

CASE REPORT: DIAGNOSIS AND INITIAL MANAGEMENT OF ACUTE CORONARY SYNDROME IN A RURAL
SETTING

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Home for the Summer Project June/July 2017

Abstract

During my home for the summer experience I saw several cases of acute coronary syndrome (ACS) both at presentation and in follow-up. I therefore decided to write my report on one of these cases with the purpose of reviewing the clinical presentation, diagnosis and management of ACS. In the case reported here, a previously healthy 58 year old male presented to a rural emergency department with a 15 hour history of persistent burning epigastric pain that radiated up his chest. Initial investigations revealed ECGs consistent with NSTEMI, however, over 12 hrs after presenting to the hospital, repeat ECGs showed ST elevation consistent with an inferior STEMI. The patient was transferred to St. Boniface Hospital where he received percutaneous coronary intervention (PCI) the following morning. Complete occlusion of the distal right coronary artery (RCA) consistent with inferioposterior STEMI was found and treated with drug-eluting stents. This case provides an opportunity to review management guidelines for both NSTEMIs and STEMIs, as well as to review the recommendations for treatment of STEMIs when presentation occurs after 12 hours of symptom onset.

Case History/Report

The patient is a previously healthy, 58 year old male with a past medical history of recurrent diverticulitis. At the time of presentation the patient was not taking any regular medications. Relevant family history included his father and brother who had myocardial infarctions (MI) at age 70 and 69 respectively.

The patient presented to the emergency department at 11:54 am complaining of persistent burning epigastric pain that radiated up his chest, since 9pm the previous evening. The patient reported the pain had a gradual onset and the pain was becoming worse today. The patient had scheduled an appointment in clinic for that afternoon with the complaint of acid reflux, but presented to the ER earlier due to worsening symptoms. The patient reported the pain was worse when supine. The pain was not made worse with respiration or on exertion. The patient also reported he felt weak and he had become diaphoretic 1 hour ago. The patient denied any shortness of breath, dizziness or nausea and vomiting.

On exam, the patient's color was normal and he did not appear to be in respiratory distress. Vitals were as follows: temperature 36.6°C, pulse 50 bpm and regular, respiratory rate 20 bpm, blood pressure 150/87 mmHg, and oxygen saturation was 100% on room air. Cardiac, respiratory and abdominal exams were unremarkable.

Initial investigations included electrocardiogram (ECG) and blood work. Troponin was elevated at 0.50 ug/L (negative is <0.08 ug/L). ECGs showed sinus rhythm with new anterolateral ST depression. The 12 lead ECG showed ST depression in leads V2-4. The 15 lead ECG showed ST depression in leads I, aVL and V2-6.

The initial diagnosis was acute coronary syndrome (ACS) and initial medical management included the following:

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Stat orders:

ASA 160 mg PO
Atorvastatin 80 mg PO
Clopidogrel 300 mg PO
Enoxaparin protocol
Nitroglycerin spray q5 min x3
Lorazepam 1 mg PO

Regular orders:

Nitroglycerin spray, 1 spray PRN
Acetaminophen 1 g q 6hrs PRN
Nitro-patch 0.4 mg x 24hr continuous
ASA 81 mg daily PO
Atorvastatin 80 mg daily PO
Clopidogrel 75 mg daily PO

The patient was transferred to the special care unit to be continuously monitored and IV access was established. Repeat troponin and ECGs were ordered for 16:00 hr and an urgent consult was sent for coronary angiography at St. Boniface Hospital. On reassessment at 16:00 hr, the patient's chest pain was still present. The patient reported the pain did improve with nitroglycerin spray, but it continued to recur. The repeat ECG still showed the slight anterolateral ST depression and now a slight ST elevation, less than 1mm in lead III. Repeat troponin was 0.85 ug/L. Cardiology at St. Boniface Hospital was consulted due to the persistent pain and now slight ST elevation to ensure optimal management. Management was continued as per above orders, in addition the patient was started in 3L nasal prongs, which maintained oxygen saturation at 97-99%. At around 18:00 hr cardiology was updated that there was no progression in the ECG changes and vitals remained stable. Cardiology recommended opioid analgesia and to report any changes in ECGs or vitals. Later in the evening the patient was still having chest pain, and a repeat ECG showed ST elevation in leads III and aVF and troponin was now 2.57. The patient's vitals remained relatively stable, with bradycardia ranging between 45-55 bpm and occasional drops below 40 bpm. The changes were discussed with cardiology and the patient was transferred via EMS to St. Boniface emergency department for cardiology consult. Upon arrival, the patient was chest pain free and was admitted to the hospital. The patient's chest pain recurred in the morning and he was brought to the cardiac catheterization lab for an urgent coronary angiogram. The mid right coronary artery (RCA) was found to have 70% occlusion and the distal RCA had 100% occlusion. Both were treated to 0% with drug eluting stents. The final diagnosis was reported as late presentation inferioposterior STEMI. The patient was admitted to the cardiac care unit for follow-up care. After several days the patient was discharged directly home with follow-up by his family doctor as an outpatient.

Literature search

A literature search regarding the clinical presentation, diagnosis and management of ACS was conducted using several databases including PubMed, UptoDate, ClinicalKey and Google Scholar. The search terms included "acute coronary syndrome", "rural management of acute coronary syndrome", and "late presentation STEMI". Additional references for this report were also found within the resources produced by the initial search results.

Discussion

Clinical Presentation

The most common presenting symptom of ACS is acute chest pain or discomfort¹. Table 1 summarizes the typical features of ischemic chest pain associated with ACS^{1,2}:

Table 1 – Typical features of ischemic chest pain

Onset	<ul style="list-style-type: none"> • Typically gradual onset
Quality	<ul style="list-style-type: none"> • Often described as discomfort instead of pain • Typically a pressure, heaviness, tightness or squeezing • Can also present as a burning pain similar to gastrointestinal (GI) pathology
Location	<ul style="list-style-type: none"> • Typically substernal or left chest pain with radiation to arms, neck, jaw, back or abdomen
Provoking factors	<ul style="list-style-type: none"> • Increased/provoked by exertion • Not altered by position or respiration

Additional symptoms are commonly associated with ACS chest pain, including diaphoresis, nausea and vomiting, shortness of breath, dizziness, and weakness^{1,2}. It is of note that presentations considered atypical for ACS are actually fairly common. It has been shown that 33% of patients with confirmed MI presented with no chest pain at all, instead having symptoms such as shortness of breath, nausea and vomiting, weakness or syncope¹. Atypical chest pain in ACS is also not uncommon. In a study of patients with confirmed myocardial ischemia, 22% presented with sharp pain and 13% with pleuritic pain¹. It is clear that ACS can have varying presentations, thus it is important to have a high index of suspicion when assessing patients. In the case of our patient, the initial presentation had features consistent with both cardiac and GI conditions. The complaint of burning epigastric/chest pain, can occur in ACS, but is also common in other conditions, such as gastroesophageal reflux disease (GERD) and peptic ulcer disease³. The fact that the pain was not worse on exertion and that it was changed with position did not fit with the characteristic features for ACS pain. The patient’s complaint of worsening pain when supine, is more consistent with GERD³. The associated symptoms of diaphoresis and weakness that our patient reported supported a cardiac cause of the epigastric/chest discomfort and appropriate investigations were ordered to confirm this suspicion.

Diagnosis

The diagnosis of ACS is based on clinical presentation, ECG changes and serum cardiac biomarker levels¹. ACS is divided into ST-elevation myocardial infarction (STEMI) and non-ST elevation ACS (NSTEMI-ACS) based on ECG changes that are described in Table 2⁴:

Table 2 – Summary of STEMI and NSTEMI-ACS ECG changes

STEMI	<ul style="list-style-type: none"> • New ST elevation of at least 1mm in 2 contiguous leads, except leads V2 and V3 • In leads V2 and V3 ST elevation must be: 2 mm or greater in men ≥ 40 yrs 2.5 mm in men < 40 yrs 1.5 mm in women
NSTEMI-ACS	<ul style="list-style-type: none"> • New ST depression of at least 0.5 mm in 2 contiguous leads, and/or • New T wave inversion of at least 1 mm in 2 contiguous leads with prominent R wave or R/S ratio greater than 1

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NSTE-ACS can be further divided into non-ST elevation myocardial infarction (NSTEMI) and unstable angina (UA), which are differentiated by cardiac biomarker levels. In acute MIs (STEMI and NSTEMI) cardiac biomarkers by definition must become elevated above the 99th percentile of the upper reference limit, indicating myocardial injury, whereas cardiac biomarkers are not elevated in UA ⁴. When diagnosing and following ACS, obtaining multiple ECGs is important, especially early on in presentation, since 20% of ECGs can initially be normal and 45% initially non-diagnostic ¹.

The ECG findings in this case were interesting because they did not initially fit with the expected findings for the final diagnosis of inferioposterior STEMI caused by distal RCA occlusion. The initial diagnosis made in this case was NSTEMI with ST depression in the anterolateral leads. The 15 lead ECG to assess for posterior involvement was unremarkable. It wasn't until later in the evening, that repeat ECGs showed ST elevation in leads III and aVF, consistent with an inferior STEMI. This case highlights the importance of repeating ECGs to monitor the progression of patients with ACS and also in consulting cardiology when ECG changes may not be as expected.

Initial Management

Distinguishing between NSTEMI-ACS and STEMI early on is important because the therapeutic goals for each condition differ ⁵. The main treatment goal in NSTEMI-ACS is to prevent total occlusion of coronary arteries and thromboembolization using antithrombotic therapy ⁵. This includes early medical management as described in Table 3 ⁶:

Table 3 – Summary of early medical management for NSTEMI-ACS

Anti-ischemic/analgesic therapy	<ul style="list-style-type: none">• Oxygen if saturation <90% or in patient in respiratory distress• Sublingual nitroglycerin, IV if sublingual is insufficient in managing pain• IV morphine if pain not controlled with nitroglycerin• Beta-blockers within 24 hrs• Statin
Anti-thrombotic therapy	<ul style="list-style-type: none">• Dual antiplatelet therapy: ASA plus a P2Y12 receptor blocker• Anticoagulation with enoxaparin, fondaparinux or unfractionated heparin

In NSTEMI-ACS cases with refractory ischemia or hemodynamic and/or electrical instability patients should be referred for urgent reperfusion via PCI ⁷. Fibrinolytic therapy is not indicated in NSTEMI-ACS as there has been no shown benefit and it may cause harm ⁷. Stable patients should be assessed for further cardiac risk in order to determine if they should be referred for early angiography and potentially PCI ⁶. This assessment can be done using various validated risk scores including, TIMI, GRACE or PURSUIT ⁶.

In STEMI, the above medical therapy in Table 3 is also initiated, but because total artery occlusion occurs the main goal of management is timely reperfusion to limit myocardial injury ⁵. Reperfusion can be done physically via percutaneous coronary intervention (PCI) or medically via fibrinolytic therapy, although PCI is the preferred method when possible as it is associated with better

outcomes⁸. The ideal time from first medical contact to PCI is less than 90 minutes, whereas the ideal time for initiation of fibrinolytic therapy is less than 30 minutes⁵. If PCI cannot be performed within 120 minutes, such as in rural communities, fibrinolytic therapy is recommended in patients having ischemic symptoms for less than 12 hrs⁸. The benefit of fibrinolytic therapy in STEMIs past 12 hrs of onset has not been established, however, in centers >120 minutes from a PCI center, it can be considered for up to a 24 hour period if a large area of myocardium is at risk, or if the patient is hemodynamically unstable⁸.

In the presented case, fibrinolytic therapy was not initiated as the patient did not meet the criteria in current ACS guidelines. Firstly, the initial diagnosis for this patient was NSTEMI, for which fibrinolytic therapy is not indicated. Secondly, once the patient's ECG showed changes consistent with a STEMI, it had already been over 24 hrs since symptom onset, which is outside the recommended time frame for this therapy. The literature reports that 8.5-40% of STEMI patients present to medical attention later than 12 hrs after symptom onset, which may limit management⁹. Although in this case the patient did present after 15 hrs of pain, presenting earlier likely would not have changed initial management since they did not meet fibrinolytic criteria on arrival at the hospital. The patient was transferred to Winnipeg and underwent PCI the following day. There is currently no definitive optimal timing of PCI in STEMIs once symptoms have been present for >12 hours, but studies support myocardial salvage is still possible past this point^{9,10}. An ongoing clinical trial is aiming to assess if there is a significant difference in patient outcomes between receiving immediate or subacute (within 24-72 hrs) PCI in late presenting STEMIs¹¹. Current guidelines do not recommend PCI in stable asymptomatic patients with complete artery occlusion if time from symptom onset >24 hrs⁸.

Conclusion

The purpose of this report was to review the clinical presentation, diagnosis and management of ACS in the context of an interesting real life case. As a medical student early in training this report reinforced several important points. First, ACS can have a variety of presentations so a high index of suspicion is required when assessing patients. Second, ECGs are the most important diagnostic tool when it comes to ACS because they are the most helpful in guiding subsequent management, and repeat ECGs are important as they may not always show expected changes. Finally, patients presenting with time from onset of ischemic symptoms >12 hours may still be eligible for revascularization.

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