

AMU Clinical Reasoning Session Key Learning Points (8.5.26)

Case 1 Summary

Young gentleman (in his 20s) was under GP follow-up for 2/12 of intermittent abdominal pains with change in bowels (but nil PR bleed or acute features of IBD), however he had a strong FH of UC. Faecal calprotectin was mildly raised at 250, hence further plans for IBD clinic referral was in progress.

The patient presented to his GP with worsening of his abdominal pain for which the GP sent the patient to SDEC. On SDEC assessment, the patient was noted to have raised inflammatory markers and pyrexia. Although he was not haemodynamically unstable nor have any peritonism – given the localised RIF tenderness; a CT AP was done which reported acute appendicitis.

Heuristics (thinking tools) from this case

1. Hallmark features in the clinical context

Hallmark features of IBD are abdominal pain with diarrhoea / change in bowels – PR bleed occurs in UC but is not essential. Systemic features such as weight loss, fatigue, fevers, mouth ulcers, arthropathy in Crohn's are also worth noting.

*In this clinical context; **young patient + insidious bowel symptoms + mild calprotectin rise + strong FH = IBD clinic referral.** Strong FH significantly increases pre-test probability. **Be careful not to anchor to IBD** – consider other causes in the young such as coeliac, meds (esp. NSAIDs), IBS, thyroid etc.*

2. Focusing on history & exam

*Not cognitively anchoring / biasing from documentation of previous history in clerking – 'background noise'. Whilst we can speculate whether his chronic symptoms and appendicitis were unrelated coincidences or appendicitis developed as a result of Crohn's inflammation in terminal ileum / appendix – this is not important at this stage. The most valuable message is **do not let a pre-existing diagnostic narrative blind you to new acute pathology.** The risk of this*

*increases **if we spend too long documenting data** – spend longer reviewing the patient – **focus on the history & exam!***

3. Disproportionate signs

*Localised RIF tenderness, pyrexia, raised WCC / CRP – prompted further investigation with CT. Note that the **abdominal pain was not peritonitic but disproportionately tender!***

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

CT AP as the next step is far more important than the diagnosis in this case.

Predicting diagnosis is often difficult and we are sometimes surprised at the unexpected finding – was acute appendicitis more likely than IBD flare in this clinical context (easier to say in retrospect)? Had we focused solely on the diagnosis of IBD flare then potentially we could have missed the CT and gone for flexi sig & Gastro r/v instead.

Structured Teaching from this case

3 red flags of abdominal presentation (GP/ED/AMU setting):

1. Pyrexia (esp. persistent) – this could be a sign of bacteraemia with abdominal symptoms a sign of systemic illness. Do blood cultures (at the very least)!
2. PR bleed (esp. persistent) – this could indicate underlying colitis.
3. Disproportionate abdominal pain (esp. localised) – note does not have to be peritonitic; it is a known phenomenon that patients (esp. elderly / frail) can present with an acute abdomen without peritonism. If disproportionate to the rest of your assessment – re-think with a low threshold for CT.

These help to differentiate acute abdominal pathology from benign pathology (such as simple gastroenteritis) and should prompt consideration for admission.

3 I's for colitis:

1. Infective colitis – any infective causes (e.g. Campylobacter, C diff, CMV). History is usually shorter with transient diarrhoea unless complicated.

2. Inflammatory colitis – history is more insidious with / without PR bleed. Not all cases are IBD (UC / Crohn's). Localized Vasculitis of GI tract – rare pathology. Common cause (esp. in the elderly) is microscopic colitis due to meds (i.e. SSRI, PPI, abx, NSAIDS, statin) – consider stopping / switching +/- budesonide. Usually requires Gastro referral with calprotectin for consideration of flexi / colonoscopy.
3. Ischaemic colitis – history is usually acute with hallmark features of PR bleed and vascular co-morbidities (esp. AF). Abdominal pain tends to be more severe.

Role of Calprotectin (important for GPs):

Calprotectin is not an IBD yes/no test – it is a marker of intestinal inflammation.

That said, calprotectin >500 with abdominal symptoms does point strongly towards IBD and prompts urgent Gastro referral.

Broadly speaking:

Calprotectin Interpretation

<50	IBD unlikely
50–200	borderline / mild inflammation possible
>200–250	more concerning for inflammatory pathology
>500	stronger signal for active IBD

Case 2 Summary

67-year-old gentleman with BG of significant vascular co-morbidities - advanced heart failure (severe LVSD with EF 20% with ICD), HTN, IHD, AAA (3.5CM), CKD3; presented with 1/12 of SOB. Bloods were largely unremarkable (mildly elevated D-dimer but normal trop & BNP). ECG chronic changes. CXR nil acute changes. Obs stable throughout without O2 demand. Clinically stable with no overt signs of organ decompensation, infection or deterioration.

During admission, Respiratory advised nil acute intervention needed (outpatient LFTs to rule out underlying COPD), with Cardiology also advising nil further needed. Patient was discharged without any acute medical treatment with an outpatient echocardiogram.

Heuristics (thinking tools) from this case

1. Acute vs Chronic pathology

*This case demonstrates progression of heart failure (chronic pathology) rather than a new diagnosis (acute pathology). Often times, investigating for **an acute pathology becomes the primary fixation** when it is either progression of chronic pathology (without acute) or the 'acute' pathology is really a reflection of progressive frailty & poor physiological reserve – **think pragmatic rather than over-investigative**.*

2. XYZ formula

***Investigations should change management. I will do X which will show me Y and I will do Z about it. If I can't get to the Z – re-consider do I need to do X?** In this case, consider if an inpatient Respiratory or Cardiology review would meaningfully have changed acute management. An outpatient echocardiogram is probably more useful although this also depends on whether there is any further room to optimise his end stage HF treatment.*

3. Hypothesis A vs Hypothesis B

*Let's say for example in this case – the D-dimer was more raised with 2L O2 demand with mildly elevated trop & BNP. Clinically the patient responds to diuresis. Your hypothesis A would still be progressive HF whilst hypothesis B could be to rule out PE. In a lot of cases in medicine – there is always a **hypothesis A which is most likely on the balance of probability whilst hypothesis B (usually the rarer diagnosis) is less likely**. The problem arises when hypothesis B becomes our main fixation – this leads to a non-pragmatic approach.*

4. Root cause thinking

Always ask in patients – why this symptom? Why in hospital or GP now? Is there an acute pathology as the root cause?

5. Red flag or red herring

In this case, the mildly elevated D-dimer was a red herring.

6. Trajectory focus rather than diagnostic obsession

*There can sometimes be a tendency in medicine to focus on establishing a definitive diagnosis. It is important to shift towards **trajectory planning rather than diagnostic hunting**. Think twice before investigating – e.g. in patients with end stage HF, consider whether repeat echo would meaningfully change management (esp. as inpatient). If the headache is benign without any red flags, then the trajectory will likely be safe for discharge with safety-netting. Often, we are reluctant to discharge patients or feel the need to investigate because we are not certain on a diagnosis. Accepting that the diagnosis is not clearly certain when the trajectory is safe is often a pragmatic & sensible clinical approach.*

If the diagnosis is complex / nuanced in an unwell patient; it is important to focus our management on the trajectory of decline - preparing for what may happen to the patient rather than the million list of differentials..

Structured Teaching from this case

HF (hallmark features)

Left ventricular failure when HFrEF (EF <50%):

- Clinical features – SOB / hypoxia, crackles / pulmonary oedema, PND
- Acute decompensation – IV diuresis (off-loading)
- **Chronic Disease Modifying Therapies (DMT)** – ACEi, beta-blockers, spironolactone (MRA), dapagliflozin (SGLT2 inhibitor), digoxin (esp. if AF)

3 indications for Cardiac Resynchronization Therapy (CRT-D) – EF <35%, dyssynchrony (LBBB, widened QRS), symptomatic despite optimal medical therapy)

Right Ventricular Failure (RVF) when HFpEF (EF >50%):

- Clinical features – peripheral oedema, raised JVP, ascites / hepatomegaly
- Symptomatic relief with diuresis and treat the underlying cause (i.e. lung disease).
No role for disease modifying therapies (DMT).

Transthoracic echocardiogram (TTE) indications

5 Indications for DOING:

1. Infective Endocarditis (IE) with high Duke's score
2. Decompensated HF with nil recent echo (last year) and not optimised on DMT
3. Severe valvular or obstructive heart disease (e.g. critical AS, HOCM)
4. Pericarditis / myocarditis – cardiac CP, rising troponin +/- unstable
5. Cardiomyopathies (ischaemic vs non-ischaemic aetiology)

5 Indications for NOT DOING:

1. Infection of another source – not to 'just rule out IE' – think twice!
2. Decompensated HF with recent echo, optimised on DMT, HFpEF
3. Murmur or syncope but not related to primary presentation ('background noise')
4. Non-cardiac CP (obviously)
5. Acute MI – echo just delays acute ACS treatment

Case 3 Summary (short discussion)

85-year-old lady with BG of HTN, dementia, AF, frailty presented with 2/52 of progressive lower abdominal pain with episode of likely vagal collapse at home. Bloods showed CRP 100 but nil else with unremarkable lactate. Apyrexial throughout with normal obs including nil tachycardia. No PR bleed or diarrhoea on examination with abdominal pain which is not acute / peritonitic. CT AP done by ED reported colitis of unclear aetiology. Requested GI Radiologist opinion whom advised acute ischaemic colitis. Pragmatic discussion with pt's husband / NOK who agreed with ward based & pragmatic (non-invasive) measures. Surgeons agreed (with outpatient flexible sigmoidoscopy as the ceiling).

Heuristics (thinking tools) from this case

1. For & Against approach

*The diagnosis of acute ischaemic colitis was not straightforward from the initial assessment. In cases of diagnostic uncertainty – **balance using for & against:***

For ischaemic colitis – age, vascular co-morbidities (esp. AF), progressive abdo pain, collapse leading to hospital could be hypotensive episode from ischaemia

Against ischaemic colitis – no PR bleed, no disproportionate abdo tenderness, no lactate, normal obs / clinically stable

*Different features would have different weighting – interpret the for & against in the clinical context. If uncertain then investigating / CT would be the safest approach. **However, given the age & frailty of this patient – pragmatic discussions with NOK would also be more patient-centred care.***

2. XYZ formula

Proceeding with GI radiologist opinion and pragmatic discussion with pt's NOK before discussing with the Surgeon (who agreed with conservative approach).

3. Diagnostic spectrum of disease likelihood

*We spoke briefly about how the threshold to further investigate is different for everyone (not black & white). **When uncertain - it is important to think of the trajectory / outcome.** For example, if the patient is young & fit – missing a diagnosis may be catastrophic, whereas if the patient is elderly & very frail – further investigating may not be in their best interests.*

4. Show your working (management process > diagnosis)

GI Radiologist opinion as the next step is more important than the diagnosis.

5. Trajectory focus rather than diagnostic obsession

This case demonstrates focusing on the age, frailty, co-morbidities of the pt and the wishes of the pt / NOK with a pragmatic (conservative) approach – trajectory focus is more important than hunting for the diagnosis of acute ischaemia.