AMU Clinical Reasoning Session Key Learning Points (29.5.26)

Case 1 Summary

60-year-old gentleman with BG of IVDU, alcoholism with no fixed abode had frequent recurrent admissions for pleuritic chest pain with self-discharges. On this occasion, he re-presented with the same chest pain which he described as 10/10 severity with haemoptysis but was also seen eating & drinking, leaving the ward for prolonged periods and repeatedly asking for high doses of morphine.

On assessment, he was described as having haemoptysis and mild unilateral leg swelling but otherwise haemodynamically & clinically stable. His CXR & ECG showed nil acute with unremarkable troponin. He had a raised D-dimer 700 and raised CRP (<100).

He again self-discharged before further management could be instigated.

Heuristics (thinking tools) from this case

1. Problem Representation (root cause thinking)

   *Try the **problem representation** framework with the 3 key questions of Who? When? What?*

   - ***Who** is the patient – demographics, epidemiology, risk factors*
   - ***When** did this happen – pattern of illness inc. duration & temp*
   - ***What** is the clinical syndrome – signs, symptoms, findings*

   *This approach helps to **structure a concise summary** with the key features from which we can formulate appropriate differentials and the steps in management. It also helps us to think about the **root cause of a patient's problem.***

   If we apply to this case:

   60-year-old gentleman with no fixed abode who is an IVDU and alcoholic with recurrent admissions and self-discharges with pleuritic chest pain. Although he

had a raised D-dimer & CRP, he was haemodynamically & clinically stable with unremarkable CXR & ECG. On this occasion, he again self-discharged.

From the summary, it becomes apparent that ***the root cause of his problems is his drug & alcohol abuse*** *– the possibility of a PE & atypical chest infection, his drug seeking behaviour and self-discharges are all consequences / complications of his lifestyle.*

2. Cognitive biasing / anchoring

It is human nature to ***bias / anchor to a pre-formed diagnosis in our minds.*** *This could be due to previous information available to us before talking to the patient. We also often form* ***a pre-conceived diagnosis of the rarest or most potentially serious pathology*** *from the pieces of information (either subconsciously or not).*

In this case, it is easy to anchor / focus on PE given the patient's chest pain, haemoptysis and raised D-dimer. Although it is a reasonable differential to consider, it is important to note that pneumonia (haemoptysis common in Staph aureus infections in IVDU) or musculoskeletal CP from repeated coughing are more likely differentials. Additionally, almost anything can cause a raised D-dimer – it could also be due to a 'normal' inflammatory state in an IVDU.

3. Red flag or red herring

The question here is whether ***the raised D-dimer is a red flag or red herring.*** *It is important in medicine to ascertain whether an abnormal finding is a red flag or red herring, as if we treat all abnormalities as red flags then we may end up barking up the wrong tree or getting lost in the midst of all the abnormalities.*

4. Acute vs chronic pathology

A key question you should always ask in a case is whether the pathology is acute or chronic. *Most acute pathologies are the result of chronic pathologies and seldomly do we encounter truly acute pathologies without any chronicity – e.g. the rapidly progressive vasculitis, lupus or unprovoked PE without any RFs.*

As explained before in previous handouts – most acute pathologies in elderly & frail patients are a reflection of the progression of their co-morbidities and poor physiological reserve. As such, our focus should be on managing their frailty – pragmatic +/- palliative approach as needed.

In this case, the patient's acute pathologies (i.e. PE or atypical pneumonia) are a reflection of his 'chronic pathologies' – i.e. lifestyle choices – IVDU and alcoholism with behavioural issues. Hence, ***our focus should not be on worrying that his PE has not been treated as a result of his self-discharge, but on warning the patient of the consequences of his actions****, as no matter what we do to treat his suspected PE or pneumonia – if the patient does not manage his drug & alcohol abuse and engage in medical services – he will be at risk eventually of a massive PE or a cavitating lung abscess!*

5. Trajectory planning over diagnostic obsession

   Instead of focusing on the diagnosis of PE, considering the patient's trajectory can be more helpful in management planning. *Even if the patient did have a PE or pneumonia, it is unlikely to be a massive PE or abscess given his relative stability and frequent self-discharges. It is entirely possible that the patient is compensating well but the trajectory still remains of someone who has full capacity into his behaviour and still chooses to self-discharge despite medical advice. As such, his trajectory if he continues with his behaviour is unlikely to bode well and there is little we can do as clinicians to change his outcome except constantly warn him.* ***This trajectory planning can provide reassurance to us as clinicians that we have done everything we can for the patient.***

Structured Teaching from this case

A common cause of bacteraemia is IVDU – **it is one of the most common causes of Staph aureus bacteraemia associated with endocarditis within the first two weeks** of presentation (acute endocarditis). After two weeks; the most common organisms change to Strep viridans & oralis bacteraemia – common oral utensils causing a subacute endocarditis.

Bacteraemia follows a 3-step process within our bodies:

1. Bacteria's Port of Entry
   First, the ***bacteria must enter our bodies.*** *IVDU is a common mode of entry, any foreign objects introduce a risk of bacterial entry (e.g. peripheral / central lines,*

catheters), cuts / wounds / abrasions, surgery or dental procedures etc. Bacteria may also spread from a pathological source within our bodies such as Strep bovis from bowel cancers.

2. Bacteria in bloodstream = Bacteraemia
   Once the bacteria have entered our bodies – it must be ***in sufficient quantities within our bloodstream to cause a bacteraemia.*** *Not everyone who catches bacteria develops a bacteraemia. The risk is* ***immunosuppression.*** *Any RFs for immunosuppression such as HIV, diabetes, immunosuppressant therapy, autoimmune conditions, alcoholism will increase the risk of bacteraemia.*

3. Disseminated / haematogenous spread of Bacteraemia
   Not all those with bacteraemia develop organ dissemination such as endocarditis, discitis, organ abscesses. The risk is ***pre-existing organ disease / dysfunction.*** *Bacteraemia will target organs in an already weakened state to cause haematogenous spread. Hence, the RFs for endocarditis are prosthetic valves, heart failure and for discitis is a degenerative spine etc.*

As such, always send blood cultures if pyrexia is present and have a low index of suspicion for further management if there are significant RFs for immunosuppression or organ dysfunction.

3 Overused Clinical Tests

Often non-specific and can cause uncertainty rather than clarity in management.

1. D-dimer

   Should only be used in DIC or specific clinical circumstances of VTE. *Over-usage / over-reliance in every CP or swollen leg can lead to over-investigations without clinical thought. Focus on your clinical assessment rather than the D-dimer!*

   Remember almost anything can raise the D-dimer and a normal D-dimer does not fully exclude a VTE if the clinical suspicion is still sufficiently high.

   Disseminated intravascular coagulation (DIC) *is the abnormal clotting of small blood vessels throughout the body due to trauma, sepsis or malignancy (consumptive coagulopathy) which can lead to an acute bleed.*

DIC = high INR, high APTR, ***high D-dimer****, low platelets, low fibrinogen*

2. Troponin

   *Should only be done in MI or pericarditis****. Do not do a troponin routinely in every CP!*** *Consider only in cardiac CP +/- ECG changes.* ***Interpret the troponin with caution if there are other acute insults*** *such as AKI, HF, sepsis as this can cause* ***T2MI (non-specific or insignificant rise).*** *Please refrain from a Cardiology referral in cases of T2MI.*

3. AXR

   Think twice before requesting an AXR. Always ***consider PR before imaging to check for overflow.***

   If the patient is acutely unwell with abdominal concerns then proceed straight to an urgent CT and inform the surgeons. Remember acute abdomen can present without peritonism. If the ***abdominal pain is disproportionate to the rest of the exam +/- lactate then consider urgent CT.***

   An ***unremarkable AXR does not fully exclude an acute abdomen.*** *This could also be as the patient's acute abdominal pain could be due to a non-bowel related pathology which will not show on AXR such as acute pancreatitis, cholecystitis, GB perforation etc.*

Case 2 Summary

77-year-old lady with BG of T2DM, HTN, paraganglioma of her bladder (under Urology follow-up) was referred to SDEC by her GP for 1/52 feeling generally unwell including fevers, rigors and increased urinary frequency. Her GP had recently treated her with 3/7 nitrofurantoin which did not settle her symptoms.

On assessment, she had no chest or abdominal signs. Bloods showed raised inflammatory markers (WCC 17 CRP 230) and AKI. Asides from a trip to India 2/12 ago – there were no other lifestyle or medication changes.

As such, she was treated as for a complicated UTI with IV antibiotics & IV fluids. Her blood & urine cultures returned negative.

Heuristics (thinking tools) from this case

1. Problem Representation (PR)

   - ***Who*** *is the patient – demographics, epidemiology, risk factors*
   - ***When*** *did this happen – pattern of illness inc. duration & temp*
   - ***What*** *is the clinical syndrome – signs, symptoms, findings*

   77-year-old lady with BG of T2DM, HTN, bladder paraganglioma presented with 1/52 of fevers, rigors & increased urinary frequency which did not respond to nitrofurantoin. She had markedly raised inflammatory markers and AKI for which she was treated as a complicated UTI.

   The root cause of the patient's presentation is likely UTI from the major RFs of her bladder paraganglioma, T2DM and increasing age.

2. Pieces of the Puzzle approach

   Somewhat similar to the Pros & Cons approach (see previous handouts), this approach tries ***to fit the pieces (clinical features) together to solve the puzzle (diagnosis).***

   In this case, the 'pieces' would be fevers / rigors / urinary frequency, raised inflammatory markers, AKI and the patient's BG which fit the 'puzzle' of UTI.

   Importantly, there may be pieces that do not fit the puzzle. *In this case, the lack of abdominal signs, lack of response to nitrofurantoin and negative cultures do not fit neatly. However, these pieces do not mean you automatically discount UTI as your primary diagnosis –* ***you are solving a puzzle not building a house of cards.*** *It is* ***your clinical judgement*** *as to whether to discard the pieces that do not fit or not – this judgement will develop the more you practice!*

   It is important then that if you do decide to consider an alternative differential (i.e. hypothesis B) – you compare the merits of each diagnosis (i.e. pros & cons, statistical likelihood).

3. Disproportionate sign – red flag or red herring?

   Some (not all) cases often have a disproportionate sign which leads you to the diagnosis. You must ***be clinically careful that this sign is indeed a red flag and not a red herring.***

   In this case, the only potentially disproportionate sign would be the patient's recent travel history to India. Whether this is a red flag for atypical infection such as TB or simply a red herring would depend on your assessment (so far in the case summary – there are no other pros of TB notable).

4. Show your working (like A level Maths – management process > diagnosis)

   In this case, focusing on the trajectory will eventually guide us to whether this is a complicated UTI or another diagnosis such as atypical infection or malignancy. As such, rather than diagnostic hunting – focusing on whether I have done the right management steps to progress the patient's care at each point is more important. Retrospectively, we are often surprised at what the test results show which is reflective of ***focusing on management steps over the diagnosis.***

Structured Teaching from this case

The following are **5 causes of raised WCC:**

1. Infection – bacterial & viral (significantly raised WCC can indicate collection)
2. Malignancy – (e.g. significantly raised WCC with lymph seen in CLL)
3. Drugs – especially steroids
4. Gastric irritation – especially due to vomiting (unremarkable CRP is often a clue)
5. Autoimmune diseases such as vasculitis

As such, raised inflammatory markers (WCC & CRP) do not always indicate infection. Interpret in the individual clinical context to assess what the underlying cause is.

5 step approach to Antibiotics (Abx):

1. Assess if the problem is bacterial - not all raised WCC / CRP are infective!

2. Broad or focal abx – look for the source with primary symptom(s), imaging & cultures, RFs and previous infections.

3. Send & review cultures – before prescribing abx. If a recent culture shows resistance to co-amoxiclav but sensitivity to co-trimoxazole then co-amoxiclav is unlikely to have an effect!

4. Beware of side effects of abx – seek advice from the pharmacist and the BNF. Common SEs of co-amoxiclav is C diff risk & cholestasis whilst macrolides are C diff risk, lowering seizure threshold & tendon issues.

5. Review abx daily +/- discuss with Microbiology – consider if the abx can be stepped down to oral. If the patient is not improving then assess whether the abx needs to be escalated or switched (sensitivities or different source).

Case 3 Summary

30-year-old gentleman usually fit & well (nil cardiac history) presented to SDEC with 6/12 progressive SOB with PND. He also complained of worsening leg oedema for the last few weeks and feeling very fatigued.

On assessment, he had marked oedema of his legs but no other systemic signs to note. CXR, ECG, bloods (inc albumin & BNP) were all unremarkable. He had normal observations throughout. The patient was discharged with outpatient echocardiogram to investigate in the first instance.

Heuristics (thinking tools) from this case

1. Problem Representation (PR)

   30-year-old gentleman usually fit & well presented with 6/12 of progressive SOB & PND with increasing peripheral oedema of his legs. Asides from marked oedema, there was nil systemic signs to exam with unremarkable CXR, ECG and bloods inc albumin & BNP.

From the summary, we can see that the primary issue appears to be heart failure.

2. Hallmark features in the clinical context

   SOB & peripheral oedema are hallmark features of right sided heart failure. A relatively unremarkable BNP can be seen in early HFpEF, obesity or constrictive / restrictive pathologies so it does not rule out HF aetiology. A nephrotic process is also not out of the question given oedema although the normal renal function & albumin goes against. So, the next question is what is the root cause of the patient's heart failure?

3. Root cause thinking

   When encountering cases ***– always think what is the underlying root cause?*** *Just like we take a weed out by its roots - we must identify the root cause in order to manage the patient's disease optimally.*

   In this case, we can think of the differentials of right sided heart failure esp with pulmonary HTN to help ascertain the root cause. Further information to assess whether there is a history of smoking, high BMI or significant FH would be important to aid in the root cause thinking.

4. Show your working (like A level Maths – management process > diagnosis)

   In cases of diagnostic uncertainty like this case – it is important to ***focus on the management steps rather than overwhelm ourselves with diagnostic hunting.*** *As such, instead of casting a wide net of every single test to investigate every single cause of pulmonary HTN – starting with an echocardiogram as the next step then progressing based on findings +/- evolution or resolution of symptoms would be the practical approach.*

   Additionally, given the chronic nature of the patient's symptoms and relative stability with unremarkable baseline tests – this trajectory allows us to confidently investigate the patient as an urgent outpatient basis rather than subject him to a prolonged inpatient stay.

Structured Teaching from this case

Pulmonary HTN is classified into 5 groups based on the underlying root cause:

1. Arteries = pulmonary arterial HTN

   Diseases affecting the pulmonary arteries – idiopathic PAH, hereditary PAH, connective tissue disorders (esp scleroderma), congenital heart disease, portal HTN, HIV infection

2. Heart = heart failure

   Most common cause – HFrEF, HFpEF, valvular disease, congenital heart diseases

3. Lungs = chronic hypoxia

   Lung diseases – COPD, ILD, bronchiectasis, OSA, OHS, high altitude exposure

4. Clots = thromboembolism

   Chronic thromboembolic pulmonary HTN (CTEPH)

5. Miscellaneous = unclear / mixed

   Rare with unclear actions – sarcoidosis, haematological disorders, CKD, Gaucher's disease, metabolic disorders