Case Study

- A 70-year-old woman describes increasing exertional dyspnoea for the last 2 days and now has dyspnoea at rest. She has a history of hypertension for the last 5 years and a 35 pack-year smoking history, but no other established illnesses. Current medications are a diuretic daily for the last 3 years. She has been prescribed an ACE inhibitor one week ago but failed to collect the prescription. She reports that her right leg is painful, hot and red since she injured her leg during the recent flight from New Zealand. She denies any chest pain, palpitation nor sweating. However, she reports being febrile.

This scenario is for education purposes only and should not be taken as medical advice.
Note that this scenario was adapted from BMJ Best Practice and tailored for medical students' learning.
Asterisk (*) is used to provide further explanation in case you are someone who prefer to understand "WHY."

Examination: DRSABCDE

- A: Out of breath, but talks in full sentences
- B: RR: 30, O2 sat: 89% on RA, Bibasal fine crackles, symmetrical chest expansion, no wheeze or stridor.
- C: HR: 104 bpm, BP: 190/90, elevated JVP by 2cm, audible S3, no murmurs, extremities warm and wet, CRT < 2 secs. Bedside ECG shows LVH, but no signs of ACS such as ST elevation.
- D: **A**CVPU – Fully alert. PEARL. BSL 4.5mmol/L
- E: Her anterior side of right leg is swollen, red with orange-peel appearance and there are regional lymphadenopathy. Bilat pitting oedema up to ankle.
- **Q: What are the most pressing issues and how would you manage them?**

Initial management

- **Q. What are the most pressing issues and how would you manage them?**
  - B and C of ABCDE should be managed. E should be managed, but it is not the priority in ED setting*
  - B: RR: 30, O2 sat: 89% on RA, Bibasal fine crackles, symmetrical chest expansion, no wheeze or stridor.
    - Sitting Up
    - Provide oxygen aiming for O2>94% via Non-invasive ventilation such as CPAP or BiPAP
    - Bibasal crackles indicate acute pulmonary oedema. Give IV frusemide.
  - C: HR: 104 bpm, BP: 190/90, elevated JVP by 2cm, audible S3, no murmurs, extremities warm and wet, CRT < 2 secs. Bedside ECG shows LVH, but no signs of ACS such as ST elevation.
    - This patient is not hypotensive. Therefore, we can give nitrates to further manage acute pulmonary oedema.
    - Patient was warm and wet – she was volume overloaded but well perfused, so she would be benefited from vasodilated and diuretics.
  - Easy mnemonic for acute heart failure is L~~M~~NOP - L: Lasix (frusemide), N (nitrates), O (Oxygen), P (Positioning – sitting up). M stands for morphine, but not recommended anymore.

- **Q. In suspected acute heart failure, it is critical to urgently identify precipitant of acute heart failure which are ACS, Hypertensive emergency, arrhythmias, mechanical issues, PE (mneumonic: CHAMP). Unless these causes are corrected immediately, AHF is extremely life-threatening. What investigation would you order to find the trigger and confirm diagnosis of acute heart failure in this case?**

* *Findings at E* sound like cellulitis instead of DVT since DVT is more evident in calf where veins are located and DVT is unlikely to present with orange-peel appearance which indicates high volume overload in lymphatics from infection such as cellulitis. However, seeing her recent travel history, DVT still should be ruled out since it could have led to PE and PE is one of trigger for decompensated HF (explained in next slide regarding trigger of heart failure)

Investigation

- **Q. What investigation would you order to find the trigger of her acute HF and confirm diagnosis of acute heart failure in this case?**

1. Diagnostic workup to confirm AHF (according to modified Framingham Criteria)

- Echo: Left ventricular dysfunction
- CXR: cardiomegaly, alveolar oedema (Kerley B lines), pleural effusion

2. Identify acute aetiology/rule out life-threatening causes/triggers*

- ACS - serum cardiac troponin
- PE – D-dimer
- FBC – anaemia
- TFT - hyperthyroidism
- CXR – rule out COPD, or lung cancer (based on smoking history and age)

*In this case, the mostly likely trigger of decompensated heart failure is uncontrolled hypertension – Her BP of 190/90 and she has not been taking ACEi. Note that hypertension due to non-compliance to medication is the most common cause of acute HF. However, PE is also a possible trigger, seeing her recent travelling and leg pain (although she has no chest pain (common feature of PE) and it sounds more like cellulitis, PE is so deadly and thus, we must rule out PE). Similarly, we also want to rule out ACS by ordering serum cardiac troponin since it is life-threatening if we miss out.

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Further management

- **Echocardiogram shows a left ventricular ejection fraction of 60%, concentric ventricular hypertrophy and no significant valvular disease. The cardiologist concluded that she has acute heart failure due to hypertension. She is now admitted to Gen Med Ward for ongoing management of APO and HF, which includes**
  - **Fluid balance chart including urine output and daily weight**
  - **UEC***
  - **1.5L Fluid restriction & sodium restriction**
  - **DVT prophylaxis from bed resting**

**UEC should always be ordered when frusemide is given – frusemide binds to Na-K-Cl cotransporter on loop of Henle, leading to loss of potassium. Hypokalemia is dangerous since it can cause cardiac arrythmia – we do not want cardiac arrhythmia, especially in people with bad heart function!*

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Reference

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