Transcript Details
This is a transcript of an educational program accessible on the ReachMD network. Details about the program and additional media formats for the program are accessible by visiting: https://reachmd.com/programs/focus-on-disaster-medicine-and-preparedness/prognostic-indicators-in-acute-chf/3374/

ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

Prognostic Indicators in Acute CHF

The patient comes in to your office. He tells you he was seen in the ED since you last saw him for florid CHF, and of course, the ED never called you to tell you, you don't have those records, but what's the best prognostic indicator of his survival for the next year? You are listening to ReachMD XM 157, the channel for medical professionals. Welcome to the clinician's roundtable. I am Dr. Shira Johnson, your host, and with me today is Dr. Frank Peacock, Vice Chair in the Department of Emergency Medicine at Cleveland Clinic. Dr. Frank Peacock is also Chairman of the Emergency Preparedness Committee. He is coeditor of the book "Cardiac Emergency" is widely published and we are very fortunate to have him on the show today. Today, we are discussing acute CHF, prognostic indicators, and risk stratification. What do we know and what has changed?

DR. SHIRA JOHNSON:

Dr. Peacock, we are very glad you could take the time out to be here.

DR. FRANK PEACOCK:

Thank you for the opportunity to be here too.

DR. SHIRA JOHNSON:

Now, first of all, tell us a little bit about BNP. What is it and how was it discovered?

DR. FRANK PEACOCK:

Well, BNP stands for B-type natriuretic peptide. It is a whole series of hormones that are used to signal various events in the body, and the way that BNP is, is that when there is stress on the heart or either pressure or tension in the myocardial wall, BNP is released. It serves like a defensive hormone. It causes you to urinate salt water. That is where natriuretic peptide comes from. So, volume goes up, you make BNP and volume goes down because you urinate it out. It also happens in other situations. People think that it is a heart failure hormone. It really is more of a ventricular stress hormone. So, if you have primary pulmonary hypertension or acute pulmonary embolus, both of which are going to result in elevated right heart pressures, BNP can rise. So, it is not a standalone task. You cannot think about if the BNP is up, it is heart failure. What you have to think of is that if the BNP is up, why your ventricular pressure is up. Most commonly, it is heart failure, but it is not always that situation.

DR. SHIRA JOHNSON:

So, it is sensitive, but not specific.

DR. FRANK PEACOCK:

It is pretty specific at higher levels. If it has a low level of BNPs so if BNP is on a scale, there are 2 kinds of BNP. There is beta-natriuretic peptide BNP that is on 0 to 5000 scale, and there is also the precursor hormone, anti-pro-BNP and that is synthesized and then chopped into pieces. One of those pieces is BNP. That is on a 0 to 35,000 scale, but either one of those that is elevated, the higher they are, the more likely it is to be heart failure, and when they are low and for BNP it is under 120, pro-BNP it is under 300, when they are low, extremely sensitive. You do not have elevated pressures, you do not have heart failure, you think of another diagnosis for that patient's symptoms, but when they are high, high being greater than 400 for BNP, greater than 900 for anti-pro-BNP, much more likely to be heart failure and the higher they are, the more the likelihood is. You noticed I left a gray zone in between.

DR. SHIRA JOHNSON:

Yes, you did.

DR. FRANK PEACOCK:

In that gray zone, you have to sort of, sort that out where it might be heart failure, it is just early or there are other reasons or it might be something else, and so you have to be a doctor at some point and say, you know, this is likely to be this situation or not, and there are heart failure patients that walk around with elevated levels of these hormones and so, one example I use is that the patient shows up in the emergency department with a BNP of 700, well, that might be heart failure or it might not be in which I have do is, find out where they live at. If they live at 700 and they are coughing up green things and running a fever, then it is pneumonia. It is not heart failure, but if they come in and it is 1300 and they normally live at 700, then it is exacerbation of the heart failure. It is about

50% change is the magic number where you start saying this is a new change because there is a natural elevation in lowering of BNPs that it just occur from a lot of reasons that we have trouble explaining. So, if the patient has a 10% change, probably not clinically relevant, a 50% change clearly is clinically important.

DR. SHIRA JOHNSON:

How widely is it being used today, and importantly, you think it is being used correctly or is it just added on to a panel of markers and left for someone else to interpret.

DR. FRANK PEACOCK:

Well, I think that is the last point you make. There is one that I worry about is that if you fail to think about the clinical scenario, you are going to be wrong a lot and no doctor really wants to do that. So, the idea of adding it on and just reacting at the number blindly is not the appropriate way to use it. Now, when you talk about BNP, it is available as a point-of-care test. Lots of places use it, not necessary is the point-of-care. They have a machine in the labs. Most ERs have some type of measurement. Last time, I saw the numbers more than 85%. Because shortness of breath is a very common diagnosis in the emergency department and one of the things that happens is patients will get elevated BNPs and the doctor <_____> why he doesn't look that sick and as if there is clinical <_____> rule and my answer is if the BNP is really elevated, you have a problem, that patient is a high risk for bad outcomes. The higher the BNP is the sooner they are going to die. It is a very tight relationship, and that is not just long term, it is also acutely. <_____> I published a study that showed if your BNP is greater than 1700 at hospital admission, your probability at death is about 6% that week. The probability of death of myocardial infarction in most hospitals is 4%, so this equals an MI and no matter what you think of how good that patient looks, the number should make you concern, and I think docs are used to that and would like EKGs. EKG shows an ST-segment elevation MI.

DR. SHIRA JOHNSON:

Right.

DR. FRANK PEACOCK:

The patient has a high-risk of dying. You do not ignore that.

DR. SHIRA JOHNSON:

You don't have a second think it either you just go with it.

DR. FRANK PEACOCK:

Yeah, you don't say, oh! the patient looks pretty good, I am going to ignore it. The answer is no and systemic.

DR. SHIRA JOHNSON:

So, tell us a little bit about the ADHERE database because that is where lot of these results have come from and been looked at. This just may know already, but give us a little background at here.

DR. FRANK PEACOCK:

Sure. ADHERE was the National Heart Failure Registry. It was sponsored by Scios, which was some sort of <_____>. They enrolled 200,000 heart failure patients in it, so it is really one of the largest and richest data sources we have to understand what the state of the art and the care of heart failure is, and we really learned a lot about heart failure that we didn't know before with the data that is so rich.

DR. SHIRA JOHNSON:

If you have just joined us, you are listening to the clinician's roundtable on ReachMD XM 157, the channel for medical professionals. I am Dr. Shira Johnson and I am speaking with Dr. Frank Peacock in the Cleveland Clinic and we are discussing prognostic indicators in CHF.

So, what did ADHERE discover about troponin and CHF and mortality.

DR. FRANK PEACOCK:

So, this is a study that we just published a couple months ago in the New England Journal of Medicine. This looked at troponins and there are 2 kinds of troponin. That is I and T, and we sort of lumped them all together and said it didn't matter if your troponin was elevated what were the outcomes because this is not fairly common event. It happens somewhere between 6% and 10% of all heart failure patients who get a troponin and it is a little bit elevated, and the question is what does that mean.

DR. SHIRA JOHNSON:

We used to call them bumped a little.

DR. FRANK PEACOCK:

Ya, and the idea was well just heart failure, and so what we did is we looked at them and we had a population of 14,000 patients with elevated troponins, and with that numbers we are able to show that the higher your troponin is the sooner you died and it was not 6 months from now. It is in the next week you died. The mortality rate from now was fairly significant, exceeded that of an anterior MI. So, it was really sort of changed to what we thought of troponin, so my answer now is that everybody who comes in with decompensated heart failure needs to have a troponin level obtained, and if it is elevated, you need to be worried about and maximize the therapy that the patient is getting. Now, you know, some patients who are end stage, you may not be able to do any more. Heart failure is a chronic disease and the end point of that is they die from it, but as a marker of death if there are some patients that <_____> and the troponin is elevated, this is somebody you should be very aggressive. They should not just get bolus Lasix and watch. They should get more aggressive therapy than that.

DR. SHIRA JOHNSON:

Now, you said, mortality in-house exceeded that for anterior MI.

DR. FRANK PEACOCK:

Well, if you look at the national data for myocardial infarction, just lump all <_____> necessary anterior ones. Lump them all together, it is about 4%. Now, we are very aggressive with those people who go to cath lab, they get stents. The risk of death from troponin elevation and patient's presumed heart failure is about 8%, so it is just a markedly increased risk of doing poorly. You can also lump that with the BNP. If you have a normal BNP and elevated troponin, your risk of death is increased.

DR. SHIRA JOHNSON:

And you said for that hospitalization.

DR. FRANK PEACOCK:

Ya, for this week, if you have very high BNP, your risk of death is elevated. If you have a high BNP and you have a high troponin, the risk of death is about 10%. So, either one, markedly elevated is a problem, together it is even a worse problem. We are now developing risk stratification <_____> very objective in terms of patient outcomes, and when I talk about outcomes, I am talking about

this hospitalization outcomes.

DR. SHIRA JOHNSON:

When you are talking about a high BNP or a high troponin, is that viewed as a percent or an absolute number.

DR. FRANK PEACOCK:

Well, for most troponin labs if it is detectable above the threshold of the hospital the 99 percentile then that is a positive troponin. For BNP, it is a linear function. The higher the BNP, the worse they do. One of our study we did quartiles. With 1700 being the top quartile, about 850 being the third quartile and that is where mortality really picked up in those higher quartiles. So, either one of those being positive as defined in those terms is an ominous finding.

DR. SHIRA JOHNSON:

Now, in the past for years, before there was technology today, clinicians know to auscultate for an S3, they know they are in CHF, many times if you have been doing this for years, you know they are in CHF before you even listen for an S3, but you did a very interesting study with acoustic cardiographic analysis. What did that show?

DR. FRANK PEACOCK:

So, what acoustic cardiographic analysis is we took out, there is a company who makes a product called <_____>. It removes the V3 and V4 leads from EKG and replaces them with microphones that also have electrocardiographic sensing capability. So, you get a dual sensor placed in the V3 and V4 position. What that does is that it allows you to record 2 kinds of data simultaneously. You get the electrocardiographic data, so you get the time intervals of the heart, and you get the acoustic data in digital fashion, so then you can process that. The advantages of that is it is instant. It is data that you can get within 10 minutes of seeing that patient. You do not even have to draw blood, and the real reason to do this is because yes everybody knows the S3 is really important, and if the patient has an S3, they stay longer in the hospital, they die at higher rate, they cost more. It is a bad marker to have an S3. The problem is doctors today and I don't know if they were better 100 years ago, but doctors today miss 4 out of 5 patients who have S3s.

DR. SHIRA JOHNSON:

Ya, I believe that.

DR. FRANK PEACOCK:

It is hard, and if you put it in an ER environment, you know, the patients will yell and scream and it is noisy, and it is even more difficult. So, what we did is we took that device and we looked at the accuracy of diagnosis. For physicians in the emergency department compare to if they had used the data so that could be obtained with electroacoustic waveforms and it doubles the rate of accurate diagnosis in the first 10 minutes. Now you can say, while in the first 10 minutes, I don't have a chest x-ray and I don't have the BNP level back yet and there is a lot of things that I would like to have I don't have, but the reality is in sick patients you have to make a decision.

DR. SHIRA JOHNSON:

Right.

DR. FRANK PEACOCK:

And you have to make it now and you don't get to have the chest x-ray and you don't get to have the BNP levels. In an hour, you have that stuff, but right now you don't, and so it improves the accuracy of early decision making in the population of patients who present with undifferentiated shortness of breath.

DR. SHIRA JOHNSON:

So, troponin, BNP markers, short-term stay may not turn out very well. You have a higher risk of death in the first week. What about the S3? Has the data on that changed?

DR. FRANK PEACOCK:

Well, it is the same type of data that if you have an S3.

DR. SHIRA JOHNSON:

You are going to have a troponin and a BNP.

DR. FRANK PEACOCK:

Well! you will be obtaining those data later, but if the S3 is there, it is also a marker of worse outcome. The patients who don't have S3s do better than patients who have S3s. Now, heart failure divides into 2 sorts of groups. There is the patient who don't have an S3 because it is just nonexistent and it can be for 2 reasons, 1, it can because there flow is just terrible and they don't make any heart sounds because they have no blood flow.

DR. SHIRA JOHNSON:

Right.

DR. FRANK PEACOCK:

Those people are clearly sick and not going to do well. The other population is the population of people who aren't sick at all and they don't have an S3 either. Interestingly enough, if you look at really young people, and by young I mean under 20, they may have S3s 15% to 20% of the time. By the time they age 40, that goes to 5%. So, it can be a normal finding when you are young. It is not normal when you are older, and heart failure is the disease predominantly in old people. We do see it in viral cardiomyopathies and some unusual reasons for young people having heart failure, but 85% heart failure is old people and so it is where the money diagnosis is.

DR. SHIRA JOHNSON:

Dr. Frank Peacock thanks for being with us today. I really enjoyed talking to you and I have a feeling you are a great teacher as well as a great physician.

DR. FRANK PEACOCK:

Thank you for the opportunity to be here today.

DR. SHIRA JOHNSON:

We have been discussing CHF and risk stratification.

I am Dr. Shira Johnson. You have been listening to the clinician's roundtable on ReachMD XM 157, the channel for medical professionals. To comment or listen to our full library of podcast, visit us at www.reachmd.com, register with the promo code radio and receive 6 months' free streaming for your home or office. Thank you again for listening.