Winter 2013

Reading

CEPCP

Professional Development:

Assessment of chest pain

The Call

It was the kind of torrential downpour that immediately soaks everything that is exposed to the elements. A heavy, steady, relentless kind of rainfall that often accompanies the strong winds of fall. The gusting winds ensured that even vertical surfaces became glistening wet. It would seem any of the fall leaves that were still hanging on to ever barer tree limbs would have to let go tonight. The winds and rain were a strong team, clearing the way for winter.

It was well past the start of a night shift. The vehicle check was complete and the base was tidied up enough that it was now time to relax for a bit. It seemed like it was going to be a quiet night, the rain would keep people inside and hopefully out of trouble, we thought. It was almost as if the lever on the recliner was wired straight to the base pager because as soon as I pulled it to comfortably recline, the tones went off. “4542 code 4, 776 Brown Avenue...”. I flipped the lever in the opposite direction and headed for the truck. My partner quickly ended the phone conversation he was having with his girlfriend and joined me.

The emergency lights reflection against the glistening, rain-soaked surfaces was almost disorienting. The wind buffeted against the truck making it sway as my partner navigated the large puddles on the mostly empty streets. I was dreading stepping out in the rain and briefly considered grabbing my rain coat. It was not an appealing thought to spend any time in the rubbery, clammy confinements of the rain coat though, unless absolutely necessary, so I decided to take my chances. Hopefully we would not be outside for very long.

"4542, you are responding code 4 to 776 Brown Avenue for a 67 year old female with chest pain. She is also nauseated. History of diabetes. No further information."

It took longer than it should have to find the house. The brand new bungalow was located in a new subdivision that was still under construction. The street did not appear in the mapping software nor on the dash mounted Garmin. After being directed to the correct street by dispatch most of the houses still had no numbers on them. The ones still under construction were completely dark. Finally a porch light flashing on and off alerted us to the correct address. The driveway had not been paved yet and the compacted dirt was quickly turning into mud. There was an unsettling feeling of softness as we backed into the driveway.

"I hope we don't get stuck.", I said to Ben, my temporary partner.

"Yeah, I bet pushing me out is going to really suck.", he said with a smirk and booked us 10-7 on scene.

The patient was a relatively old looking 67 year old female. We were directed to where she was seated, in a recliner watching TV, by her husband who seemed very used to this type of emergency. There was no apparent upset or alarm in his voice or actions as he led the way.

"Good evening, my name is Phil, this is Ben, we are paramedics with the ambulance service. What is your name?"

"Margaret. I'm sorry to bother you boys but I am worried about this pain. I just want to be on the safe side." she said, whilst rubbing her chest with her left hand.

Ben was already applying the oxygen and was getting ready to get some vital signs and apply the monitor. I decided to explore the pain a bit further. The big question burning in my mind was whether or not this might be cardiac ischemic chest pain that I should treat with nitroglycerin and ASA, and maybe even by-pass to a cath lab. As I felt Margaret's radial pulse steadily beat against my finger tips I began asking questions.

Description of the pain

I think everyone has a fundamental understanding of what 'cardiac' chest pain is described as. When we are asking a patient about their pain, words like 'pressure' and 'squeezing' usually alerts us to the pain being of potential cardiac origin while sharp, reproducible pain often steers us away from a cardiac cause. But what about pain described as 'burning'? How absolute are the pain patterns? If the pain is reproducible does that mean that it is definitely not cardiac? I have scoured current literature trying to answer these questions. Although there is a lot of good information out there, a lot of questions remain unanswered. Let's work through a standard OPQRST-A question sequence and see how the answers to those questions can help us arrive at a correct working assessment.

Onset - "Did your pain start gradually or did it come on all of a sudden?"

This question is the first in the industry standard OPQRST acronym, but what does it really tell us? Is there a link between the onset of the pain and the cause of it? There is. Sort of. Pain that is of maximum severity right at the onset is not likely to be from a cardiac cause. Rather, cardiac ischemic pain usually increases gradually, reaching its maximum within the first few minutes. Clearly this is a fine line and simply asking the patient if the pain came on "all of a sudden" may not

adequately differentiate between pain that was truly at max intensity right away, from pain that took a couple of minutes to develop. It may be worthwhile to dig a little deeper. Pain that is severe right from the start is more likely to be a thoracic aneurysm, with potential lethal consequences.

"What were you doing when the pain started?"

This question also falls in to the "onset" category. Most paramedics are well aware that angina often comes on during exertion and is relieved by rest. But what about actual infarcts? It turns out they are often triggered by physical exertion too (Swap and Nagurney 2005). As most of us know, the first snowfall of the year usually triggers a number of AMIs and even cardiac arrests. It also turns out that there is a correlation not only with physical exertion but other stressors as well. Anger and major life events have proven to trigger AMIs in susceptible individuals. Even driving, or riding in traffic has shown to increase the risk of an MI in the hour that follows (Peters, Von Klot et al. 2004). However, many MIs are not triggered by any identifiable external factor so even though chest pain that came on at rest makes it a little less likely to be an MI, it certainly does not even come close to ruling it out.

Provocation - "Does anything make it better or worse?"

Chest pain that is reproducible by palpation or respiration makes a cardiac cause less likely but does not rule it out (Lee, Juarez et al. 1991). It has been shown that chest pain that is reproducible still has a 36 percent chance of being caused by cardiac ischemia (Body 2008).

It is also important to think about how reproducible the pain really is. Pressing on an arthritic, elderly person's chest and asking if it hurts is not likely to yield clear information about how reproducible the chest pain really is. Like every other case of pre-hospital assessment, use the information in context of all the other information. You cannot rely on any one assessment to rule out a cardiac cause of the chest pain.

Quality - "Can you describe the pain for me?"

The type of pain that is felt during an acute coronary syndrome can vary a great deal from person to person. There are no absolutes and getting as detailed as possible on what the patient is feeling is very important.
The classic pressure, tightness and squeezing are very common descriptors of cardiac chest pain for both males and females. Interestingly there is a difference between males and females with respect to the non-classical pain presentations. Females more often describe their pain as stabbing and sharp.

Another, important finding is that pain described as burning, or indigestion very often turns out to be of cardiac origin so do not let that type of pain description mislead you to believing that the pain is caused by heartburn. However, there are many other descriptions of discomfort that have ultimately turned out to be of cardiac origin. Many patients are reluctant to call their discomfort "pain" at all but may instead call it a "stiffness", "cramp" or "ache". I actually had a patient once who could only describe her pain as a "queer feeling" in her chest. It is important to listen to the words used by the patient and use those in your continued questioning. If, for example, the patient described the pain as an ache then the next question should be "Does your *ache*

go anywhere else?", not "Does your *pain* go anywhere else?". This acknowledges that you are paying attention to the patient and ensures that there is no confusion of which symptoms you are discussing.

Radiation - "Does the pain go anywhere?"

It is relatively well known that cardiac chest pain usually radiates to the left arm, shoulder and neck. This has been well supported by research (Goodacre, Locker et al. 2002). In fact, research suggests that radiation to either shoulder or to either or both arms are suggestive ischemic chest pain (Goodacre, Locker et al. 2002). As with most of the 'typical' features it is important to keep things in context. Only about 30% of AMI patients present with pain that is radiating, so non-radiating pain is certainly not enough to rule out a cardiac cause (Marsan, Shaver et al. 2005). Even here there is a difference between men and women with women reporting more radiation to the right arm/shoulder and twice as likely to report radiation to the back (Patel, Rosengren et al. 2004).

Severity - "On a scale of 1 to 10 how would you rate your pain?"

This question is important to ask if for no other reason than to be able to quantify the effect of any treatment we render. It is well appreciated that it is a very subjective question and that the patient who claims that their pain is a "Twelve out of ten." but who says so without any sign of distress probably does not share our view of severe pain. However, It does give us a measure of how much the pain is relieved in response to our treatment. Interestingly research has shown no link between severity and likelihood that the pain is from ischemia (Swap and Nagurney 2005).

Timing - "When did your pain begin?"

This is an important question as it may impact whether or not a patient can be bypassed to a cardiac catheterization lab. It does not reveal much useful information that we can use to rule in or out cardiac versus non-cardiac chest pain though. The exception being pain that has been present for over 48 hours without any ECG changes, which is unlikely to be caused by cardiac ischemia (Goldman and Kirtane 2003).
Another consideration is that typical, stable angina pain only lasts for 2-20 minutes (Bugiardini 2006). Any pain that has lasted longer than that is not stable angina.

What about pain that is spontaneously resolved, or that resolves with nitroglycerin? Surely that cannot be an AMI, right? Actually, it is not uncommon for a patient with an active AMI to have resolution of their pain prior to re-perfusion treatment (Goldman and Kirtane 2003). Do not ask me to explain how that happens, but it does.

Associated symptoms and 'silent MI'

There are usually a few associated symptoms, along with chest discomfort, that a patient with an AMI will have. These are the same symptoms that those patients that have silent MIs (MI without chest pain) often complain of. Let's go through some of the common ones.

Nausea

Nausea is a very common associated symptom that is strongly linked to AMI (Milner, Funk et al. 2002, Patel, Rosengren et al. 2004). It is more common amongst women than men (Patel, Rosengren et al. 2004). For example, in one small study of females who suffered MIs, ten out of the twelve patients reported having

nausea (Albarran, Clarke et al. 2007). In summary, nausea and/or vomiting by a patient who you suspect might have cardiac chest pain should be a red flag.

Nausea is also a very common presentation of a silent MI. In one study 24.3% of patients with a silent MI complained of nausea as their chief complaint (Brieger, Eagle et al. 2004). Hence, a 12 lead should be considered if a patient at risk presents with nausea, even in the absence of chest pain.

Shortness of breath

A great rule of thumb is to always ask a patient with chest pain if they are short of breath and vice versa. A thorough chest auscultation is also indicated in all cases of chest pain. If the patient is having an AMI, fluid may begin to back up and cause pulmonary edema which may present as wheezing and/or crackles. On the other hand, friction rubs, absent breath sounds or coarse, localized crackles may lead you to a different working assessment.

In terms of using the presence of shortness of breath to help rule in cardiac ischemia there seems to be a link, a gender specific one. Shortness of breath is strongly linked to AMI in females (Patel, Rosengren et al. 2004, Albarran, Clarke et al. 2007). Often mild shortness of breath is the initial presenting symptom for females suffering AMI (Albarran, Clarke et al. 2007).

Shortness of breath is a fairly common presentation of patient's with silent MIs. The reason for this is simply due to their inability to feel the pain of cardiac ischemia. They do not notice any signs until the left ventricle begins to fail, and fluid backs up into the pulmonary circulation (Coronado, Pope et al. 2004). In one study researchers found that shortness of breath was the only symptom experienced by 25% of patients with an ECG-positive stress test (Stern 2005). Another study found that almost half of patients with a silent MI had shortness of breath as their presenting complaint (Brieger, Eagle et al. 2004). The elderly and those with diabetes are more likely to have only shortness of breath as their complaint, and also tend to have a worse outcome (Stern 2005). Hence, shortness of breath in the absence of other symptoms must be investigated with a 12/15 lead ECG.

Paleness / Diaphoresis

The pale, grey, sweaty look of a truly sick patient is one that automatically sets off alarm bells with paramedics. It is the 'look test' fail or the 'sick' in 'sick or not sick' question. It should come as no surprise that it is linked to AMI (Swap and Nagurney 2005)(Malach and Imperato 2006). The signs are caused by the sympathetic nervous system which is very good at discerning when things are going horribly wrong. For some reason diaphoresis is more common amongst men (Patel, Rosengren et al. 2004) but is a very significant finding in females. In fact, one study found that diaphoresis was the strongest predictor of an acute coronary syndrome among women, even stronger than chest discomfort (Milner, Funk et al. 2002).

Diaphoresis have also long been recognized as a presenting sign of some silent MIs. Over a quarter of silent MI patients will have diaphoresis as their chief complaint (Brieger, Eagle et al. 2004). Hence we are correct in letting our spidy-senses tingle when we see that ghostly patient that is pale and sweaty.

Dizziness

Dizziness is another symptom that is often enquired about when assessing a patient with suspected cardiac chest pain. But what does it tell us? Is a patient with dizziness more likely

to be having an MI? The answer is no. There is no real link between the complaint of dizziness and AMI (Milner, Funk et al. 2002). In fact, in men, the presence of dizziness makes it less likely that they are having an AMI (Milner, Funk et al. 2002).

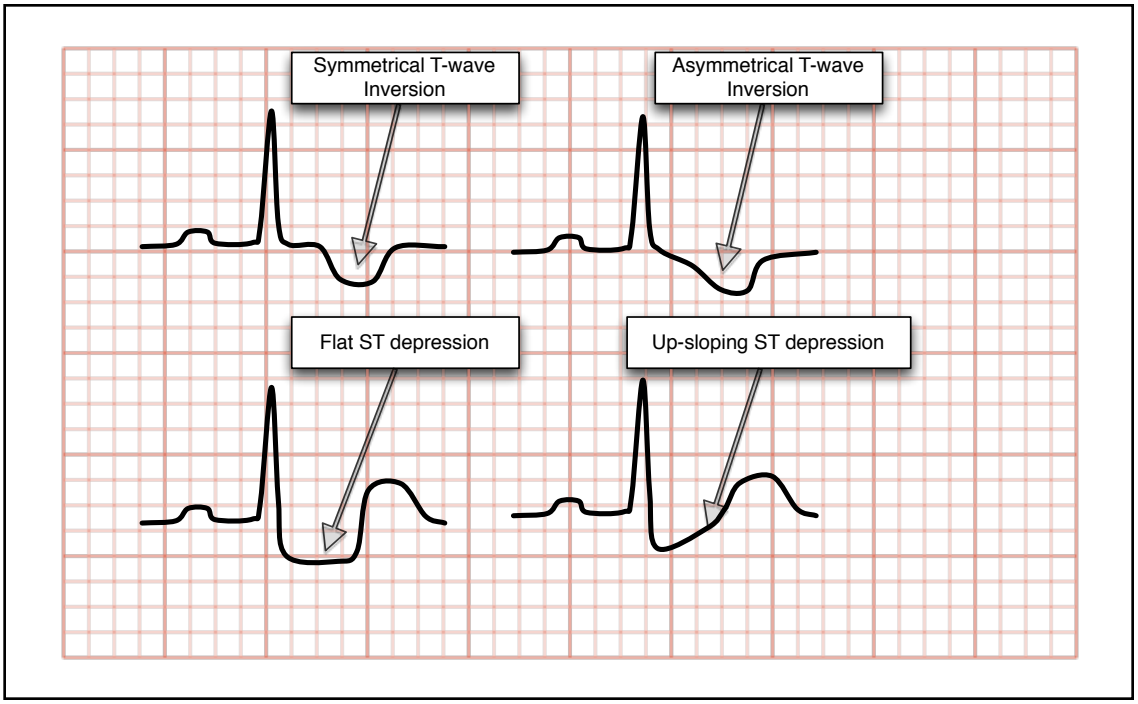
However, before we completely dismiss dizziness; it is sometimes a symptom of a silent MI (Brieger, Eagle et al. 2004). So it is definitely worth exploring a complaint of dizziness further to rule out a cardiac cause.

12 Lead Findings

The 12 lead ECG has taken on an ever increasing role in the pre-hospital setting over the past several years. It is now part of the routine assessment arsenal for a large part of our patients. We have all been trained to recognize ST elevation and determining if the leads that are showing the ST elevation are anatomically contiguous. There should also be an awareness that the absence of ST elevation does not necessarily mean an absence of an acute MI. However, there are a lot of additional information that we can obtain from a 12 lead ECG.

T wave inversion and ST depression for example are often signs of cardiac ischemia or even an evolving MI. For example, one study showed that of patients with ST elevation 80% had an acute MI and for those with ST depression or T-wave inversion 20% had an MI (Lee and Goldman 2000). Another study found that 32% of patient's with T-wave inversion and 48% of those with ST depression will have an AMI diagnosed by blood work (Body 2008).

When it comes to ST depression and T-wave inversion all are not created equal. Some are significant and some are not. T-wave inversion caused by cardiac ischemia is symmetrical whereas other types of T-wave inversion tends to be 'down-sloping', meaning that the first part slopes down on a shallow angle and then abruptly goes up (Thaler, 1995). The ST depression caused by ischemia or infarction tends to be flat and not slope up or down (Dubin, 1996).



Risk Factors

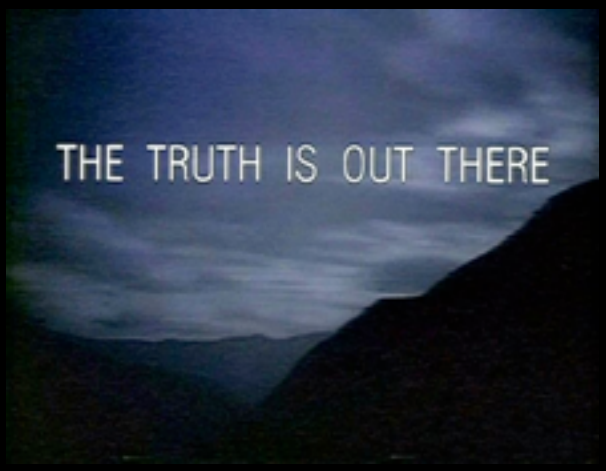
Let's say that you have asked all the questions about the patient's chest pain and you are still not convinced one way or the other. The pain sounds like it might be cardiac but there are a few little things that don't quite fit. There is not enough 'proof' to rule it in or out. What can you do? One thing to consider is the presence of cardiac risk factors. There are five major risk factors that have been identified as significantly putting a patient at risk for atherosclerosis and consequently AMI. The presence of one or more of these risk factors might make you re-consider ruling a patient out.

Smoking

The exact way in which smoking increases the risk for atherosclerosis is not well understood. There are multiple factors that contribute to risk but what is known is that smoking damages the inner lining of the blood vessels (endothelium). It also reduces levels of nitric oxides which is the body's way of dilating vessels. In addition to that, it increases the levels of 'bad' cholesterol. All in all cigarette smoking increases the risk of having an MI two to four times over non-smokers!

For a smoker who quits, the risk is cut by 50% in the first year and is reduced to that of a non-smoker after 10 years (Copstead & Banasik, 2010)

Syndrome X



Syndrome X might sound like something out of a science fiction novel, but is not nearly as paranormal as it sounds. Having said that it is somewhat mysterious. Let's say, for example, that you had a stress test done and the results were positive, meaning you developed chest pain and ST changes while huffing and puffing on a treadmill. Your Doctor would probably send you for an angiogram so that the site of occlusion could be found and a stent placed. Then imagine being told that there was no blockage found! You would be among the 20% of patients with a positive stress test for whom this happens. You would also be diagnosed with syndrome X. No alien abduction, no David Duchovny needed. So what gives? Why was the test positive if the arteries are clear? There are a few different theories. It could be caused by coronary spasm, microvascular dysfunction or increased pain sensitivity. More tests would be needed before a treatment regime could be provided. In the meantime you would have one of the coolest sounding diagnosis out there. (Kaski 2004)

Diabetes
A diabetic patient often have periods of relatively high blood sugar levels (depending on how well the patient controls the levels). The sugar is damaging to blood vessels. The damage done by the sugar translates to most of the long term complications of diabetes. In the larger vessels the damage leads to accelerated atherosclerosis.

In addition, type-II diabetes often co-exist with other medical problems such as high cholesterol, hypertension and obesity. This combination of these conditions is termed 'metabolic syndrome' and places the patient at very high risk of atherosclerosis.

High cholesterol
The exact mechanism by which cholesterol leads to accelerated atherosclerosis is very complex and still under investigation. Suffice it to say that the link between low density lipoprotein (LDL) and atherosclerosis is strong. Although LDL levels can be partially controlled by diet and exercise there are some genetic links to high cholesterol. A serum LDL level of 600 to 1,000 mg/dL for example may lead to death before the age of 20! This level of high cholesterol is very rare but a more usual level of 250-500 mg/dL often lead to AMIs in patients as young as the early forties (Bullock & Henze, 2000).

Family history
Clearly there is a genetic link to heart disease and AMI, but how strong is it? I'm pretty sure my great aunt died from heart failure in her 90's. Does that make me susceptible to suffering the same fate? Is that truly a 'family history'? Not really. Coronary artery disease (CAD) prior to 55 years of age in a first-degree relative (parent, sibling or child) is considered significant. With such a history the risk of developing CAD increases 2 - 5 times over normal.

Hypertension
The exact reason why hypertension increases the risk for atherosclerosis is not fully understood. What is known is that it does increase the development of atherosclerotic plaque, possibly from the added stress on the vessel walls. It also increases the oxygen demand of the left ventricle. As mentioned previously it is often seen in patients with metabolic syndrome.

Summary
In summary, there are many risk factors that contribute to the development of atherosclerosis. However, the five listed above are the big ones that dramatically increase the risk. It is good to be very familiar with these risk factors so that you can raise the index of suspicion appropriately when faced with a person complaining of vague symptoms but with several of the risk factors present. It may even be the feather that tips the scale towards treating your patient with vague chest pain as cardiac ischemia.

Meanwhile back on Brown Ave...

Despite my thorough and organized questioning of Margaret I was left unconvinced that her discomfort was from cardiac ischemia. To say that her description of the pain was vague would be an epic understatement. She did not seem sure when it started or if she had even had it before. When I asked her to take a deep breath and tell me if that made the pain worse she took a deep breath in, held it for an alarmingly long period of time and slowly

released it through puckered lips and said "I don't know dear, my arthritis makes everything sore."

There were some significant risk factors though. Margaret was a type-II diabetic with hypertension and high cholesterol. She had not smoked "..since Reagan was in office." (why anyone would link their smoking to the US presidency remained a mystery to me), so smoking was likely no longer a major factor.

Her family history was also a bit vague. Margaret claimed her father died suddenly when she was in her 30s (he had just turned 53), but she was not sure why. Even though her theory of a mistress having poisoned her dad was interesting, it did not help shed light on Margaret's cardiac risk factors.

It wasn't until Ben handed over the 12 lead ECG with a serious look and a tense "Here is the 12 lead, you may want to have a look at that." that I became fully clear on what was happening. The ST elevation in the inferior leads was significant enough to make them look like chairs lined up in a row. I was just about to ask Ben to do a 15 lead but he was already getting some new electrodes out and explaining the process to Margaret. As I watched him lean her forward to attach V8 and V9 I noticed that her T-shirt was soaked through with sweat.

Even though Ben and I remained calm and organized the sense of urgency and speed at which we moved was turned up a few notches. We soon had Margaret secured on the stretcher and moved her quickly through the relentless rain and muddy driveway into the ambulance. The right ventricular involvement we found on the 15 lead ruled out nitroglycerin but ASA was given before we left the house. Ben said he was sure he could make it to the cathlab in the 43 minutes we had remaining from our hour (we had spent 17 minutes on scene). "Are you good to go?" he yelled to the back and put the transmission in drive. "Yep, all good." I replied. As soon as Ben hit the gas it became apparent that our plans were coming unraveled. The truck strained for a moment against the mud's grip then the tires broke free and started spinning. The sound of mud flinging into the fenders making my heart stop for a moment. At the same time I looked at the cardiac monitor and at Margaret. This was turning out to be a very, very bad night. "Ben, we are not good to go! You better get back here, we have a problem!".

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