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What Cardiac Markers Can Tell Us in the ED

You are listening to ReachMD XM157, the channel from medical professionals. Welcome to a special segment focusing on heart health. I am Dr. Shira Johnson your host and with me today is Dr. Joe Lex Associate professor of Emergency Medicine at Temple University in Philadelphia. Dr. Lex was named outstanding educator of the year by ACEP The American College of Emergency Physician, and the award actually changed name to be re-named after him. He has been teaching, writing, and talking about his passion, Emergency Medicine, for many years. He is here today to educate us about what we do not know and about what we have probably forgot about the appropriate use of cardiac markers, which is an ever-changing art and science in the Emergency Department.

DR. SHIRA JOHNSON:

Welcome Dr. Lex**.**

DR. JOE LEX:

Oh thanks, it is great to be here and thank you for asking me to talk about something that I really do have a passion about the use and misuse of laboratory studies in the Emergency Department.

DR. SHIRA JOHNSON:

What misconceptions are there about the emergency uses of cardiac markers?

DR. JOE LEX:

I think the big thing that we have to get across to people is the only use for cardiac markers is to determine whether there is infarction. What we have right now the troponins, the CK-MB even the myoglobin will only turn positive if there is myocardial damage. We tend to think that somehow these are going to help us to figure out who has unstable angina and you have to take that off the table right away. None of these makers that we are using commonly now are going to tell us who has unstable angina. If you think that is what you are dealing with do not except these makers to get you in out of trouble or help you make some clinical decision unless they are positive.

DR. SHIRA JOHNSON:

We all trained knowing about CPK and LDH and we are all familiar at least with troponin and troponin I. What else do we need to know about the troponins that may be, we do not know.

DR. JOE LEX:

Troponin was originally touted as the answer to our problems. It was very specific for myocardial damage, and that is what was pushed early. What has happened since then, is people are discovering a lot of other things that will cause the troponin to go positive and I am not sure that word has gotten out there. I have heard anecdotes of people for instance in the Intensive Care Unit, which showed positive troponin and are suddenly started on heparin with the assumption there having a non-STEMI MI. Yet, if you look at all players in the Intensive Care setting about 40% of them are going to have a positive troponin, which probably has nothing to do with damage to their heart.

DR. SHIRA JOHNSON:

So what is the reason for that, the positive troponin in the ICU.

DR. JOE LEX:

Because, there are other organs, which will release troponin, and also even when the heart is not having a typical ischemic episode, I should say infarcting episode, it cannot release troponin for other reasons. There are plenty of case series out there; of the people with cardiac amyloidosis for status post atrial fibrillation, vasospasm, dilated cardiomyopathy, hypertrophic cardiomyopathies. All of these can cause a bump in the troponin and in the intensive care setting primary pulmonary hypertension, pulmonary embolism, renal failure, sub-arachnoid hemorrhage all of these have been reported to cause a post-positive troponin.

DR. SHIRA JOHNSON:

This is an expression, I heard used a lot clinically when there is a bump in troponin and you cannot really explain it, spilling a little, so

may be we should just admit him. Is that valid that thinking.

DR. JOE LEX:

I think it depends on what your presentation is. I think it would be hard to ignore a positive troponin in somebody in the proper clinical setting, and I would hope that is the only reason people would send the troponin level is in the proper clinical setting. If you suspect the patient may be having an infarct, so you can't ignore it, but I do not know that you have to get aggressive in treating it. If you do not have anything else to hang your head on.

DR. SHIRA JOHNSON:

So, what is meant by delta cardiac markers?

DR. JOE LEX:

Delta cardiac marker is a concept that Francis Fesmire came up with several years ago. At least he has done most of the research on it. It is the idea that, even with "in normal range" cardiac biomarkers. If there is a change in the biomarker over a defined period of time and in the Emergency Department that is defined usually as 3-hours sometimes as 6-hours. This is significant and requires more aggressive therapy or if there is no change in the biomarker in that 3-hour or 6-hour window, then you can safely say this patient is not having an infarct and can move on to some other diagnosis.

DR. SHIRA JOHNSON:

But again, you are saying clinically that does not mean the person should or should not be admitted, you are just ruling out an acute event correct.

DR. JOE LEX:

Well you are ruling out an acute infract, if you still suspecting the patient has ischemia and this is angina or delta is not going to help your delta troponin or delta CK-MB will not help you.

DR. SHIRA JOHNSON:

What else are some of the significants of mildly elevated markers.

DR. JOE LEX:

They may not have any significant if they are mildly elevated, but unfortunately they cannot be ignored. We have set-up this baseline of having the answer for any abnormal study and if it is abnormal we have to answer for. Out of the Emergency Department, what we frankly do is admit the patient and at let inpatient team sorted out.

DR. SHIRA JOHNSON:

Ya, inevitably that I do not like is for that and know why we are doing it, but as you said that you can ignore a slightly positive test.

DR. JOE LEX:

You really can by definition of course 1 and 20 tests is going to be abnormal.

DR. SHIRA JOHNSON:

You are saying statistically because of.

DR. JOE LEX:

Statistically, because we put this p-value as 0.5. Now with troponin, they have actually tried to tighten that down a little bit. If you look at some other research on troponin I, they are trying to eliminate false positives by changing the p-value to about 0.1, and that means you hope there would be only a one of 100 chance of a false positive, but that is a moving target like any upper limit of normal that is a moving target.

DR. SHIRA JOHNSON:

If you are just joining the channel, you are listening to a special segment on heart health on Reach MD. The channel for medical professionals on Dr. SHIRA Johnson and on speaking with Dr. Joe Lex from Temple University, we are discussing the use of cardiac markers and the sensitivity. So in simple terms because of our radio show we got no visuals or disposals, can you take through some of the acute stages of myocardial damage and ischemia, and relate that so what we have seen with markers.

DR. JOE LEX:

Sure, and I appreciate you using the term markers by the way. I saw here people referring to these as cardiac enzymes. Remember that CPK is really the only enzyme we are talking about. I think this is a hold over from the days when all we had was LDH and SGOT and CPK. This were truly are enzymes, where troponin is not an enzyme, it is a marker. So thank you for asking for using the correct terminology. What happens there is a cascade that occurs before we reach myocardial necrosis and dysfunction and there are makers being studied at each stage of the way. The first thing that happens is plague destabilization. Some of the markers that are being looked out for that include Matrix Metalloprotein is 9, myeloperoxidase. After destabilization, comes plague rupture.

DR. SHIRA JOHNSON:

If any of that proven or is any of that measurable in the clinical setting or is that is to research still.

DR. JOE LEX:

They are researched still so far. A couple of these you may recognize the names of, but I can promise you the careers are being built on researching these right now, and eventually I want to get to the point where I can tell you about one of my fears of what is going to happen if each of these comes on the market, but going from plague destabilization, we go into plague rupture, and there you can measure things like what growth factor, pregnancy associated, plasma protein A also known as PAPPA. Once the plague ruptures, there is an increase in acute phase reactance like C-reactive protein. After rupture comes ischemia and plague with activation molecules. Ischemia, we have ischemia modified albumin, which got a lot of a few year ago, but is kind of fall that off the table and then there is also unbound free fatty acids, which can be measured. Then, we finally get to necrosis after the ischemia and that is where we are in now.

DR. SHIRA JOHNSON:

So up to here we do not have anyway of measuring at least not accept in a research setting. We don't have anyway to measuring these stages you just took us through correct.

DR. JOE LEX:

Exactly, but there are at least 2 or 3 markers being studies at every step of the way and even before plaque destabilization, I am sure you have heard about the proinflammatory cytokines, which have been studied also interleukin-6, tumor necrosis factor. So, what I fear is some of these are going to show weak positivity and get published and people are going to jump on a bandwagon and then we are going to end up with these panels of chest pain biomarkers, which we are going be sending 6 or 7 tests, in addition to sending the troponin or the CK or the BNP. We will send an unbound free fatty acid level or a CRP or placental growth factor level.

DR. SHIRA JOHNSON:

And can you imagine the cost and the interpretation of that.

DR. JOE LEX:

Exactly, not only the cost, but the more test you send the more likely you are to get a positive. So that is a huge problem that I therefore see happening, which is why this is one of my passion, which is why I want Emergency Physicians to be aware of this. So, that they don't get caught flat-footed like we were with BNP, like we were with D-dimer may be like some people were with C-reactive protein.

DR. SHIRA JOHNSON:

How does ACEP or the American College of Emergency Physicians recommend the use of makers in the Emergency Department and do the cardiologists agree with that?

DR. JOE LEX:

Aaa the second part of the question I don't have an answer for you.

DR. SHIRA JOHNSON:

Hmmm.

DR. JOE LEX:

But I will tell you, I will tell you the first part. ACEP has been publishing clinical policies for many years and the most recent clinical policy on this was in the September 2006 issue of emergency medicine. It is called critical issues in the evaluation and management of a doubt patient's with non-ST segment elevation, acute coronary syndromes. With lead author on this, by the way was the same Dr. who did all of the work on delta biomarkers and his clinical polyps he talks about myoglobin, CK-MB mass, troponin I, and troponin T and one statement I think it is very important that needs to be emphasized. This is a direct code. It says no single serum marker used alone as sufficient sensitivity or specificity to reliably identify or exclude acute myocardial infraction within 6-hours after symptom onset.

DR. SHIRA JOHNSON:

So that sounds key within 6-hours after onset.

DR. JOE LEX:

Yes, no biomarker can either identify or exclude within 6-hours after symptom onsets. So, that is a very important part of the clinical policy the people need to be aware of.

DR. SHIRA JOHNSON:

Medicine is an odd as well as the science and a weight losing, the use of risk factors, and history along with cardiac markers and physical exam, and are rushed to go ahead with the advancing science, are we really forgetting what it is for practicing and why over there.

DR. JOE LEX:

I think so, one of the reasons I move from the community to the academic setting a few years ago was because I felt this way. I felt that a lot of people coming out of training programs were unable to make clinical decisions without ordering a lot of tests and I thought I might be able to make it difference. I am not sure that I am making a difference to be honest with you, but I am still holding on for dear life trying to convince people that it is still in the history, it is still in the physical. The lab studies don't help you that much, but unfortunately what I see is people are congratulated far more on knowing how to order an obscure study or how to get all the information out of the computer, than they are rewarded for recognizing jugular venous distention or an S3 by actually examining a patient.

DR. SHIRA JOHNSON:

And also I heartedly agree with you. We have the wrap it up, but I want to give my thanks today to Dr. Joe Lex from Temple University who has been out guest. We have been discussing the intelligent use of cardiac markers at the bedside. I am Dr. Shira Johnson, you have been listening to a special segment on heart health on Reach MD the channel from medical professional. Please visit out website at reachmd.com, which features our entire library to on-demand podcasts or call us toll free with your comments and suggestions at 888-639-6157 that is 888-639-6157 and thank you as always for listening.