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Troponin: think before you request one

Syed Anjum Gardezi
Royal Gwent Hospital,NHS Wales,United Kingdom

Abstract

Acute myocardial infarction (ACS) is one of the most common presentations in acute hospital settings. Troponin (cTn) has emerged as one of the most sensitive biochemical markers for the diagnosis of ACS. However, if used inappropriately and in the absence of true clinical context then it can be elevated in a number of non cardiac conditions and lead to false clinical diagnosis, inappropriate workup, and increased patient stay in hospital. The cost of unnecessary clinical testing is another aspect of the problem.

At Royal Gwent Hospital in Newport (one of the busiest district general hospitals in Wales) we retrospectively analysed the nature of troponin requests over a random period of one week, specifically looking for the indications and final diagnostic impact. In many cases it was found that requests were made without any clinical justification. One of the main and probably unavoidable reasons for this was that requests were made from triage before patient was assessed by a clinician. However, steps were taken to clarify common clinical indications for suspected cardiac diagnosis in which troponin was useful. Additionally, the "tick box" practice for inappropriate laboratory investigations was discouraged. A repeat audit was done on similar basic principles and a measurable improvement was identified, with a potential for significant impact in future.

Problem

Measurement of cardiac troponin (cTn) has revolutionised the evaluation and management of patients with suspected acute coronary syndrome (ACS). Recent consensus statements endorse the use of cTnI or cTnT as the biomarker of choice for such application.[1]

Elevated cTn values outside of ACS are not uncommon and reflect cardiomyocyte necrosis from a wide array of cardiac, pulmonary, and systemic diseases.[2] A clinician must be familiar with the broad differential diagnosis of an elevated cTn to avoid false attribution of “acute myocardial infarction” (MI) in a patient without an ACS, thus reducing the burden of unnecessary work and admission rates for the hospital.

Another aspect of the problem is the financial impact of haphazardly testing in hospitals that are part of an NHS which is already struggling to keep up with its demands and supplies at present. Under observation it was noted that in most cases a troponin request was just a routine "tick box" practice that led to a lot of unnecessary processing. This then led to requesting these tests even before patient was assessed by a clinician.

It was also identified that troponin was often being used as a differentiating tool with the aim of providing a conclusion in the absence of an appropriate clinical history or examination that should be the mainstay in any clinical scenario. An improvement project specifically focusing on such potentially avoidable and otherwise quite useful investigations can be of great value for the hospital trust, as well as allowing the NHS to keep up with its aim of better quality with low costs.

Background

Cardiac troponin T and troponin I are the most specific and sensitive laboratory markers of myocardial cell injury and therefore have replaced creatine kinase MB as the gold standard.[3,4] The new definition of acute myocardial infarctions was based on elevations of cardiac troponins in blood in the setting of ischemia.[4]

The compelling clinical value of troponins resides in its superior prognostic potential in predicting the outcome of patients presenting with symptoms of unstable angina.[5] Therefore, the American Heart Association/American College of Cardiology (AHA/ACC) guidelines and the European Society of Cardiology (ESC) Task Force Report on acute coronary syndromes without ST elevation have attributed troponin measurements as having a central role in the diagnostic work-up and therapeutic decision making.[6,7]

Rising troponin values reflects irreversible myocardial cell necrosis. Accordingly, abnormal values have been described in various conditions not related to acute coronary disease, like myocarditis, pulmonary embolism, acute heart failure, septic shock, and as a result of cardiotoxic drugs. This is in addition to occurences after therapeutic procedures like coronary angioplasty, electrophysiological ablations, or electrical cardioversions.[8]

The National Academy of Clinical Biochemistry issued a guideline in 2007 which stated that, “In the presence of a clinical history suggestive of ACS, the following is considered indicative of myocardial necrosis consistent with myocardial infarction: maximal concentration of cTn exceeding the 99th percentile of values (with optimal precision defined by total cv [coefficient of variation] <10%) for a reference control group on at least one occasion during the first 24 hours after the clinical event.”[9]

The use of the 99th percentile cutoff for cTn positivity does not imply that 1% of the population suffers from myocardial damage. Rather, this cutoff is useful only when applied to patients with a high

pretest probability of ACS.

Baseline measurement

An audit was made at Royal Gwent Hospital in Newport, Wales on the indications and number of troponin requests done over a week period in acute areas of the hospital. One random week was chosen for this particular purpose (October 20th to 26th). The accident and emergency (A&E) department and the medical assessment unit (MAU) were the acute areas studied in this audit. Records of all requests were obtained from biochemistry department and analysed retrospectively.

A) A total 213 troponin requests were sent to the lab over a week

B) Among these, 170 were from A&E and 43 from MAU

C) 153/213 requests were for a possible cardiac symptom

D) 60/213 requests were completely irrelevant

E) Only 19 out of these came out to be positive. However, only 13 patients were labelled as acute coronary syndrome (ACS) subsequently giving a diagnostic yield of almost 6.10% (figure 1)

See supplementary file: ds5088.docx - “baseline measurements”

Design

Troponin is a very useful test if used in right clinical context. In an attempt to rationalise the requesting process, both A&E and MAU were targeted. The aim of this project was to help reduce the number of inappropriate requests, as well as actually defining an "appropriate request" was.

An important part of reducing the inappropriate requests was to eliminate the inappropriate "tick box" practice on biochemistry request forms. Since most of the blood requests were done from triage the next step was to focus on the clinical staff in that area, increasing their awareness about appropriate use of this biochemical marker and also simplifying the process by providing some general guidance.

Strategy

PDSA cycle 1: After identifying the problem, a plan was made to analyse all the troponin requests over a week including their source, reason of request, their outcome, and the approximate diagnostic yield. This was included in baseline measurements.

PDSA cycle 2: Initial results of the baseline measurements were shown to all clinical staff (nurses and doctors). Results were presented in a clinical gathering. A few suggestions were made for improvement, including:

1. Requesting troponin only when ACS was suspected
2. Discouraging the random tick box practice
3. Poster display of results in all clinical areas and specifically in MAU and A&E.

PDSA cycle 3: Making a short guidance note for triage staff; and to seek help if in doubt.

This included requesting troponin only in presentations like chest pain, shortness of breath, collapse/syncope, and palpitations (see supplementary file 2).

Results

After making the above interventions, some improvement was seen in the requesting process for troponins. Even so, it was also highlighted as one of the grey areas that still had a lot of room for improvement. Following are the results noted in the review audit under the same basic structure done after a month following PDSA cycle 2:

A) Total of 165 troponin requests were sent to the lab over a week

B) Among those 132 were from the accident and emergency (A&E) department and 33 from the medical assessment unit (MAU)

C) 141/165 requests were for a possible cardiac symptom

D) 24/165 requests were completely irrelevant

E) Of these, 22 came out to be positive. However, only 14 patients were labelled as having acute coronary syndrome (ACS), subsequently giving a diagnostic yield of almost 8.5%.

From the results above it can be concluded that almost 58 less troponin were requested after the above measures were taken in a week. This factor, in addition to the indirect influence on hospital and patient burden, would reduce inappropriate spending by almost £100 (one troponin is almost £2) less per week, which would of course equate to much greater amounts over a larger scale.

Another assessment was done after the third PDSA cycle, this time measuring only the effect of the final intervention on the amount of troponin requested. This was done almost two weeks after PDSA cycle 3 and identified that:

A) A total of 159 requests sent over from both MAU and A&E

B) 130 were from A&E and 29 from MAU

C) 144/159 requests were for a possible cardiac symptom

D) 15 requests were still sent for no apparent relevant symptoms.

These results certainly depicted an improvement in requesting this important biochemical marker which though not ideal but was encouraging (figure 2).

See supplementary file: ds5087.docx - “After change”

Lessons and limitations

An important learning point in this project was identifying the financial impact of such apparently minor clinical changes like requesting a troponin (although this is just one point raised among many others). During our first audit we were interested to note that some of the triage staff (including A&E staff) felt it a bit biased to use the posters like "Are we requesting too many troponins?" However, suggestions were made to rephrase that to "How to improve our troponin requests and when to request."

Clinical diagnosis is a systematic process. Due to an ever increasing workload and time constraints we as clinicians are increasingly moving away from process of history and clinical examination, relying too much on investigations.

Conclusion

Troponin is a very useful clinical marker to diagnose Acute myocardial infarction. However, it becomes positive in a number of non cardiac conditions. A positive result is only significant in the presence of an appropriate clinical history. A troponin should only be requested when a patient is suspected to have an acute myocardial infarction (MI).

It is a test that can easily be added on to the same routine sample of biochemistry if suspected later during clinical assessment. If this practice is followed appropriately for not only troponin requesting but also for other investigations then a good proportion of hospital admission workload can be reduced and valuable NHS money can be used alternatively.

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Declaration of interests

None declared.

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