

AMU Clinical Reasoning (CR) Session 18.9.26

Case 1 Summary

50-year-old gentleman with BG of MI (2024) & smoker of 20+ pack years presented with few days of episodic chest pains described as a tight constricting pressure lasting a few mins at a time. He stated the pain was recurrent & progressive with SOB but denied any exertional or positional component. He denied any other associated symptoms with nil viral illness, drugs or lifestyle changes. However, he did admit to a lot of stress recently.

On examination, he was comfortable at rest with nil systemic signs. His observations were all within normal range. ECG was SR with nil acute changes & troponins 35 -> 25. CXR and the rest of his blood tests were unremarkable. As such, he was discharged with safety netting.

The patient presented the next day with worsening chest pain – repeat troponin was 20,000s – he was promptly reviewed by Cardiology and transferred for further ACS management including invasive angiography.

Application of CR Frameworks

1. Understanding the Problem

Problem Representation (PR)

WWW:

Who – age, functional status, relevant co-morbidities (RFs)

When – primary complaint/s: duration & tempo

What – positive (+ relevant negative) findings

50-year-old gentleman with BG of MI 2024 & smoker of 20+ pack years presented with progressive episodic chest pains described as a tight constricting pressure lasting few minutes – associated with SOB but no other systemic symptoms or

signs. He denied any drug use or lifestyle factors except recent stress.
Observations, bloods, CXR, ECG were all nil acute except troponins 35 -> 25.

Risk factors & Root cause (RR)

The PR for this case is relatively straightforward – chest pain in a relatively young patient with stable parameters and a meagre trop rise. The relative difficulty is determining whether this is cardiac chest pain or stress induced MSK.

*For all cases **begin with PR and think RR** – what are the risk factors and what can the root cause be? This patient has cardiac risk factors – MI in 2024 and significant smoking history hence the root cause could be cardiac – ACS.*

So, what's the difficulty? Read on...

Diagnostic Discrimination

- Disproportionate sign/s – the lack of acute ischaemic changes on ECG and meagre troponin rise / change with otherwise normal assessment appear to be disproportionately unremarkable for ACS.
- Red flag or red herring – is the mildly elevated troponin a red flag? Is the recent stress a red herring?
- Hallmark features – **the pathogenic triad for ACS is suggestive history, ECG changes and troponin rise.**

*This patient has a mild troponin rise with cardiac risk factors but what about **his history – this I believe is the crux of this case.***

History & Exam (Data Acquisition)

*In medical school, we are taught that history-taking accounts for up to 75% of the assessment. I often say that Cardiology is simultaneously the most surgical & medical specialty in medicine because it is one of the most interventional / procedure based but also because **history-taking is the most important factor in cardiac / ACS assessment!***

Consider in this patient – he has significant cardiac risk factors (prev MI & smoker) with only a mild troponin rise hence the decision as to whether to treat this as ACS or not rests solely on the history!

*Delving into his history ('When' of PR) – there are a few concerning features. Firstly, his chest pain sounds **potentially cardiac** – described as a 'tight constricting pressure'. Secondly & significantly, it is **progressive in tempo** (crescendo), **non-exertional** (unstable) and **short in duration** (few days is more alarming than few weeks). Thirdly, it is very difficult to attribute the chest pain to anything else (absence of infective, muscular, VTE or iatrogenic aetiology) and as such, also associating it with stress could be premature closure (stress can also contribute to ACS).*

*In the context of a **cardiac sounding chest pain history** – the troponin of 35 becomes somewhat more of a red flag given that this also cannot be explained by anything else (no other tests or signs to suggest an alternative reason). Additionally, it would be useful to acquire data on his MI in 2024 – the significance of stenosis on coronary imaging and whether he had any stents.*

As such, this patient has a developing NSTEMI (confirmed by later presentation with troponin 20,000s).

2. Generating Hypotheses

Hypothesis Testing

Hypothesis testing in this case is relatively straightforward – hypothesis A vs B would be ACS vs stress induced chest pain. The difficulty would be which is more likely and whether the patient requires urgent cardiac management. Consider the following CR framework to help.

For & Against Approach (Evidence Evaluation)

For ACS chest pain – cardiac sounding pain, progressive & non-exertional nature of short duration, no other significant cause identified, mild troponin rise, significant cardiac risk factors (prev MI, smoker, age of 50)

For stress induced chest pain – recent stress, relatively mild trop rise, unremarkable examination & tests

Organising & evaluating the evidence – we can see that ACS is the more pertinent pathology hence hypothesis A – the more likely dangerous alternative to rule out.

3. Decision Making

Show Your Working (Metacognition)

Metacognition is the process of thinking about how we are thinking. Type 1 thinking is reflexive & quick whilst type 2 thinking is analytical & longer.

We see numerous patients presenting with chest pain at the front door – esp. SDEC. The vast majority have nil acute pathology and can be discharged with safety netting where type 1 thinking is utilised more often than not.

*However, if we conform to the same illness script for all chest pains then there is a risk of not detecting the more nuanced presentations that require more type 2 thinking. It is therefore important to pick up on **risk factors & focus on the history - switch to type 2 thinking with further analysis if disproportionate red flags** are detected.*

*Conversely, quite the opposite can be true. A high troponin or D-dimer is often interpreted as a T1MI or PE with CTCA or CTPA done when actually they were representative of the presentation with pneumonia & AKI. The key **is interpreting the test results in the clinical context – focus on the history & exam**. Only investigate for MI or PE if the clinical context supports the test findings.*

*Often times, we assess patient's upside down – instead of the history & exam guiding the troponin & D-dimer, we let the troponin & D-dimer guide our history & exam. I'll repeat myself - **focus on the history & exam!***

Trajectory thinking > diagnostic hunting

*Alongside focusing on the history – **trajectory thinking is perhaps the most important skill that this case highlights**. In medicine, it is impossible to be absolutely certain on a diagnosis and impossible to be completely risk free.*

*Uncertainty and risk are even higher in a front door setting with undifferentiated cases. **One of the best ways to manage uncertainty & risk is to switch to trajectory thinking from diagnostic hunting**.*

*Consider this case – it is difficult to be certain that the patient has ACS on initial presentation. The **key trajectory is the progressive nature of the chest pain** (non-exertional for only a few days) – suggestive of a relatively higher risk trajectory than if it was non-progressive occurring over weeks or months.*

Importantly, the patient was discharged with clear safety netting advice which prompted him to return the next day. As such, safety-netting provided an important additional layer of protection thus enabling prompt re-presentation. The key learning is to recognise a concerning clinical trajectory even when initial investigations appear relatively reassuring.

Availability bias

*Availability bias is an important cognitive bias framework where one over-applies a recent diagnosis to another case. For example, in this case, it would be important that future chest pains with a mild troponin rise with similar risk factor profile is not automatically concluded as ACS purely based on experiencing this case. **Each case must be assessed independently of each other.***

Case 2 Summary

95-year-old gentleman with BG of AF (DOAC), HTN, CKD3a usually independent & mobile (Performance Status 1) presented with 3-month history of progressive dysphagia, dysphonia and dysarthria. He had associated weakness causing recurrent falls & weight loss attributed to his dysphagia but denied any other systemic symptoms.

On assessment, he appeared cachectic and frail. He was alert & fully orientated. He had marked dysphagia with drooling of saliva and a dysarthric speech. He also had mild rigidity in his right side with bradykinesia. Otherwise, there were no other systemic signs. Blood tests were unremarkable with stable observations.

Given his progressive frailty with neurodegenerative signs – discussions around diagnosis & prognosis were had with the patient agreeing to DNAR & ward based (Best Supportive Care) approach. Subsequent Neurology review and CT TAP revealed metastatic cancer of lung primary with likely paraneoplastic phenomenon.

Application of CR Frameworks

1. Understanding the Problem

Problem Representation

95-year-old gentleman with vascular co-morbidities (AF, HTN, CKD) with PS 1 presented with 3/12 of dysphagia, dysphonia & dysarthria associated with falls. He appeared markedly cachectic & frail with a clearly dysarthric speech alongside right sided rigidity and general bradykinesia but preserved cognition. Given his resultant frailty & likely irreversible neuro-degenerative diagnosis – patient agreed to a Best Supportive Care approach.

Diagnostic Discrimination

- *Disproportionate sign/s – dysphagia, dysphonia and dysarthria with significant weight loss resulting in cachexia and falls.*
- *Hallmark features – the neurological features appear to be due to a neuro-degenerative process alongside the rapid history of deterioration suggestive of Progressive Supranuclear Palsy (PSP). Given his age & weight loss – an underlying malignancy is also likely (which it later turned out to be) but the lack of other systemic features is somewhat uncharacteristic.*

2. Generating Hypotheses

Hypothesis A vs B

Hypothesis A – Neuro-degenerative disorder (i.e. Parkinson plus syndrome such as PSP given falls, dysarthria, dysphagia with Parkinsonism signs)

Hypothesis B – Malignancy with paraneoplastic features (cachexia & weight loss with age albeit thought less likely)

(Although the patient had vascular risk factors – he had no lateralising neurology with haemodynamic stability suggesting that embolic stroke was unlikely).

Pre-test Probability (Bayesian reasoning)

*This case is an interesting demonstration of the tension between **clinical context vs pre-test probability**. Although the patient's clinical features were more in keeping with a primary neuro-degenerative disorder in the clinical context, malignancy with paraneoplastic phenomenon was arguably more likely on pre-test probability (esp. with PSP being statistically rare).*

*This reinforces that we must always **consider clinical context and pre-test probability together and neither should be used in isolation**. Often times, significantly more weight is given to the clinical context without or with minimal consideration of the pre-test probability – remember uncommon cases are uncommon and an uncommon presentation of an uncommon condition is even more uncommon!*

3. Decision Making

Trajectory thinking > Diagnostic hunting

*The first case and this second case both demonstrate **the clinical importance of trajectory thinking** albeit with opposite outcomes. Both cases have a high-risk trajectory but the first case requires an active escalation approach in a relatively young patient whereas this second case requires a pragmatic palliative approach in an elderly patient.*

*Therefore, although this case is interesting from a diagnostic point of view – we must not lose sight of the fact that we are treating the patient in his best interests hence **pragmatism and holistic care should be at the centre of our practice**.*

XYZ formula (Diagnostic Stewardship)

*Leading on from above, we must always **consider whether management is in the patient's best interests and will meaningfully change outcome**.*

In this case, Neurology review and CT scan has diagnostic & prognostic merits but has limited value in meaningfully changing the outcome given whether the diagnosis turns out to be PSP or paraneoplastic – prognosis remains guarded overall.

*As such, it is far more important to have a **compassionate discussion with the patient and his NOK to ascertain his understanding and wishes**. The patient may not wish to know of his diagnosis if it will not change management (and may feel upset if he does) hence doing a CT without discussion would be the wrong action. On the other hand (esp. in patients previously functionally well) – they may wish to know their diagnosis & prognosis even with the understanding that it would not change their management / trajectory as it may offer psychological closure and understand / set expectations around prognosis.*

Moreover, in some cases, investigating the diagnosis & prognosis may impact on the patient's palliative care management including eligibility for fast track (end of life) discharge planning.

*Overall, these highlight the **importance of discussing with the patient and always thinking & asking how management will meaningfully change the outcome** - if it doesn't or the patient does not wish for it then it would be in their best interests not to do it!*

Key Takeaways

- 1. Clinical context is crucial** – History & examination should guide the interpretation of investigations, not the other way around. Practice recognising disproportionate red flag signs where present.
- 2. Think trajectory, not just diagnosis** – The tempo and progression of symptoms are often more important than diagnostic certainty in determining clinical risk and the need for intervention.
- 3. Balance clinical context with pre-test probability** – always consider how statistically likely the disease is in addition to its likelihood in the clinical context. Remember common conditions are common and rare conditions remain rare!
- 4. Diagnostic stewardship** – The ultimate goal of clinical reasoning is not simply to establish a diagnosis but to make decisions that meaningfully benefit the patient - taking into account their trajectory, prognosis and wishes. Think twice before starting a management if it will not meaningfully change management. Shift the focus from solely treating the diagnosis to treating the patient!