

A Physician Asks the Lab to Send Cyanide to the Emergency Room - What Is Your Guess?

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A 33 years old male was brought to ER with complaints of fever and difficulty in breathing. Before his current presentation, he had no known medical problems. The patient's pulse oximetry reading is SpO₂ 98% on air. However, his measured pO₂ in room air was 30 mmHg. Despite this evidence of life-threatening hypoxemia, he had no pulmonary signs, and

nothing abnormal was detected on his chest X-ray film. His 2D echo showed an average ejection fraction of 60%, with no evidence of diastolic dysfunction or valvular abnormalities. Thus, no obvious etiology for hypoxemia on ABG was evident, and the results obtained were inconsistent with the patient's current clinical presentation.

What are the possible explanations for the discrepancy between the pulse oximetry reading and the oxygen saturation on the arterial blood gas?

Possible explanations for 'pseudo-hypoxemia' include

- Equipment failure
 - faulty pulse oximeter
 - faulty blood gas analyzer
 - Venous blood sample
 - A blood sample is taken from a site affected by localized hypoxemia, e.g., ischaemic limb
 - Excessive oxygen consumption following blood sample collection (e.g., massive leukocytosis or thrombocytosis)
- Dyshemoglobinemia can also cause a discrepancy between SpO₂ and SaO₂ but will not cause the decrease in measured PaO₂ as seen in this case.
- In carbon monoxide poisoning, SpO₂ will read in the 90s despite high levels of COHb — but the PaO₂ should still be high.
 - In methemoglobinemia, the SpO₂ plateaus at about 86% with increasing levels of MetHb, but again the PaO₂ will not be decreased.

What would other blood tests be beneficial?

The full blood count. (Fig 1)

How to describe the blood test result shown?

The key findings are

----- MMC Diagnostics -----

Blood Gas Report

1267 11.00 Nov 23 2021

Pt ID 1645 / 21

Measured 37.0°C

pH 7.44

pCO₂ 34.4 mm Hg

pO₂ 28.2 mm Hg

Na 135

K 4.0

CL 100

Corrected 38.6°C

pH 7.439

pCO₂ 47.6 mm Hg

pO₂ 23.5 mm Hg

Calculated Data

HCO₃ act 28.2 mmol / L

HCO₃ std 22.5 mmol / L

BE 5.6 mmol / L

O₂ Sat 38.3 %

Entered Data

Temp 38.6 °C

ctHb 8.5 g/dl

FiO₂ 0.21 %

- Massive leukocytosis, due to a large number of blasts
- Normocytic anemia, mild macrocytosis, and increased RDW
- Thrombocytopenia

What is the likely underlying diagnosis?

- Acute myeloid leukemia
- Hyperleukocytosis is almost always due to hematological malignancy.
- The anemia and thrombocytopenia are consistent with bone marrow failure.

What other complications is this patient at risk of?

- Hyperviscosity syndrome due to massive leukocytosis (e.g., altered mental state, stroke,

pulmonary leukostasis, renal insufficiency, and priapism)

- Bleeding and disseminated intravascular coagulation (DIC)
- Infections due to immune suppression
- Syncope and ischemia due to anemia (decreased oxygen delivery)
- Acute respiratory distress syndrome (ARDS) due to hematopoietic mediators, leukostasis, and leukemic cell lysis/pneumopathy
- Complications of treatment (e.g., chemotherapy side effects, tumor lysis syndrome, transfusion reactions, bone marrow transplant)

What is Spurious hypoxemia?

Spurious hypoxemia is a disparity

LABORATORY INVESTIGATION REPORT

SPECIMEN	INVESTIGATION (METHOD)	RESULT	UNIT	REF. INTERVAL
HEMATOLOGY				
	Blood Grouping	" A1 "		
	Rh Typing	Positive		
COMPLETE BLOOD COUNT				
EDTA W. Blood	Hemoglobin	7.18	gms%	13.0 - 16.0
EDTA W. Blood	Total RBC Count	2.48	Millions/cumr	4.00 - 5.00
EDTA W. Blood	PCV / HCT	22.4	%	41.00 - 50.00
EDTA W. Blood	Mean Corpuscular Volume (MCV)	90.3	fl	81.10 to 96.00
EDTA W. Blood	Mean Corpuscular Hemoglobin (MCH)	31.5	pg	27.20 to 33.20
EDTA W. Blood	Mean Corpuscular Hemoglobin Concentration(MCHC)	34.8	%	32 to 36
EDTA W. Blood	Total WBC Count	108.84	10 ³ Cells/cl	4.00 - 11.00
Differential WBC Count				
EDTA W. Blood	Neutrophils #	56.46	10 ³ /uL	2.0 to 7.0
EDTA W. Blood	Lymphocytes #	12.81	10 ³ /uL	1.0 to 3.0
EDTA W. Blood	Monocytes#	38.72	10 ³ /uL	0.1 to 0.8
EDTA W. Blood	Eosinophils #	0.00	10 ³ /uL	0.02 to 0.50
EDTA W. Blood	Basophils #	0.85	10 ³ /uL	0.02 to 0.10
EDTA W. Blood	Neutrophils	51.8	%	55.0 - 75.0
EDTA W. Blood	Lymphocytes	11.8	%	15.0 - 30.0
EDTA W. Blood	Eosinophils	0.0	%	1.0 to 5.0
EDTA W. Blood	Monocytes	35.6	%	2.00 to 10.00
EDTA W. Blood	Basophils	0.8	%	Upto1
EDTA W. Blood	Platelet Count	35	10 ³ /Cu.mm	150.0 to 450.0
EDTA W. Blood	Mean Platelet Volume (MPV)	0.0	fl	6.5 to 12.0
EDTA W. Blood	Platelet Distribution Width (PDW)	0.0	fl	9.0 to 13.0

between measured and actual $pO_2(a)$ due to in vitro consumption of oxygen by blood cells (white cells and platelets). This phenomenon, known as "oxygen steal" or "leukocyte/platelet larceny," is suspected when there is no other obvious explanation for hypoxemia on ABG, especially in the setting of normal oxygen saturations by the pulse oximetry.

What is the pathophysiology of spurious hypoxemia?

The pathophysiology of spurious hypoxemia has been thought to involve oxygen consumption by increased numbers of leukocytes inside the syringes (leukocyte larceny) before analysis.

Why Spurious hypoxemia occurs?

It is associated more with an increase in the number of cellular elements like immature WBCs, platelets, and reticulocytes than with others like mature RBCs or mature WBCs. It has been postulated that immature cellular elements, especially leukemic cells and, more specifically, blast form of granulocytes, have the highest metabolic rate and consume oxygen faster than mature cellular elements. Mature RBCs have an anaerobic metabolism and thus, do not consume oxygen.

When to suspect Spurious hypoxemia?

When managing life-threatening patients, differentiating between true hypoxemia and spurious hypoxemia is a critical skill. Hence, spurious hypoxia is suspected when there is a mismatch between the oxygen saturation on ABG compared to continuous pulse oximeter readings in a patient with the background of hyperleukocytosis or thrombocytosis.

Does Spurious hypoxemia have any clinical significance?

- Yes, spurious hypoxemia may have deleterious consequences if not adequately recognized.
- First, PaO_2 may be used as criteria to initiate mechanical ventilation in patients with

hematological malignancies and clinical signs of respiratory distress. Coexisting conditions such as pain, anxiety, and cerebral leukostasis may also cause tachypnea, reinforcing the pivotal role of PaO_2 in clinical decision-making.

- Second, during mechanical ventilation, unrecognized spurious hypoxemia may trigger an inappropriate increase in FIO_2 and expose patients who are already prone to respiratory complications to additional pulmonary oxygen toxicity.

Does it occur only in Acuteleukemias?

Though classically described in the setting of acute leukemias, it has been reported in association with chronic leukemias.

How could the abnormal blood gas result be avoided?

For accurate results, rapid sample analysis is crucial in such circumstances, which can be obtained using Bedside point-of-care analyzers and continuous blood gas analysis. However, these gadgets are expensive, and hence, alternate methods are devised to overcome this problem. Abnormalities in PaO_2 , $PaCO_2$, and pH may be mitigated in the presence of massive leukocytosis by

- processing the sample rapidly
- transporting the sample on ice

How can KCN confirm this condition?

KCN is a metabolic inhibitor of respiration. The addition of KCN to blood gas samples prevents oxygen consumption by inhibiting leukocytes, thereby preventing the decrease in O_2 .

In addition to KCN, what other methods can be used to confirm this suspicion?

Cooximetry and pulse oximetry measure O_2 saturation.

How reliable is pulse oximetry to confirm this suspicion?

Pulse oximetry measures oxygen saturation of hemoglobin directly being unaffected by plasma oxygen tension. Hence, normal SpO₂ values confirm pseudohypoxaemia in such conditions and reliably guide oxygen therapy.

When KCN or Co-oximetry is not available, how to confirm this suspicion?

Using precooled syringe (kept in the freezer for 30 minutes and taken out just before drawing blood) rapidly reduces oxygen consumption by cells in the sampled blood.

Any other metabolic abnormalities noted with leukocyte larceny?

The massive leukocytosis can also result in **pseudohypoglycemia** due to ongoing glucose metabolism after a blood sample is collected.

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