Calgary Zone Department of Emergency Medicine

Grand Rounds Q&A - May 21, 2020

Q&A

Do you need to factor in duration of chest pain? I.e. if they only had 5 min of chest pain - stopped exertion and took NTG (and their chest pain resolved) - can you still rely on this pathway?

> This pathway and our data do not distinguish between duration of chest pain, and thus the results should be valid for all durations. However, someone that has clear exertional chest pain that resolves with rest would seem to have a high risk history. High risk clinical history should always trump biomarker results in my opinion.

Does Cardiology believe in the pathway, particularly around the observational zone? There seems to be high practice variation among cardiologists consulted for some of these patients, particularly with small but real 2 hour troponin rise, or persistently in the observational zone with a good story.

> I can't speak to all cardiologists, but Cardiac Sciences leadership has agreed to and signed off on the pathway. I think we should continue to inform and educate whenever possible, especially among the residents.

When comparing a patient's current troponin level to that patient's troponin "baseline", what do you consider a significant delta for these two values?

> I would say certainly a variation of less than 4ng/L would be in line with our data and pathway. However, if the patient's last 10 results varied between 30 and 45 with negative workups, I would certainly accept that range.

Do we know the lab variation in result of the hs-cTnT? A delta of less than 4 seems very kow if variation in result is +/- 3. Also any comment on validation of pathway in patients with CKD?

> Analytic variability from the lab is generally in the +/- 1ng/L (max 2ng/L) in the vast majority of cases. Moerover, we know the pathway works well in our practice environment. However, pre-analytic variability, including a sample being diluted when drawn from a saline lock do occasionally happen. This seems to be exceedingly rare however.
>
> As for patient's with CKD, pathway absolutely works for rule out (just a MUCH smaller proportion of patients will meet rule-out criteria). Rule-in will be much less specific, especially using the absolute rule. I would look more at clinical history and rise over time (deltas) as a marker of acute injury.

Does this change for people with very early presentation? I.e. they arrive with pain that has been ongoing for only 30 min or so. The second troponin, then, would be 2.5 hrs post onset. Should we keep them for a third, or hold on the time zero troponin until 1 hr post pain onset?

> Only change is that for patients with less than 3-hours of symptoms we cannot use the single rule-out on presentation (hs-cTnT < 5ng/L)
>
> All others go down the 2-hour pathway. Duration of symptoms does not influence the interpretation of the 2-hour pathway.

Follow up to Tom's question

Deltas in CKD - same deltas or is there further evidence applied in this patient population? Deltas interpreted with clinical history of course. Thanks!

> Unfortunately, there is no one delta that we can recommend for all patients, especially as troponin clearance changes with worsening renal function.
>
> However, if I see a ESRD on HD patient with a good chest pain history/no obvious alternate cause and a rule-in delta of >=10ng/L at 2-hours, I would recommend being cautious. In most cases that would include consulting Cardiology and sending a 4-hour troponin.
>
> With a low probability story, another alternate cause or a 2-hour delta in the observational zone (4-9ng/L) I might either discharge or send a 4-hour troponin based on my judgment of the patient's risk.

What was the discrimination (C-statsitic) for Heart Score in Connor's study?

> 0.89 in undifferentiated patients, 0.90 in ruled-out patients

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trop I HS pathway (used in urgent care and rural ER) requires a 0h and 2h value for all scenarios (there is no "one and done" test). Is there any research/data that would support a single value cut off of trop I hs (measured at >=3h from onset of chest pain) to rule out ACS?

> Yes, there are.
>
> The local lab has been reluctant to advocate for it until more quality control data have been collected.
>
> Hopefully within the year.

To Andrew's question regarding research direction: would be interested to see if established, easy-to-use /remember risk stratification tools (ie. HEART score) can be better recalibrated for our population of interest (CP NYD excluding NSTEMI/UA).

live answered

Is there any relationship b/w the TroponinI at the urgent care clinics and our TNT? Do we just have to start over numerically once they arrive in the ED? Why are there two different troponins used in the region?

live answered

It seems like the risks for people absolutely rulled out by Ecg and TNT is very low. Perhaps out pt testing should be reserved for those who have negative testing and a good story and those who go into the observational pathway but are ultimately ruled out in the ED. Would looking at the data we already have support this?

I agree, and we are looking at this.

With discrimination that great, I think re-calibrating the Heart Score is a great idea. MACE outcome but also looking at each specific component individually as I bet the calibration depends on the outcome.

Also will you consider looking at HEART score discrimination and calibration among “observe” population from Calgary pathway? Obviously with shorter term outcomes (even AMI)

I'm inclined to agree with you.

Also about the observational group, but sample size is too small with just local data.

Thanks

Are all out patient chest pain clinics of similar “quality”? For years I have used total cardiology but wondering if the ED group has equal confidence in CERA, Vita, Bloomberg, etc.

Challenging and politically loaded question that I cannot reliably answer.

Would be great to have some data on timing of consults, receipt of consult letters and outcomes.

Quality is a tough one--by what metric would we measure quality? I'm not aware of any direct comparisons.

Great rounds. Thanks again

What about cardiology commonly referring to demand ischemia and no need to admit these people. To me that just often means in the right clinical setting they gave themselves a stress test and failed and need further workup and/or monitoring for adverse events. Comments from your experience?

Really depends.

1. Main thing is do you think the issue is an acute/subacute coronary artery occlusion? They are the patients that benefit from DAPT/heparin. Or do you think it is a fixed stenosis unmasked by another medical condition - in that case, treat the condition first, then consider further risk stratification after.

2. If they are calling it “demand ischemia”, ask the consultant what is causing the demand? If they cannot identify a clear cause, must assume an acute coronary artery occlusion/ACS

I think if they were having symptoms, for sure. I don't have the short term risk after a "positive" stress test at the tip of my fingers, but you could conjecture it may be comparable. I think the clinical question is whether the patient is symptomatic vs. are they having ECG signs of flow-limiting disease. It's a tough question for which I'm not awrae of good evidence.