1. A 65 year old female with a history of hyperlipidemia presents with unstable angina. She is medically managed and subsequently receives coronary catheterization. Her angina is under control but she is oliguric for the next 2 days in hospital. Her creatinine has doubled from her initial level on presentation and is continuing to rise by day 6. Which of the following findings is not likely to be seen?

a. Low serum eosinophils
b. Livedo reticularis
c. Pyuria
d. Confusion

Answer: a. Low serum eosinophils

There are two primary considerations for this patient’s AKI: contrast-induced nephropathy (CIN) and cholesterol emboli syndrome (CES). Both of these syndromes are the result of coronary catheterization, although their etiologies are very different. In the case of CIN, the contrast dye can cause a pre-renal AKI or ATN through direct nephrotoxicity and vasoconstriction of renal vasculature1. On the other hand, CES occurs when the catheter dislodges an atherosclerotic plaque (often when passing through the aorta) – releasing fragments that can occlude smaller vessels2.

It can be challenging to distinguish CIN and CES clinically. Often the key is to trend a patient’s creatinine. While it is not a firm rule, the creatinine will peak in the first 2-3 days of CIN before decreasing3. Conversely, in CES, the creatinine continues to increase in a step-wise fashion and can lead to chronic kidney disease and dialysis in the less fortunate3.

In this case, the patient’s creatinine has continued to rise by day 6, raising the concern of CES. Common findings of CES relate to tissue-ischemia from vascular occlusion by the cholesterol emboli. Ischemia in the peripheral tissues (often the feet) will lead to livedo reticularis.

Answer: c. Consideration for home oxygen

This is a typical presentation of Chronic Obstructive Pulmonary Disease (COPD). This is manifested by the patient’s PFTs revealing an obstructive lung disease with irreversibility and reduced gas-exchanged (manifested by the reduced DLCO)4. He also has classical symptoms of shortness of breath and exercise intolerance4. He is also demonstrating signs of cor pulmonale – pedal edema and an echocardiogram revealing right heart strain4.
Although he is being managed appropriately on an ICS + LABA (Advair) and an AC (Spiriva), there is room to improve this patient’s care. Specifically, the focus at this point should be reducing the frequency of COPD exacerbations and improve long-term mortality. There are 3 evidence-based strategies for improving mortality in COPD: supplemental home oxygen, lung reduction surgery & smoking cessation. Therefore, c) is the correct answer. As per the Canadian Thoracic Society Guidelines, continuous Oxygen therapy (keeping SPO2 greater than 90% for 18 hours per day) offers a mortality benefit in hypoxic patients. This is defined as patients with a resting partial pressure of less than 55mmHg on ABG or less than 60 mmHg in the presence of bilateral ankle edema, cor pulmonale or a hematocrit greater than 56%. This patient would likely be classified as having moderate COPD – a good candidate for home oxygen as long as he quits smoking.

3. A 55 year old man presents to the emergency department after being found unconscious in a puddle of his own vomit. His friend describes the vomitus as dark brown with chunks of blood in it. This patient has no history of alcohol abuse or liver disease but was recently in line for a TAAVI for aortic stenosis. His vitals are as follows: HR = 105, BP = 100/80, SpO2 = 96%, RR = 14, T° = 37.0. On examination, his GCS is 10 and he is diaphoretic with weak pulses and an III/IV crescendo-decrescendo systolic murmur in the RUSB – otherwise, his exam is unremarkable. What is the next best step in management?

a. Call GI for urgent endoscopy
b. Draw bloodwork then start blood transfusions with the according amount of packed RBCs
c. Start IV PPI and IV octreotide infusions
d. Initiate IV fluid resuscitation

Answer: d. Initiate IV fluid resuscitation

One consideration here is Heyde’s syndrome – a rare entity where the development of aortic stenosis is concurrent with vascular malformations in the GI tract. Although unconfirmed, some current theories posit that shear forces around the stenotic valve cause a form of Von Willenbrand’s Disease, leading to coagulation abnormalities that contribute to GI bleeding.

Diagnosing Heyde’s syndrome is not necessary in this case, however. The key is to recognize the GI bleed and stabilize the patient by managing its subsequent hemodynamic effects. This patient is in hypovolemic shock on the grounds of altered mental status, tachycardia and hypotension. Therefore, an urgent GI consult and endoscopy/colonoscopy (a) is inappropriate at this time until the patient is stabilized. In a similar vein, drawing bloodwork is important but the hemodynamic instability warrants prompt attention first – (b) is incorrect as well. Furthermore, while blood transfusions may be appropriate based on bloodwork, resuscitation must be started before the patient is appropriately cross and typed. Therefore, (d) is the correct answer: IV resuscitation with (likely with crystalloids) must be attempted early and aggressively to improve circulation. An UGIB secondary to PUD is a reasonable consideration here and this patient will likely receive IV PPI soon. However, there are no concerning physical exam findings or history suggestive of variceal bleeding/liver disease – IV octreotide would not be warranted in this case and (c) is therefore incorrect.

4. A 65 year old woman presents to the emergency room with a 1-day history of blue lips and shortness of breath. She has primary immunodeficiency syndrome, for which she is on dapsone for PCP prophylaxis. Her vital signs on admission are: HR = 109, BP = 104/78, SpO2 = 83%, RR = 18, T° = 36.8. Despite attempts to improve supplemental oxygen delivery, her SpO2 is unchanged. Her initial physical exam, CXR, ECG, and basic bloodwork are unremarkable. What is the next best choice in management?

a. Provision of a short-acting bronchodilator and/or loop diuretic
b. Intubation and mechanical ventilation
c. ABG
d. CT chest with pulmonary embolism protocols

Answer: c. ABG

This case is concerning for acquired methemoglobinemia because of her normal ABG, signs of peripheral cyanosis and low pulse oximetry despite attempts to improve oxygen delivery. When hemoglobin is oxidized from ferrous form to ferric form – either because of acquired or congenital causes – the affinity for oxygen is decreased. This is left-shift of the oxygen dissociation curve. Tissues are thereby deprived of oxygen, causing a spectrum of peripheral cyanosis to end-organ damage. The pulse oximetry in methemoglobinemia will usually be around 80-85%. Pulse oximetry devices only measure arterial haemoglobin absorbance at two wavelengths: 660nm and 940nm. The ratio of absorbance at these wavelengths yields the oxygen saturation value. An absorbance ratio of 0.43 corresponds to 100% saturation. Methemoglobin absorbs light equally at both wavelengths and causes pulse oximetry devices to misinterpret the patient’s oxygen saturation level.
wavelengths resulting in consistent SpO2 readings of about 85%-90%. This patient likely developed methemoglobinemia as a side-effect of dapsone.

The most appropriate early diagnostic tool in confirming suspicion of methemoglobinemia is an ABG revealing a characteristic chocolate-brown colour sample, a normal PaO2, and a co-oximetry demonstrating an abnormal percentage of methemoglobin (>1%). Even if methemoglobinemia is not on your radar, an ABG is the best choice in this situation because it illustrates oxygen delivery and supply at a tissue level to corroborate the pulse oximetry levels suggesting hypoxia. This is an easy test to perform and provides early and useful results to guide further diagnostic and management decisions.

In this situation, provision of a SABA and/or loop diuretic is unwarranted. There are no specific findings concerning for bronchospasm, tachypnea or respiratory distress to warrant trial of a bronchodilator. Similarly, there are no symptoms or signs of volume overload to warrant a loop diuretic. Intubation may be appropriate in the future if there is refractory hypoxemia and signs of peripheral cyanosis. However, it’s too early for this right now – this patient is maintaining her airway, is not tachypneic, and is reasonably stable despite her low SpO2. Consideration of a pulmonary embolism is fair given the complaints of SOB, signs of hypoxemia and tachycardia. A clinical decision rule should be applied here (such as Well’s or PERC), which she should score low on in combination with a normal CXR and ECG. Therefore, a CT chest with PE protocols may be reasonable down the road but not now – there are other diagnostic tools that are easier to use with more utility.

References: