

Horizon Scan Report 0013

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Diagnostic Technology: Point-of-care test for cardiac troponin**Clinical Question:**

In patients presenting with chest pain of suspected cardiac origin, is a point-of-care (POC) troponin test able to differentiate between myocardial infarction (MI) and angina and assist in patient stratification in the Emergency Department.

Advantages over Existing Technology:

Diagnosing cardiac ischaemia requires highly specific cardiac biomarkers, and in the past decade cardiac troponins (cTn) have emerged as the preferred choice over creatine kinase for diagnosis [1]. The demand for accurate testing of cTn was prompted by a redefinition of myocardial infarction by the European Society of Cardiology (ESC) and the American College of Cardiology (ACC) as any amount of myocardial necrosis caused by ischaemia [1]. Two cardiac troponin subunits, Troponin I (cTnI) and T (cTnT) can be measured and any elevation in troponin levels indicates some degree of myocardial necrosis. A number of laboratory assays offer reliable troponin measurements, however, many are unable to provide results within one hour of testing [2]. Alternatively, POC testing can significantly reduce the turnaround time from testing to results, allowing more immediate patient triage and effective treatment or discharge [3]. An electrocardiogram (ECG) is used to supplement diagnosis of ischaemia, though this is insufficient by itself, since myocardial infarction may not display ST-elevation and other conditions may also cause ST-segment changes [4]. In addition to saving time, some POC tests for troponins have been modified to simplify performance, reduce the chance of operator error, and use a blood sample that does not require pre-treatment [2].

Details of Technology:

Several POC troponin tests were identified:

1. Abbott i-Stat Analyser (Abbott Point of Care, USA). Supplied in the UK by Abbott Laboratories Ltd, Point of Care Division. Measures cTnI using a 16 μ L sample (obtained by a fingerstick). The device has a reported detection limit of 0.02 μ g/L and a cut-off level of 0.08 μ g/L. Results are available in less than 10 minutes [5].
2. Roche cardiac reader (Roche Diagnostics Ltd, Roche Diagnostics). Measures cTnT using a 150 μ L sample (obtained by a fingerstick). The device has a reported detection limit of 0.03 μ g/L and a cut-off level of 0.1 μ g/L. Results are available in less than 15 minutes [5].
3. Stratus CS System (Dade-Behring, Glasgow, DE, USA). Measures cTnI using whole blood samples anti-coagulated with heparin. The device has a detection limit of 0.015 μ g/L and a cut-off level of 0.07 μ g/L. Results are available in 15 minutes [6].
4. Biosite Triage System (Biosite Diagnostics Inc., San Diego, CA). Supplied in the UK by Alere, Stockport (previously Inverness Medical). Measure cTnI using a 250 μ L sample (obtained by a fingerstick). The device has a reported detection limit of 0.05 μ g/L and a cut-off level of 0.05 μ g/L. Results are available in less than 20 minutes [5].

A 2006 report from the NHS Purchasing and Supply Agency compared three cTn POC devices: Abbott i-Stat, Roche Cardiac Reader, and Biosite Triage system. They found the three systems reliable and easy to use, although the Roche Cardiac reader software was less intuitive and the device had excessive cabling that made the system cluttered [5]. Similar testimonies were reported in a study using the i-Stat cTnI assay, where users described the system as “easy to use” [17].

Patient Group and Use:

- Patients with chest pain and suspicion of acute coronary syndrome (ACS)
- Stratifying patients and diagnosing myocardial infarction

Importance:

The onset of chest pain with suspected cardiac origin may be due to acute myocardial infarction or unstable angina and patients need to be rapidly and reliably stratified for further management. The National Service Framework for Coronary Heart Disease (NSF CHD) estimated that in England more than 110,000 people die each year from CHD, despite being a preventable disease. Additionally, the NSF CHD estimated that each year 275,000 people have heart attacks and more than 1.4 million people suffer from angina [8].

Typically patients presenting with chest pain are assessed clinically, and those without clear ischaemia will be investigated with several ECGs and blood tests for Tn, often in the ED or chest pain assessment units for 12-24 hours. Only when results of these are available is it possible to have a definitive diagnosis, and correct treatment or discharge [9]. More rapid evaluation of patients could help to streamline patient flow, leading to not only lower costs (fewer patients observed, and for less time), as well as more rapid diagnosis of ischaemia and more rapid treatment. More importantly, studies have shown that 4-6% of patients in the United States (60,000-90,000 per year) are improperly discharged from the ED when they actually have MI [9].

The British Cardiovascular Society (BCS) working group on the definition of myocardial infarction recommends that troponin increase should be regarded as only one of the independent predictors of risk in patients with ACS and that troponin assays should meet the European Society for Cardiology (ESC)/ACC recommendations for precision [21]. The BCS, NICE and SIGN guidelines recommend that in patients with a suspected acute coronary syndrome, blood samples for cardiac troponin (troponin T or I) should be taken a minimum of 12 hours after the onset of symptoms [20,22,23]. The ACC and American Heart Association (AHA) guidelines emphasize that a 30-minute turnaround time is necessary for a rapid assessment of chest pain, and current laboratory tests are unable to meet such a requirement [6]. POC tests provide results well under the 30-min standard and they are becoming increasingly sensitive for lower levels of cTn. One primary concern with the use of cTn is the delayed measurable increase in circulating levels following the onset of chest pain, using many of the methods available. Levels rise approximately 4 hours after the event, are clinically measurable after 6 hours, and continue to increase until approximately 24 hours [5]. Thus measuring Tn too early in the course of chest pain could paradoxically reduce identification of recent ischaemic insult. With increased analytical sensitivity at lower levels, POC tests for cTn could reliably stratify patients in significantly less time and improve the efficiency in diagnosis and treatment of patients with chest pain [4]. An appropriate biological marker for cardiac ischaemia must be highly specific to cardiac tissue, have an affordable, rapid, and high quality assay, and must add clinical value when compared to other diagnostic tools [10].

Previous Research:*Accuracy compared to existing technology*

One of the major limitations in POC tests for cTn is the accuracy at low levels compared to laboratory assays. The ESC/ACC consensus committee defines increased cTn as a value exceeding the 99th percentile for a reference group, with acceptable imprecision coefficient of variation defined as 10% or less [1]. Comparison between techniques is difficult due to inconsistencies in detection limits and standardization in both POC and laboratory tests, suggesting the need for a standard reference material across tests [5].

A 2008 US study compared the Abbott i-Stat POC test with an automated laboratory assay for cTnI (Siemens TnI-Ultra, Siemens Medical Solutions Diagnostics). The POC test had a 99th percentile cut-off of 0.08 µg/L with an imprecision (CV) of 15% (0.012 µg/L), while the laboratory assay had a 99th percentile

cut-off of 0.04 $\mu\text{g/L}$ with an imprecision (CV) of 10%. In a sample of 557 patients admitted to the emergency department with suspected acute coronary syndrome that measured positive by the POC test, a comparison with the laboratory assay showed a positive predictive value (PPV) of 95.5%. A random sample of 137 negative results from the POC test was run for comparison on the laboratory assay and the results gave a negative predictive value (NPV) of 94.2%. None of the false-negatives had values higher than 0.1 $\mu\text{g/L}$, and by changing the laboratory cut-off value to 0.1 $\mu\text{g/L}$ the new sensitivity and specificities were 100% and 98.6%, respectively. The report concluded that the i-Stat POC test provides rapid patient stratification that is usually in agreement with the laboratory assay, with a low percentage of positive and negative values misclassified [11].

An Australian study from 2007 conducted a similar comparison between the Abbott i-Stat POC system and a laboratory assay (Beckman Coulter Access Accu – TnI Beckman Coulter). Recommended cut-off values were the same as the aforementioned study (0.08 $\mu\text{g/L}$ for POC, 0.04 $\mu\text{g/L}$ for the laboratory), however, a common cut-off value of 0.1 $\mu\text{g/L}$ was used for their initial evaluation. In a sample of 332 patients attending the ED with chest pain, sensitivity was 92.2% (95% CI, 83.8%-97.0%) and specificity was 98.4% (95%, CI 96%-99.6%). When the cut-off for the laboratory assay was lowered to 0.06 $\mu\text{g/L}$ (the 10% CV provided by international literature) the sensitivity of the POC test dropped to 70.1% (95% CI, 60.5%-78.6%), revealing the poor low-end performance of the i-Stat test. Overall, the study found that the POC test had high reliability compared to the laboratory standard, though lower concentrations of cTn present an area of weakness for the POC test [12].

The utility of the Roche cardiac reader for diagnosing MI was tested in a study conducted in China between 2001 and 2004 on 502 consecutive patients admitted within 24h of the onset of chest pain. TnT-positive patients (160) and TnT-negative patients (323) were followed up for 30 days. The cut-off value of the POC test for cTnT was 0.1 $\mu\text{g/L}$ and positive MI was subsequently identified by a quantitative laboratory cTnI test (AccuTnI 33340 device, Access immunoassay system, Beckman Coulter, USA) alongside ECG and other ischaemic symptoms. Sensitivity and specificity of the POC test were 95.2% and 93.8%, with PPV=86.9% and NPV=97.8%. The study concluded that the Roche POC device is a useful tool for risk stratification in patients with acute chest pain [3].

An Australian study comparing the Roche cardiac reader to a laboratory assay (Roche Elecsys 2010 immunoassay analyser) for measuring cTnT used a sample of 133 consecutive patients presenting to hospital with chest pain. The POC test had a detection limit of 0.1 $\mu\text{g/L}$ compared to 0.03 $\mu\text{g/L}$ for the laboratory assay. For the Roche POC test, qualitative readings between 0.03-0.09 $\mu\text{g/L}$ (appearing as “low” on the device) as well as quantitative readings >0.1 $\mu\text{g/L}$ were considered positive and any reading ≥ 0.03 $\mu\text{g/L}$ on the laboratory assay was considered positive. The POC test had a sensitivity of 75%, specificity of 100%, PPV of 100%, NPV of 95% and a total accuracy of 95% ($\kappa = 0.831$; $p < 0.001$). The study concluded that the sensitivity of the POC cTnT test needs to improve in order to reliably stratify patients with chest pain [13].

Impact compared to existing technology

A study published in 2006 (USA) examined the effects of the Stratus CS POC test on turnaround time, patient length of stay (LOS), and costs. Of the 545 patients with signs of acute coronary syndrome, 271 patients were monitored using central laboratory cTnI testing (PreCS group), and 274 patients were monitored by the POC cTnI assay by nurses who had undergone a one-month training period (PostCS group). Turnaround time significantly decreased in the PostCS group (Post CS mean 19.5 min; PreCS mean 76 min), and the savings per patient in the PostCS group averaged \$4,281 (\$12,882 vs. \$17,163). The average LOS decreased by 8% for the PostCS group (2.19 days vs. 2.36 days). The study concluded that the Stratus CS POC testing device is both cost effective and clinically useful for stratifying patients with signs

of ACS. They acknowledged inconsistencies that exist between different POC tests and recommend an evaluation of any POC test before implementing it in clinical practice [14].

To examine the impact of POC cTnT testing in the ED, a university-affiliated hospital in the Paris area sampled 860 patients presenting with signs of ACS between November 2002 and April 2004. Patients were randomly divided between POC testing (Stratus CS, cTnI) and central laboratory testing (Dimension RxL-HM analyzer, Dade Behring) and a common cut-off value of 0.1 ug/L was used as a positive cTnT indicator. The study focused on identifying patients with non-ST elevated ACS and found that the average time to anti-ischemic therapy was 45 minutes less for the POC group (median 151 minutes vs. median 191 minutes). They believe that the 45 minutes saved by using POC testing is clinically relevant in the ED, allowing for earlier invasive treatment. However, the study did not observe a decrease in the overall LOS for the POC group [15].

A 2005 ED study (USA) examined the effect of cTnI testing on patient LOS in patients presenting with chest pain. The average LOS for 232 patients before the introduction of POC testing was 7.1 hours (95% CI, 6.6-7.7 hours), compared to an average LOS of 5.2 hours (95% CI, 4.6-5.8) for 134 patients tested with a POC device (Stratus CS POC test). Sensitivity and specificity calculations used the laboratory results as a gold standard, and they found a sensitivity of 100% (95% CI 63% to 100%) and a specificity of 96% (95% CI 92% to 99%) for elevated cTnI. It was shown that POC cTnI testing significantly reduced patient LOS without compromising patient safety [16].

A recent 12-week randomised controlled trial in Australia which tested the effect of POC troponin testing compared to laboratory testing in the ED for patients presenting with ACS reported that POC testing reduced LOS by approximately 48min (95% CI 12 to 84), but this was not statistically significant ($p=0.063$). However, there was a statistically significant absolute increase of 10% (95% CI 4.3 to 16.6) in the number of people discharged with the target LOS of less than 8h. The authors commented that the degree of benefit would be dependent on the acceptance of the test by personnel, as well as the ED setting [24].

A multicentre randomised controlled trial of the use of a panel of POC cardiac markers (creatinine kinase, myocardial type, myoglobin and troponin I) compared to usual care in the ED of 6 acute hospitals in the UK (RATPAC trial; 2243 patients) showed that POC assessment was associated with an increased rate of successful discharge (OR 3.81, 95% CI 3.01 to 4.82), reduced median length of initial hospital stay (8.8 vs 14.2h; $p<0.001$), but no difference in mean length of initial stay, mean inpatient days over follow-up or major adverse events [25]. The study also concluded that troponin I alone was sufficient for diagnosis. In contrast, a multicentre randomised controlled trial comparing laboratory and POC troponin testing (i-STAT cardiac troponin I assay, Abbott) across 4 EDs in the USA (DISPO-ACS trial; 2000 patients) showed that the effect of POC testing on length of stay in the ED varied between settings, with one setting showing a longer time to discharge for patients receiving the POC test than those receiving the laboratory test [26]. It should however be noted that in the DISPO study physicians were given a choice as to their action, whereas in the RATPAC study they were required to follow a rigid protocol, therefore results from the DISPO study could be influenced by the choices made by physicians.

Guidelines and Recommendations

The NICE and SIGN guidelines on chest pain of recent onset recommend taking a blood sample for troponin I or T measurement on initial assessment in hospital as the preferred biochemical markers to diagnose acute MI. The guideline further recommends a second troponin measurement to be taken 10 to 12 hours after the onset of symptoms [20, 23].

Guidelines of the BCS, ESC, ACC, AHA, and the National Academy of Clinical Biochemistry (NACB) recognize cTn (I or T) as the preferred biomarker for detection of MI [1, 22]. The ESC/ACC consensus

recommends that an increase in cTn above the 99th percentile of the reference population is a useful standard for diagnosing myocardial infarction, with an acceptable imprecision (CV) of <10% at the cut-off value [6].

The 2007 ACC/AHA guidelines for managing MI in the absence of ST-elevation (non-ST-segment elevation myocardial infarction, NSTEMI) state that “to date, bedside testing has not succeeded in becoming widely accepted or applied.” Hesitations in implementing POC testing may arise from the qualitative and semi quantitative nature of many POC tests or concerns about low-end accuracy compared to more sensitive laboratory tests [18].

In a 2001 publication, the International Federation of Clinical Chemistry recommended quality specifications for cTn assays [19]. The paper addresses antibody specificity, calibration, sample dilution, analytical imprecision and detection limit, and assay specificity - drawing attention to the inconsistencies between the growing number of assays. Such variations should be kept in mind.

Cost-effectiveness and economic impact:

No cost effectiveness studies on troponin point of care (POC) testing were identified, however a few studies that looked at costs and outcomes separately were identified [14; 27-32]. To summarise, the studies showed that troponin POC testing provides a cost-saving strategy by (a) reducing unnecessary hospital admissions; (b) reducing patient length of stay in hospital; (c) increasing productivity – through rapid turnaround time for testing and (d) reducing cost associated with carrying out laboratory tests. The studies also conclude that the use of troponin provides rapid diagnosis which is clinically relevant and has a direct positive impact on patient management and outcome.

The impact of incorporating POC cardiac troponin I testing on assay turnaround time, patient length of stay, financial matrixes and patient outcomes was investigated in a US study that focused on patients presenting to emergency departments and cardiology services at risk of Acute Coronary Syndrome (ACS) [14]. The study concluded that there was a decrease in time of provision of results to healthcare provider (pre POC mean 76 min; post POC mean 19.5 min; $p < 0.001$) as well as a reduction in charge per patient admission (\$4281 savings) following implementation of troponin I POC testing. Similar costs savings were also seen in a UK study that looked at implementing POC analyser systems in hospitals [32]. The study reported an annual cost saving of £46,000 using rapid troponin T. In the US study total charges per patient admission decreased by 25% post POC vs. pre POC (\$17,163 vs. \$12,882); the mean length of stay also decreased 8% ($p = 0.05$) from pre POC (2.36 days) to post POC (2.19 days). One year survival was greater in the $< 0.1 \mu\text{g/l}$ patients (pre POC 96.2%, post POC 97.2%) compared to the $> 0.1 \mu\text{g/l}$ patients (pre POC 77.7%, post POC 75.5%); both $p < 0.001$. Kaplan–Meier survival curves showed early separation by 30 days in each group. The study concluded that the use of troponin I testing provides a more efficient and timely management of patients, resulting in better utilization of personnel, beds, and communicable services; saving money as a result.

Another study that evaluated the performance of an accelerated critical pathway for patients with suspected coronary ischemia and which utilized clinical history, electrocardiographic findings, and triple cardiac marker testing (cardiac troponin I, myoglobin, and creatine kinase-MB), reported a 40% decrease in coronary care unit bed utilization [29]. The use of troponin POC testing has the potential to significantly reduce hospital admissions as seen in a UK study where the aim was to determine whether patients presenting with chest pain who are at low to intermediate risk for ACS can safely be discharged from A&E using triple cardiac marker - TCM (creatinine kinase [CK-MB], myoglobin, troponin I) [30]. The results showed that almost one third of patients who presented with chest pain with low to intermediate probability of ACS were safely discharged from A&E using TCM. Six month re-admission rate with ACS in this group

of patients was only 1% with no deaths. The study concluded that paired TCM can be used to safely discharge this group of patients and reduce hospital admissions and readmissions.

This review highlighted the need for future research to be concentrated on undertaking cost effectiveness studies of troponin POC testing in hospital A&E departments to allow more rapid triage of patients who present with chest pain to appropriate treatment settings. More evidence on health-related quality of life outcomes associated with MI, and its long-term consequences, are required so that cost-effectiveness studies can use cost per QALY as the outcome. Only low to intermediate risks were included in most studies, there is a need to include more severe cases in order to obtain complete healthcare costs.

Research Questions:

Are POC Tn tests sufficiently accurate at the lower limit for them to be used as rule-out tests?

What is the role of POC testing in Primary Care and the Out of Hours setting?

What is the role and feasibility of POC testing by paramedics?

Suggested next step:

- a) Examining the patient pathway of patients with acute chest pain in primary care and OOH settings to determine whether POCT Tn would change the patient pathway. Patients with chest pain are currently referred directly to hospital without a GP consultation; therefore the utility of POCT Tn testing in primary care is unclear.
- b) Examining the role of POCT Tn testing by paramedics, by answering questions such as whether POCT Tn can it be used to rule out MI and avoid transporting patients with chest pain to the emergency department or whether it can be used to rule in MI to start therapy.

References:

1. Apple FS, et al. The diagnostic utility of cardiac biomarkers in detecting myocardial infarction. *Clinical Cornerstone*, 2005. 7 Suppl 1:S25-30
2. Christenson, R.H. et al. Cardiac point of care testing: a focused review of current National Academy of Clinical Biochemistry guidelines and measurement platforms. *Clinical Biochemistry*, 2009. 42(3):150-7
3. Guo X, et al. The predictive value of the bedside troponin T test for patients with acute chest pain. *Exp Clin Cardiol*, 2006. 11(4):298-301
4. Reichlin T, et al. Early diagnosis of myocardial infarction with sensitive cardiac troponin assays. *New England Journal of Medicine*, 2009. 361(9):858-67
5. Burtonwood C, et al. Three point of care devices for troponin measurement [report 06020]. London: NHS Purchasing and Supply Agency, 2006.
6. Amodio G, et al. Cardiac Biomarkers in Acute Coronary Syndromes: A Review. *Current Vascular Pharmacology*, 2006
7. McDonnell B, et al. Cardiac biomarkers and the case for point-of-care testing. *Clinical Biochemistry*, 2009. 42(7-8):549-61
8. Evidence Review. Fatty acid binding protein as a marker for cardiac events. Purchasing and Supply Agency, Centre for Evidence Based Purchasing, March 2010.
9. Caragher TE, et al. Evaluation of quantitative cardiac biomarker point-of-care testing in the emergency department. *Journal of Emergency Medicine*, 2002. 22(1):1-7
10. Zaninotto M, et al. New biochemical markers: from bench to bedside. *Clinica Chimica Acta*, 2007. 381(1): 14-20
11. Bock JL, et al. Comparison of emergency department patient classification by point-of-care and central laboratory methods for cardiac troponin I. *American Journal of Clinical Pathology*, 2008. (1):132-5
12. Loten C, et al. Validation of a point of care troponin assay in real life Emergency Department conditions. *Emergency Medicine Australasia*, 2009. 21(4): 286-92
13. Hallani H, Leung DY, Newland E, Juergens CP. Use of a quantitative point-of-care test for the detection of serum cardiac troponin T in patients with suspected acute coronary syndromes. *Internal Medicine Journal*, 2005. 35(9):560-2.
14. Apple FS, et al. Decreased patient charges following implementation of point-of-care cardiac troponin monitoring in acute coronary syndrome patients in a community hospital cardiology unit. *Clinica Chimica Acta*, 2006. 370(1-2):191-5
15. Renaud B, et al. Impact of point-of-care testing in the emergency department evaluation and treatment of patients with suspected acute coronary syndromes. *Academic Emergency Medicine*, 2008. 15(3): 216-24
16. Singer AJ, et al. Point-of-care testing reduces length of stay in emergency department chest pain patients. *Annals of Emergency Medicine*, 2005. 45(6): 587-91
17. Apple FS, et al. Analytical performance of the i-STAT cardiac troponin I assay. *Clinica Chimica Acta*, 2004. 345 (1-2): 123-7

18. Takakuwa KM, et al. The usage patterns of cardiac bedside markers employing point-of-care testing for troponin in non-ST-segment elevation acute coronary syndrome: Results from CRUSADE. *Clinical Cardiology*, 2009. 32(9):498-505
19. Panteghini M, et al. Quality specifications for cardiac troponin assays. *Clinical Chemistry and Laboratory Medicine*, 2001. 39(2):175-9
20. National Institute for Health and Clinical Excellence Guideline 95: Chest pain of recent onset. Assessment and diagnosis of recent onset chest pain or discomfort of suspected cardiac origin. March 2010
21. Fox KA, Birkhead J, Wilcox R, Knight C, Barth J; British Cardiac Society Working Group. British Cardiac Society Working Group on the definition of myocardial infarction. *Heart*. 2004 Jun;90(6):603-9.
22. British Cardiac Society Guidelines and Medical Practice Committee and Royal College of Physicians Clinical Effectiveness and Evaluation Unit. Guideline for the management of patients with acute coronary syndromes without persistent ECG ST segment elevation. *British Cardiac Society Guidelines and Medical Practice Committee and Royal College of Physicians Clinical Effectiveness and Evaluation Unit. Heart*. 2001 Feb;85(2):133-42.
23. Scottish Intercollegiate Guidelines Network. Acute coronary syndromes. National clinical guideline 93. February 2007.
24. Loten C, Attia J, Hullick C, Marley J, McElduff P. Point of care troponin decreases time in the emergency department for patients with possible acute coronary syndrome: a randomised controlled trial. *Emerg Med J*. 2010 Mar;27(3):194-8.
25. Goodacre SW, Bradburn M, Cross E, Collinson P, Gray A, Hall AS; on behalf of the RATPAC Research Team. The Randomised Assessment of Treatment using Panel Assay of Cardiac Markers (RATPAC) trial: a randomised controlled trial of point-of-care cardiac markers in the emergency department. *Heart*. 2010 Sep 30. [Epub ahead of print]
26. Ryan RJ, Lindsell CJ, Hollander JE, O'Neil B, Jackson R, Schreiber D, Christenson R, Gibler WB. A multicenter randomized controlled trial comparing central laboratory and point-of-care cardiac marker testing strategies: the Disposition Impacted by Serial Point of Care Markers in Acute Coronary Syndromes (DISPO-ACS) trial. *Ann Emerg Med*. 2009 Mar;53(3):321-8
27. Azzazy HM, Christenson RH. Cardiac markers of acute coronary syndromes: is there a case for point-of-care testing? *Clin Biochem* 2002 Feb;35(1):13-27.
28. Collinson PO. The need for a point of care testing: an evidence-based appraisal. *Scand J Clin Lab Invest Suppl* 1999;230:67-73.
29. Ng SM, Krishnaswamy P, Morissey R, Clopton P, Fitzgerald R, Maisel AS. Ninety-minute accelerated critical pathway for chest pain evaluation. *Am J Cardiol* 2001 Sep 15;88(6):611-7.
30. Rathore S, Knowles P, Mann AP, Dodds PA. Is it safe to discharge patients from accident and emergency using a rapid point of care Triple Cardiac Marker test to rule out acute coronary syndrome in low to intermediate risk patients presenting with chest pain? *Eur J Intern Med* 2008 Nov;19(7):537-40.
31. Shebuski RJ. Utility of point-of-care diagnostic testing in patients with chest pain and suspected acute myocardial infarction. *Curr Opin Pharmacol* 2002 Apr;2(2):160-4.
32. Herpichboehm B. Point of care Analyzer Systems in Hospitals: Contributors to Cost-Efficiency. *J Lab Med* 1999;23(2):093-6

Comments:

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