second day is diagnostically ambiguous and should be presented as such. It exceeds the diagnostic threshold for diabetes mellitus of 126 mg/dL, but a single fasting measurement obtained during acute illness and during high-dose corticosteroid therapy cannot establish that diagnosis, and confirmation on a separate day in the stable state, or a glycated haemoglobin, would be required.⁷

The distinction is far from academic. Among 1,961 adults with community-acquired pneumonia in a European multicentre cohort, 15% had known diabetes; among the remainder, 5.0% had undiagnosed diabetes and 37.5% had prediabetes. Male sex, body mass index of 25 kg/m² or

1115

more, and hyperglycaemia at admission were associated with undiagnosed diabetes, with an odds ratio of 2.93 for admission glucose of 6–11 mmol/L and of 44.76 for glucose of 11 mmol/L or above. Critically, patients with undiagnosed diabetes had a 180-day mortality of 12.1% compared with 3.8% in those without diabetes.²¹

This patient's admission glucose of 13.8 mmol/L falls into the highest of those categories. The probability that she has previously undiagnosed diabetes or prediabetes is therefore appreciable, and the instruction to repeat fasting glucose at outpatient follow-up was appropriate — although a glycated haemoglobin, which is unaffected by acute illness and would simultaneously have permitted retrospective calculation of the stress hyperglycaemia ratio, would have been the more informative test.^{15,21}

Clinical Outcome in the Context of the Published Evidence

Comparison of this patient's course with published cohorts places the outcome in perspective. Non-diabetic patients admitted with community-acquired pneumonia and hyperglycaemia have been shown to follow a more severe clinical course than patients with known diabetes, with more frequent intensive care admission, greater use of invasive mechanical ventilation, later attainment of clinical stability and longer hospital stay. Against that background, a four-day admission concluding with room-air saturation of 95%, a clear chest and no requirement for ventilatory support represents a favourable trajectory.²³

Causal attribution must nevertheless remain restrained. The recovery reflects the combined effect of oxygen, bronchodilators, corticosteroid, magnesium, antimicrobials and insulin, delivered concurrently in a single patient. It is not possible to isolate the contribution of glycaemic control, and the observed sequence is consistent with, but does not demonstrate, the working hypotheses set out in Section 1.6.

Table 19. Summary of key evidence informing the management of this case

Study / guideline	Design and population	Principal finding relevant to this case	Ref.
Sepsis-3	International expert	Sepsis defined by organ dysfunction	1

consensus definitions	task force	(SOFA increase ≥ 2); SIRS criteria including leucocytosis abandoned	
Surviving Sepsis Campaign 2021	International guideline, 93 statements	Immediate empirical antimicrobials where sepsis likelihood is high	2
ATS/IDSA CAP guideline 2019; ATS update 2026	GRADE-based clinical practice guidelines	Severity-based empirical regimens; radiographic diagnosis with severity assessment	3,4
Lim et al., Thorax 2003	Derivation and validation, 1,068 patients	CURB-65: 30-day mortality 3 % at score 2 rising to 17 % at score 3	5
GINA strategy report	International asthma strategy	Criteria for severe and life-threatening exacerbation; silent chest as a life-threatening sign	6
ADA Standards of Care 2026	Annual clinical practice recommendations	Insulin from glucose ≥ 180 mg/dL; targets 140–180 (critical) and 100–180 mg/dL (non-critical)	7
NICE-SUGAR, NEJM 2009	RCT, 6,104 ICU patients	Intensive control (81–108 mg/dL) increased 90-day mortality and severe hypoglycaemia	8
Korytkowski et al., JCEM 2022	Endocrine Society guideline	Scheduled basal insulin preferred to sliding-scale monotherapy; guidance on glucocorticoid-associated hyperglycaemia	10
McAlister et al., Diabetes Care 2005	Prospective cohort, 2,471 CAP patients	Admission glucose > 11 mmol/L: 73 % higher mortality risk, 52 % higher complication risk	17

Lepper et al., BMJ 2012 (CAPNETZ)	Multicentre cohort, 6,891 CAP patients	Acute hyperglycaemia independently predicted 28- and 90-day mortality in non-diabetic patients	18
Schuetz et al., Diabetologia 2014	Observational cohort, 880 CAP patients	Hyperglycaemia in non-diabetic patients: adjusted OR 3.0 for adverse course; higher inflammatory markers	20
Jensen et al., CID 2017	Prospective cohort, 1,961 CAP patients	5.0 % undiagnosed diabetes, 37.5 % prediabetes; 180-day mortality 12.1 % vs 3.8 %	21
Yan et al., Cardiovasc Diabetol 2024	Retrospective cohort, 2,312 septic ICU patients	U-shaped relationship between stress hyperglycaemia ratio and 28-day mortality	13
Lee et al., Crit Care Med 2020	Prospective study, 1,262 ICU patients	Relative, not absolute, hyperglycaemia independently predicted in-hospital mortality	16
Kulkarni et al., BMJ Open 2022	Systematic review, 118 RCTs	Hyperglycaemia in 10 % of corticosteroid arms, 22 % in respiratory illness; IV > oral risk	25
Korakas et al., J Clin Med 2022	Prospective study, 1,005 acutely ill children	Asthma increased odds of admission and in-hospital hyperglycaemia 5.58- and 7.86-fold	26
Staggers et al., JACI Pract 2023	Retrospective cohort, 108,219 asthma patients	Elevated triglyceride–glucose index: 6 % higher hazard of severe exacerbation	29

ADA = American Diabetes Association; ATS = American Thoracic Society; CAP = community-acquired pneumonia; GINA = Global Initiative for Asthma; ICU = intensive care unit; IDSA =

Infectious Diseases Society of America; OR = odds ratio; RCT = randomised controlled trial; SIRS = systemic inflammatory response syndrome; SOFA = Sequential Organ Failure Assessment.

Adjunctive Therapies and Comorbid Findings

Magnesium sulphate

Intravenous magnesium sulphate was administered as a single 2 g infusion over two hours. GINA supports intravenous magnesium in moderate or severe exacerbations while explicitly no longer recommending nebulised magnesium in adults, adolescents or children. Magnesium relaxes bronchial smooth muscle by antagonising calcium influx and possesses anti-inflammatory properties. Its administration was therefore appropriate, and its potential to lower serum potassium alongside β_2 -agonist and insulin therapy reinforces the argument that electrolytes should have been monitored during this admission.⁶

Mucolytic and proton-pump inhibitor therapy

N-acetylcysteine was prescribed for sputum described as present but difficult to expectorate, a symptom that the hyperglycaemia-related mechanisms described above would be expected to aggravate. Omeprazole addressed both the dyspeptic symptoms and the gastroprotective requirement created by high-dose systemic corticosteroid therapy, a consideration reinforced by the finding, in a systematic review of short-course corticosteroids, of low-certainty evidence for an increased risk of gastrointestinal bleeding of 13 additional events per 1,000 patients treated.²⁷

Cardiovascular findings

The combination of radiographic cardiomegaly, an irregular first and second heart sound, possible left atrial abnormality on electrocardiography, borderline first-degree atrioventricular block, aortic atherosclerosis, hypertension and a remote history of valvular disease and of ischaemic stroke describes a patient at appreciable cardiovascular risk who has been lost to specialist follow-up on two separate occasions. Although acute decompensated heart failure was reasonably excluded during this admission by the absence of raised jugular venous pressure, crackles, peripheral oedema and radiographic pulmonary congestion, transthoracic echocardiography and formal rhythm assessment remain indicated. In a patient with a previous stroke and an irregular pulse, the exclusion of atrial fibrillation carries direct implications for anticoagulation.

CONCLUSION AND RECOMMENDATIONS

Conclusion

This report describes a 58-year-old woman with no known diabetes mellitus who presented with sepsis secondary to community-acquired pneumonia complicated by impending respiratory failure and a concurrent acute severe asthma exacerbation with life-threatening features, in whom the single most abnormal admission laboratory value was a random blood glucose of 248 mg/dL against an entirely normal complete blood count. She recovered within four days on high-flow oxygen, empirical antimicrobial therapy, systemic and inhaled corticosteroid, repeated bronchodilator nebulisation, intravenous magnesium sulphate and basal insulin, without requiring invasive or non-invasive ventilation and without any hypoglycaemic event.^{1,6,7}

Four conclusions follow. First, sepsis must be diagnosed by organ dysfunction rather than by leucocytosis; the normal white cell count in this patient was a distractor, and adherence to the Sepsis-3 framework together with the Surviving Sepsis Campaign recommendation for immediate empirical antimicrobial therapy was the decision that protected her.^{1,2}

Second, the chest radiograph in a patient with asthma is characteristically non-specific, and neither the presence of a bronchitic pattern nor the absence of lobar consolidation resolves the question of whether pneumonia is present. Radiographic findings in this population must be interpreted alongside the history, the physical examination and the severity scores, and the supine anteroposterior technique further limits both parenchymal and cardiothoracic interpretation.^{3,6}

Third, stress hyperglycaemia in this setting is triply determined — by the endogenous neuroendocrine and cytokine response to hypoxaemia and infection, by high-dose systemic corticosteroid therapy, and by repeated β_2 -agonist administration — and it is not benign. Elevated admission glucose in non-diabetic patients with community-acquired pneumonia independently predicts complications and death, and the mechanisms by which it does so, from impaired neutrophil function to increased sputum viscosity and respiratory muscle weakness, act directly on the respiratory system.^{17,18,20,25}

Fourth, insulin is the correct therapeutic instrument in this context. It is rapidly titratable, free of the organ-specific hazards that disqualify oral agents in sepsis, and supported by guideline

1120

recommendations to initiate therapy above 180 mg/dL while targeting 100–180 mg/dL in non-critically ill patients — a target range whose upper boundary was defined by the harm demonstrated with intensive control in the NICE-SUGAR trial. Measuring blood glucose in every severely breathless patient, irrespective of diabetes history, is a low-cost intervention with a plausible and evidence-supported effect on outcome, and it deserves to be treated as an integral part of the emergency assessment of dyspnoea rather than as an incidental finding.^{7,8}

Recommendations

For clinical practice

1. Measure random or capillary blood glucose in every patient presenting with severe dyspnoea, regardless of any stated history of diabetes mellitus, and record the result alongside the vital signs.^{7,17}
2. Add glycated haemoglobin whenever admission hyperglycaemia is detected in a patient not known to have diabetes, so that stress hyperglycaemia can be distinguished from undiagnosed diabetes and the stress hyperglycaemia ratio can be calculated.^{15,21}
3. Initiate scheduled insulin when glucose persistently exceeds 180 mg/dL, targeting 100–180 mg/dL in non-critically ill patients, and avoid sliding-scale insulin as sole therapy.^{7,10}
4. Where hyperglycaemia is predominantly glucocorticoid-driven and the steroid is given in divided doses, consider adding intermediate-acting insulin timed to the corticosteroid, or prandial and correction components, rather than relying on a nocturnal basal analogue alone.^{10,11}
5. Prescribe systemic corticosteroid for acute asthma at guideline-recommended doses rather than at suprathreshold doses, since higher doses confer no demonstrated advantage while measurably increasing hyperglycaemia and other adverse effects.^{6,25,27}
6. Do not withhold or delay empirical antimicrobial therapy in a patient with suspected infection and organ dysfunction because the leucocyte count is normal.^{1,2}
7. Monitor serum potassium, magnesium and phosphate when β_2 -agonists, insulin, corticosteroid and magnesium sulphate are administered concurrently, given their combined effect on respiratory muscle function.⁶

8. Interpret the silent chest as a life-threatening sign of severely reduced airflow rather than as evidence of improvement, and reassess speech, mental state, respiratory rate and wheeze audibility at short intervals.⁶
9. Arrange definitive glycaemic reassessment after recovery, using glycated haemoglobin or a repeat fasting glucose four to twelve weeks after discharge, and ensure that the patient understands why this matters.^{7,21}
10. Complete the outstanding cardiovascular evaluation in patients with radiographic cardiomegaly, irregular heart sounds and a history of valvular disease or stroke, since the exclusion of atrial fibrillation has direct therapeutic implications.

For institutions and health systems

1. Incorporate point-of-care glucose measurement into emergency department triage protocols for patients presenting with dyspnoea, chest pain or altered consciousness.⁷
2. Develop and implement a standardised, nurse-driven inpatient insulin protocol with predefined correction thresholds and scheduled monitoring; a multidisciplinary initiative of this kind reduced corticosteroid-induced hyperglycaemia from 48 % to 26 % and shortened intensive care stay by 0.8 days without increasing hypoglycaemia.³⁷
3. Prioritise the availability of arterial blood gas analysis, basic renal chemistry, glycated haemoglobin and blood culture in secondary-level hospitals receiving referrals for severe dyspnoea, since their absence directly degraded severity scoring in this case.^{1,5}
4. Establish an antimicrobial stewardship pathway for community-acquired pneumonia that guides both initial regimen selection and de-escalation, including at the point of discharge.^{3,4}
5. Ensure structured handover to outpatient services for patients discharged with newly detected hyperglycaemia, uncontrolled asthma or unevaluated cardiac findings, since this patient had already been lost to two specialist services before this admission.^{12,21}

For research

1. Conduct prospective cohort studies characterising the prevalence, magnitude and prognostic significance of stress hyperglycaemia specifically in patients with overlapping obstructive

airway disease and lower respiratory tract infection, a population under-represented in existing cohorts.^{13,17}

2. Evaluate the stress hyperglycaemia ratio as a triage and prognostic instrument in emergency departments in low- and middle-income settings, where it may add discriminatory value at minimal cost.^{15,16}
3. Undertake randomised comparisons of insulin regimens — basal alone, basal plus correction, and neutral protamine Hagedorn timed to corticosteroid administration — in patients receiving high-dose systemic corticosteroid for acute asthma, an area where current evidence is of very low certainty.^{10,11}
4. Determine the optimal systemic corticosteroid dose and route for acute severe asthma with explicit measurement of glycaemic adverse effects as a co-primary outcome alongside efficacy.^{25,27}
5. Establish the incidence of subsequently confirmed diabetes mellitus among patients discharged after an episode of stress hyperglycaemia complicating a respiratory admission, and test whether structured post-discharge screening improves long-term outcome.²¹

DECLARATIONS

Informed consent

Written informed consent was obtained from the patient for the publication of this case report and the accompanying anonymised clinical images. All personal identifiers have been removed from the text and from all figures.

Ethical approval

Case reports describing routine clinical care of a single patient, published with the patient's written consent and without identifying information, are exempt from formal institutional ethics committee review under the prevailing institutional policy. Institutional permission for publication was obtained.

Author contributions

All authors contributed to the clinical management of the patient, to the conception and design of the report, to the acquisition and interpretation of the clinical data, and to the drafting and critical

1123

revision of the manuscript. All authors have read and approved the final version and agree to be accountable for all aspects of the work.

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Conflicts of interest

The authors declare that they have no competing interests.

Data availability

All data relevant to this report are contained within the manuscript and its figures. Further anonymised clinical details are available from the corresponding author on reasonable request, subject to institutional approval.

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Abbreviations

ADA, American Diabetes Association; ATS, American Thoracic Society; BMI, body mass index; CAP, community-acquired pneumonia; CRP, C-reactive protein; CURB-65, Confusion–Urea–Respiratory rate–Blood pressure–age 65; DPP-4, dipeptidyl peptidase-4; ED, emergency department; FiO₂, fraction of inspired oxygen; FPG, fasting plasma glucose; GCS, Glasgow Coma Scale; GINA, Global Initiative for Asthma; GLP-1, glucagon-like peptide-1; HbA_{1c}, glycated haemoglobin; ICU, intensive care unit; IDSA, Infectious Diseases Society of America; IL-6, interleukin-6; MAP, mean arterial pressure; NRM, non-rebreather mask; PaCO₂, arterial partial pressure of carbon dioxide; PaO₂, arterial partial pressure of oxygen; PEF, peak expiratory flow; qSOFA, quick Sequential Organ Failure Assessment; RBG, random blood glucose; SGLT2, sodium–glucose cotransporter-2; SHR, stress hyperglycaemia ratio; SIRS, systemic inflammatory response syndrome; SOFA, Sequential Organ Failure Assessment; SpO₂, peripheral oxygen saturation; TNF- α , tumour necrosis factor-alpha.

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