Confusing Confusion

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Patient History | Physical Exam | Tests Ordered | Imaging

HPI
- 72-year-old male
- During the summer, he was found down in his home with vomitus on and around him
- GCS on EMS arrival was 6, improved to 14 upon arrival to ED
- Mental status waxing and waning throughout transport and after arrival to ED

ROS
- Unable to obtain due to confusion

PMHx
- Type 2 DM, Obstructive Sleep Apnea, GERD, HLD, HTN
- No recent surgery
- No known allergies
- Medications: metformin, dulaglutide, atorvastatin, metoprolol

Later in the ED, the patient was able to provide greater insight into events prior to EMS arrival:
Patient reported that he woke up and felt off, but went to go start his car to go out to breakfast like they normally do. The car was having issues starting, so he sat at the table to have breakfast at home instead. He reported that he was sitting at the table and had some abdominal pain, nausea, and a headache.

Vitals	
Blood Pressure	139/109 mmHg
Pulse	126 BPM
Temperature	36.9 ^{o}C
Respiration Rate	34 Breaths/min
Weight	77.1 kg
SpO_2	100%
BMI	29.18 kg/m^2

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Abnormal Physical Exam Findings

- Acute distress
- Tachycardic
- Tachypneic
- Pinpoint pupils
- Generalized abdominal tenderness, worse in lower quadrants
- Lethargic
- GCS eye: 4
- GCS verbal: 4
- GCS motor: 6
- Spinous process tenderness

Other Exam Findings

- Normal appearance of skin
- Normal breath sounds
- Normal heart sounds
- Abdomen flat, no guarding or rebounding
- Neck supple, no rigidity
- No lower extremity edema
- Sensation is intact
- No motor deficits or weakness

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Lab Abnormalities	Result Value	Reference Range
White Blood Cells	21.4 thou/cmm	4.0 – 10.5
Absolute Neutrophils	17.9 thou/cmm	1.8 – 7.8
Venous pH	7.15	7.31 – 7.41
Venous pCO_2	37 mmHg	41 – 51
Venous sO_2	56%	>75%
Venous Base Deficit	15.2 mmol/L	0.0 – 6.0
Glucose	510 mg/dL	65 – 99
Creatinine	1.41 mg/dL	0.53 – 1.30
Carbon Dioxide	12 mmol/L	21 – 31
Anion Gap	24	3 – 11
β-Hydroxybutyrate	1.39 mmol/L	<0.5
Lactate	12.7 mmol/L	0.5 – 2.2
Osmolality	317 mOsm/kg	300 – 900

HS Troponin

Hour 0	Hour 1	Hour 2	Reference Range
244 ng/L	963 ng/L	2,585 ng/L	<21

Other Tests:

- Acetone was Negative
- POCUS Echocardiogram found Global Hypokinesis

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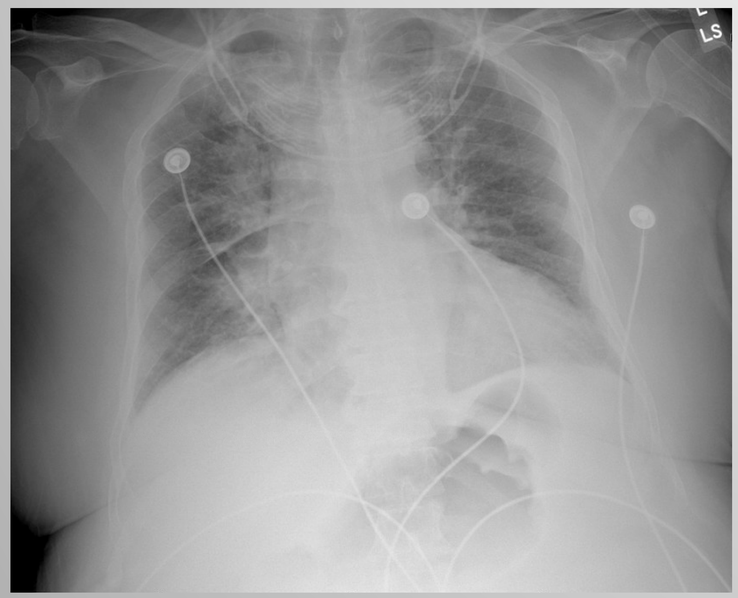
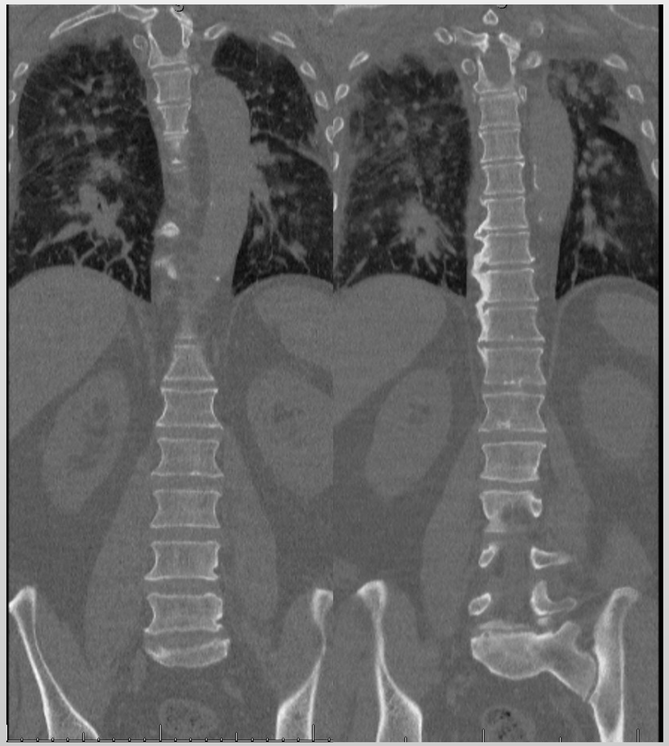
Chest X-Ray



CT Chest, Abdomen, Pelvis



CT Cervical Spine, Head

All imaging is clear of signs of trauma

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Final Diagnosis: Carbon Monoxide poisoning leading to NSTEMI and stress-induced hyperglycemia

Confirmatory tests	Result Value	Reference Range
Carboxyhemoglobin	34.1%	0.5 – 1.5
Methemoglobin	<0.7%	<1.5

Information obtained after initial presentation and workup

- Patient's wife was brought to ED later during a 2nd EMS call after collapsing in the house.
- EMS dispatched fire to the scene during 2nd call and found a CO level of 700 ppm inside the house.

Confusing Confusion- Diagnosis & Disposition

CO Level (PPM)	Effects
0	Normal O_2 level
9	Maximum permissible short-term exposure
10-24	Possible health effects with long-term exposure
25	Maximum permissible exposure for indoor car parks
50	Maximum permissible exposure in workplace
200	Fatigue and headache after 2-3 hours of exposure
400	Headaches within 1-2 hours, life threatening after 3 hours
800	**Headache, nausea, and dizziness after 45 minutes. Unconsciousness after 1 hour of exposure, death within 2-3 hours**
1,000	Loss of consciousness after 1 hour of exposure
1,600	Headache, nausea, and dizziness after 20 minutes of exposure, death within 1-2 hours.
3,200	Headache, nausea, and dizziness after 5-10 minutes. Collapse and unconsciousness after 30 minutes of exposure and death within 1 hour.
6,400	Death within 30 minutes
12,800	Death within 1-3 minutes of exposure

Once able to obtain the patient's name and connect him to his wife, more information about the events prior to EMS arrival came to light. The patient and his wife were out to dinner the night before and may have left the car running in the garage. When he attempted to start it in the morning, the patient had a massive CO exposure, due to the garage being a "hot zone." This is presumably why his symptoms were worse than his wife's.

Confusing Confusion- Diagnosis & Disposition

Treatment for CO poisoning

The patient and his wife were transferred to an outside hospital for hyperbaric oxygen treatment in the setting of severe CO poisoning.

Treatment for NSTEMI

After transferring to an outside hospital, the patient underwent a left heart catheterization. No coronary artery blockages were found.

- Both the patient and his wife had a complication-free hospital stay and were discharged home 48 hours post-transfer.
- The patient's stress-induced hyperglycemia had self-resolved by the time of his discharge, not requiring any specific treatments.

Confusing Confusion- Diagnosis & Disposition

CO Toxicity & Hyperbaric Therapy

- Given the unsuspecting time of year for a carbon monoxide poisoning it is important to recognize sources of CO such as automotive exhaust, methylene chloride inhalation, and smaller gasoline-powered motor exhaust. Use of these sources is not as restricted to colder months as heaters.
- The utility of hyperbaric therapy for a patient with CO poisoning can sometimes be in doubt. It is indicated for:
    - pregnant women with >20% CO levels
    - Anyone with >25% CO levels
    - Loss of consciousness (on scene or hospital)
    - Altered mental status
    - Signs of end organ dysfunction
    - Acute myocardial ischemia
- Our patient presented with 4 of the criteria.

Other Organ Dysfunction

- The NSTEMI is likely secondary to the effects of CO displacing oxygen.
- Myocardial tissue is highly sensitive to the relative ischemia from the CO, causing a type II NSTEMI. The patient's clean catheterization report further supports this notion as the NSTEMI etiology.
- With regards to the levels of hyperglycemia seen in a patient with otherwise relatively good glycemic control, hypoxemia leads to stress catecholamine release, causing spikes in blood sugar.
- The acidemia seen is due to the fact that hypoxemia decreases oxygen delivery and CO impairs oxidative phosphorylation, both leading to increased lactate production. The increase in lactate leads to the lactic acidosis, as was seen during initial presentation.

Questions?

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