CEPCP

12 / 15 Lead Self Study Package

Spring 2008

Section Two

Introduction

This section of the review package will attempt to consolidate knowledge of 12/15 lead interpretation, anatomy & physiology and assessment skills into a comprehensive approach to better assess and manage chest pain patients. No assessment finding, vital sign or ECG should be viewed in isolation, rather they should be considered as pieces of the puzzle that our chest pain patients often are. After reading this section and completing the exercises included, I challenge you to consider this information when managing patients suffering chest pain or other complaints indicative of an AMI. Hopefully, this will lead to a deeper understanding and safer management of these challenging patients.



Anterior and Lateral AMIs

Anterior (and some lateral) infarcts are usually caused by an occlusion somewhere along the Left Anterior Descending coronary artery (LAD) (William, Harrigan & Chan 2006 p.1673). The number of anterior leads (V1-V6) involved and whether there are reciprocal changes in the inferior leads (II, III and AvF) can provide clues as to how severe the infarct is (William, Harrigan & Chan 2006 p.1673). Simply stated, if there are more leads involved and/or there are reciprocal changes the severity is higher. The shape of the S-T segment can also be an indication of how serious the infarct is and what type of complications can be expected. A 'Tombstone' pattern is associated with high rates of complications (dysrythmias or cardiogenic shock) and mortality (Balci & Yeslidag 2003). The 'Tombstone' shape is described as;

- Absent 'R' wave (or less than 0.04 s. duration)
- S-T segment convex upward, merging with ascending (upward) limb of the QRS complex
- Peak of S-T higher than the 'R' wave



- S-T segment merging with ascending limb of 'T' wave



Tombstoning pattern of anterior MI (Balci & Osman 2003)

As with any myocardial infarction (MI), anterior and lateral infarctions create a risk of a patient developing v-fib or v-tach due to ischemic, irritable heart muscle.

Section 2: 12-Lead ECG Interpretation

Also, a variety of heart blocks may occur due to necrosis of various parts of the conduction system (Zimetbaum & Josephson 2003).

Additionally, if a large enough portion of the left ventricle is affected, blood will 'back up' into the pulmonary circulation and cause pulmonary edema (William, Harrigan & Chan 2006 1642).



Posterior Wall MIs

Posterior wall MIs usually occur as a result of a Right Coronary Artery (RCA) occlusion, or occasionally a left circumflex artery occlusion (William, Harrigan & Chan 2006 p.1676). The standard 12 lead ECG does not 'look' at the posterior wall of the left ventricle. Moving V5 and V6 around to the back will allow those leads to view the posterior wall of the left ventricle (those leads then become V8 and V9).



Even though posterior infarcts are often part of a lateral or inferior MI, they can occur in isolation (William, Harrigan & Chan 2006 p.1677). S-T depression in V1 and V2 often occur as a reciprocal change to a posterior infarction so performing a 15 lead ECG on those patients is rather important (William et al. 1999). Although opinion varies on how many MIs may be missed by not 'looking' at the posterior wall, a couple of studies have found that 6.5 - 7 % of MIs showed S-T elevation ONLY in the posterior leads (Zalenski et al. 1993; Melendez, Jones & Salcedo 1978).

Inferior Wall AMIs

The inferior wall of the left ventricle is a relatively common site of MIs. The Right Coronary Artery (RCA) usually supplies this part of the ventricle but in about 20% of cases, the circumflex artery (a branch of the left coronary artery) wraps all the way around the left ventricle and supplies the inferior wall. So, an inferior wall MI is most commonly caused by RCA occlusion but can also be caused by an occluded circumflex artery (Zimetbaum & Josephson 2003).

Dysrhythmias are a relatively common complication of inferior wall MIs. Both the right



To figure out which artery is most likely causing an inferior MI simply compare the elevation in leads II and III. If there is more elevation in lead III than in lead II chances are good that it is a RCA occlusion that is causing the MI (Zimetbaum & Josephson 2003).

and the inferior left ventricle are connected to the vagus nerve and when these areas become injured there is often a strong vagal stimulation which lead to sinus bradycardia or AV blocks (Zimetbaum & Josephson 2003; Chockalingam et al. 2005).

Another important point to consider is that S-T elevation in monitoring mode (which is commonly lead II) *may* indicate an inferior wall MI. However, keep in mind when the monitor is in monitoring mode it may mask S-T elevation or on the other hand show false S-T elevation. Also remember that there must be S-T elevation in more than one inferior lead in order to have a 'positive' 12 lead finding. Always confirm with a 12 lead ECG.



Signs and symptoms of an inferior wall MI are, for the most part, the same as with any MI. Chest pain is the most common complaint and many patients also complain of shortness of breath (Chockalingam et al. 2005). A classic symptom of an inferior wall MI specifically, is nausea and vomiting, thought to be a result of the vagal nerve stimulation (Antman & Braunwald 2007).

Right Ventricular AMIs

Right Ventricular Infarcts (RVI) are most often due to an occlusion of the RCA and almost always occur in conjunction with an inferior wall MI (Kinch & Ryan 1994). However, the presence of an inferior wall MI doesn't always mean RVI or vice versa. Approximately half of inferior wall MIs have right ventricular involvement and conversely, in rare circumstances, a RVI can occur without the inferior wall being affected (Kinch & Ryan 1994).

It is important to determine if a patient with chest pain is having an RVI as the treatment that you provide could have serious adverse effects on patients with a RVI. Patients with RVI often become very dependent on their pre-load (amount of blood entering the heart from the veins) to maintain adequate cardiac output (Kinch & Ryan 1994; Ferguson et al. 1989). Reducing the patient's pre-load by administering nitroglycerine could lead to a dangerous fall in blood pressure and should be avoided (Kinch & Ryan 1994; Ferguson et al 1989; Antman & Braunwald 2007). The risk of adversely dropping a patient's blood pressure in this setting has not been studied on a large scale but one small study

found that 9 out of 10 patients with a RVI suffered a hypotensive episode, 7 of those appeared to be caused by nitroglycerin administration (Ferguson et al. 1989).

The best, most accurate way of determining if a patient is having a RVI is to obtain a 15 lead ECG and looking for S-T elevation in V4R (Zimetbaum & Josephson 2003). Make a habit of obtaining a 15 lead ECG whenever a patient is showing an inferior injury pattern on the 12 lead ECG (elevation in II, III and AvF). Remember, there is a 50% chance that they are also having an RVI!



Prehospital providers can play a key role in identifying RVIs as the S-T elevation in V4R is known to be relatively brief and may be missed if a 15 lead ECG is not obtained early (Chockalingam et al. 2005).

Continuing Medical Education

Because most of RVIs occur in conjunction with an inferior wall MI the signs and symptoms of an inferior wall MI (chest pain, shortness of breath and nausea / vomiting) are also indicative of a RVI. However, there are some signs and symptoms that are indicative of a RVI specifically and are important to assess for.



Jugular venous distention (JVD) that is present in a semi-sitting position is an indicator that the patient's right ventricle is not able to keep up and blood is 'backing up' into the venous system.

Kussmaul's sign (JVD that becomes evident when the patient inhales) is also a very reliable sign of RVI (Kinch & Ryan 1994; McGee 1998). The absence of either of these signs makes a RVI highly unlikely (Dell'italia et al. 1983).

RVIs often significantly reduce cardiac output due to a variety of mechanical and electrical factors and as a result the patient is often hypotensive (Chockalingam et al. 2005). Finally, because the problem isn't causing a back-up into the lungs, the lung sounds are usually clear.

In summary, JVD (or JVD on inhalation), hypotension and clear lungs in a patient with an inferior MI should alert you to the possibility of a RVI. Perform a 15 lead ECG to confirm your suspicion by looking for S-T elevation in V4R. If you suspect a RVI do not administer nitroglycerin. However, this does not mean that you **must** obtain a 15 lead ECG before giving nitroglycerin every time you treat a chest pain patient, just stay alert to the signs and symptoms of RVI and know that nitroglycerin should not be administered in a patient with a confirmed RVI.

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