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STEMI WATCH

VOLUME TWO:

DIAGNOSIS AND BEYOND

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This activity has been approved for 10.0 nursing contact hours using a 60-minute contact hour.

ACTIVITY OBJECTIVES

After reading *STEMI Watch: Diagnosis and Beyond*, the participant will be able to:
1. Discuss current scientific research and data regarding the diagnosis of ST segment elevation myocardial infarction (STEMI).
2. Discuss the pathogenesis and treatment of STEMI.
3. Cite new information regarding new drugs for STEMI and new uses for traditional drugs.
4. Identify ancillary issues of importance for healthcare providers who treat STEMI patients.
5. Discuss advances in STEMI treatment.

Physicians and nurses participate in this CME/CE activity by reading the articles, using the provided references for further research, and studying the relevant questions at the end of the book. Participants will then be directed to a website, where they will complete an online assessment to show what they've learned. They must score 100 on the assessment in order to complete the activity, but they are allowed to answer the questions multiple times if needed. After they have successfully completed the assessment, they will be directed to an online activity evaluation form. Once that is submitted, they will receive their credit letter.

TARGET AUDIENCE

This activity is intended for cardiologists, coronary care nurses and staff, emergency medicine physicians and nurses, emergency medical service providers, ECG staff, percutaneous coronary intervention center staff, and freestanding emergency department staff.

EXPIRATION DATE

March 31, 2020

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FINANCIAL DISCLOSURES

In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, Dr. Crawford, Dr. Zhao, and Dr. Macabasco-O'Connell report no financial relationships relevant to this field of study.

Editorial and Production: Ms. Coplin, Ms. Hatcher, and Mr. Landenberger report no financial relationships relevant to this field of study.

Authors: Financial disclosure information for authors can be found at the beginning of their respective articles. This publication does not receive commercial support.

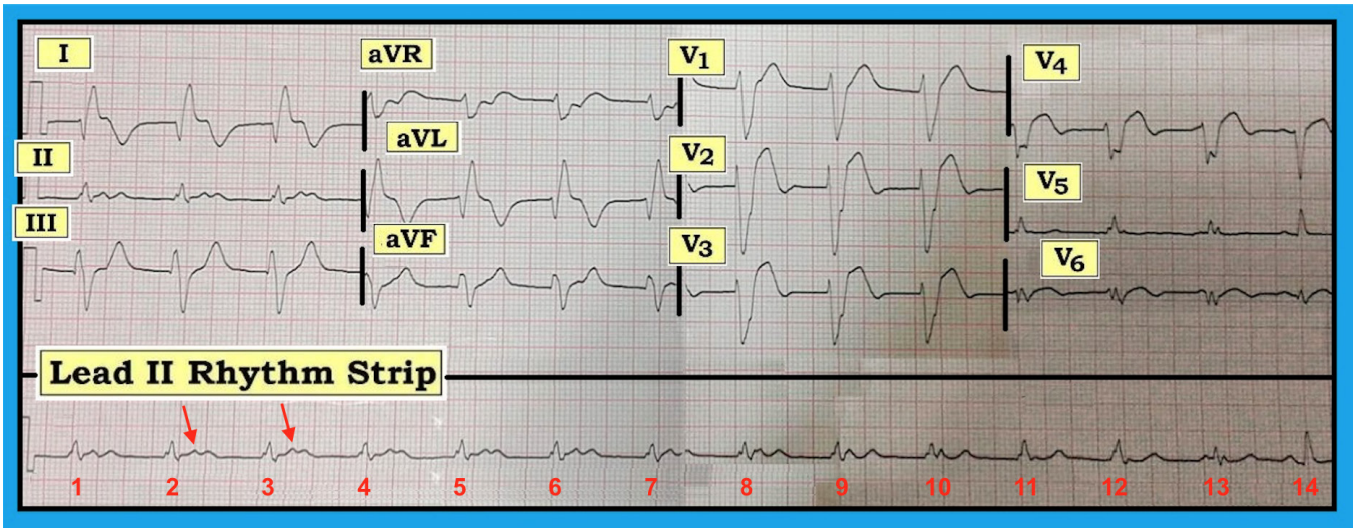
TABLE OF CONTENTS

ECG REVIEW

Wide Rhythm in a Patient with Chest Pain

By Ken Grauer, MD, Professor Emeritus in Family Medicine, College of Medicine, University of Florida

Dr. Grauer is the sole proprietor of KG-EKG Press, and publisher of an ECG pocket brain book.



The ECG in the figure above was obtained from a 44-year-old man who presented to the ED with new-onset chest pain. He was hemodynamically stable at the time this tracing was recorded. If no other history was available, how would you interpret this ECG? What do you suspect is going on clinically?

This is a challenging case. Quick perusal of the 12-lead ECG suggests that the QRS complex is markedly widened, at least in most leads on the tracing. The reason the QRS complex does not initially appear to be wide in lead II is that the terminal part of the QRS in this lead is nearly isoelectric to the baseline. That this is the case should be apparent from comparison of lead II with simultaneously recorded leads I and III. Thus, although the QRS complex does not appear to be wide in the long lead II rhythm strip at the bottom of this tracing, the QRS is wide. However, conducting sinus P waves are absent for all but the last two beats on the tracing. The presence of an almost regular wide-QRS rhythm at about 80/minute without sinus-conducted P waves defines these first 12 beats as accelerated idioventricular rhythm (AIVR).

- AIVR is an extremely common reperfusion rhythm in the setting of evolving acute ST elevation myocardial infarction (STEMI).
- On occasion, both Q waves and ST-T wave changes suggestive of ongoing ischemia/infarction may appear in ventricular beats or ventricular rhythms. This appears to be the case here, as there are Q waves, ST elevation, and deep T wave inversion in leads I and aVL beyond that expected for simple AIVR. Additionally, there are reciprocal changes in lead III, and disproportionate J-point ST elevation in leads V1, V2, and V3. In a patient with new chest pain, these findings suggest acute evolving STEMI, with probable reperfusion based on the presence of the AIVR rhythm.
- The other interesting finding relates to atrial activity. Early on, there is AV dissociation (red arrows). Eventually, sinus P waves resume prior to the QRS, with the result that the last beat on the tracing (beat #14) is a sinus-conducted "capture" beat. QRST appearance of this single sinus-conducted beat in simultaneously recorded lateral chest leads shows lack of r wave development by lead V4, and abnormal ST coving plus elevation in leads V4 and V6 that supports our conclusion of evolving STEMI.

For a further discussion of this case, please visit: http://bit.ly/2a2tvP9.

ABSTRACT & COMMENTARY

One Procedure or Two? Study Examines Staging for Multi-vessel PCI in NSTEMI

By Jeffrey Zimmet, MD, PhD
Associate Professor of Medicine, University of California, San Francisco; Director, Cardiac Catheterization Laboratory, San Francisco VA Medical Center

Dr. Zimmet reports no financial relationships relevant to this field of study.

SYNOPSIS: Patients with non-ST elevation myocardial infarction and multi-vessel disease were randomized to percutaneous coronary intervention (PCI) of all significant lesions during the index procedure, or to staged PCI. Those undergoing single-stage PCI had lower rates of major adverse cardiovascular and cerebrovascular events at 1 year, driven by lower rates of target-lesion revascularization.

SOURCE: Sardella G, et al. Single-staged compared with multi-staged PCI in multivessel NSTEMI patients: The SMILE Trial. *J Am Coll Cardiol* 2016;67:264-272.

In patients presenting with non-ST elevation myocardial infarction (NSTEMI) and multi-vessel disease for whom a percutaneous coronary intervention (PCI) is a treatment strategy, multiple questions remain about the extent and timing of treatment. This is a common scenario. Some reports have estimated that as many as 50% of NSTEMI patients present with multi-vessel disease. Although full revascularization is clearly the standard when patients undergo coronary artery bypass surgery, the landscape is more murky for PCI-treated patients. When pursuing a complete revascularization approach, how should one accomplish it? Should one approach only the "culprit" lesion during the index procedure? Should one undertake treatment of the non-culprit vessels during the same session?

The SMILE (Impact of Different Treatment in Multivessel Non ST Elevation Myocardial Infarction Patients: One Stage Versus Multistaged Percutaneous Coronary Intervention) trial, performed at two centers in Italy, addresses the latter question. SMILE was an unblinded investigation of 548 consecutive NSTEMI patients, randomized in a 1:1 fashion to single- or multi-stage PCI. Those assigned to multi-stage revascularization had PCI of only the culprit lesion at the initial procedure and subsequently underwent a second procedure between three and seven days later during the index hospitalization. Researchers used transradial access in > 80% of cases, although that number dropped to approximately 65% during the second procedure in the multi-stage group. Researchers employed fractional flow reserve (FFR) in approximately 25% of patients. The baseline characteristics of each group were similar, as were the use of drug-eluting stents, completeness of revascularization, and medical regimens at time of hospital discharge.

At one year, the rate of major adverse cardiovascular and cerebrovascular events (MACCE) was significantly lower in the single-stage group (13.63% vs 23.19%; hazard ratio [HR], 0.549; 95% confidence interval [CI], 0.363-0.828; $P = 0.004$). This difference was driven primarily by a higher rate of target vessel revascularization (TVR) in the multi-stage group (n = 40 [15.20%] vs n = 22 [8.33%]; 95% CI, 0.310-0.878; $P = 0.01$). Cardiac and overall death, myocardial infarction (MI), stroke, and hospitalization for unstable angina were not different between groups. The Bleeding Academic Research Consortium (BARC) type 1 bleeding (minor bleeding that is "not actionable" and does not generally cause the patient to seek treatment) was higher in the multi-stage group, although the more clinically-meaningful BARC types 2, 3, 4, and 5 were not significantly different between groups.

The authors concluded that in patients with NSTEMI and multi-vessel disease, single-stage PCI during the initial procedure is superior to multiple procedures in terms of MACCE and minor bleeding at 1 year.

Commentary

One of the most surprising things about SMILE is that the study was designed with the assumption that multi-stage revascularization would be superior to single. As the study reached the opposite conclusion, we are left asking what happened and whether we should believe the results.

The first thing we should realize is that this study does nothing to address what is likely the more interesting question regarding multi-vessel disease: the issue of whether complete revascularization itself is superior to more-selective revascularization. SMILE used complete revascularization as the default approach and only examined the timing of PCI.

Pushing the Envelope on STEMI Response

By Dorothy Brooks, Author, *ED Management*

Ms. Brooks reports no financial relationships relevant to this field of study.

Can EDs, cardiologists, and emergency medical service (EMS) providers do more to accelerate heart-saving care to patients with ST-segment elevation myocardial infarctions (STEMI)? An important new project, spearheaded by Duke Health and the American Heart Association (AHA), suggests the answer is yes. But findings from this work also reveal that such improvement requires a more regionalized approach to STEMI care, better coordination between emergency medicine and EMS, and a focus on more rigorous outcome indicators — a tall order in a healthcare system that is highly fragmented and influenced by competitive pressures.

The new findings cover an 18-month period of outcomes from the Mission: Lifeline STEMI Systems Accelerator. The project included 484 hospitals, 1,253 EMS agencies, and nearly 24,000 patients in 16 regions across the United States.[1] The goal was to increase the number of STEMI patients who receive percutaneous coronary intervention (PCI) within the time parameters recommended by prescribed guidelines: within 90 minutes of first medical contact with emergency responders if the patient is taken to a hospital that is capable of fully handling the PCI, or within 120 minutes if the patient must be transferred to a second hospital for PCI.

Currently, roughly 50% of STEMI patients do not receive PCI within the recommended time window. However, over the course of this project, that percentage improved among participating organizations. For patients who were brought by EMS to hospitals capable of performing PCI, the proportion receiving this intervention within 90 minutes increased from 50% to 55%. Among patients who needed to be transferred to a second hospital, the proportion grew from 44% receiving PCI within the recommended 120-minute window to 48% meeting this standard.

Although not all observers are impressed with the modest improvements achieved in this large-scale project, the authors contend that additional benefits are possible as the coordination strategies leveraged at the demonstration sites are optimized in the coming years. Further, they note that the project provides a roadmap for other health systems to follow in improving care for STEMI patients as well as other time-sensitive cardiovascular conditions. Indeed, the Mission: Lifeline program now is setting its sights on accelerating brain-saving care to stroke patients.

Target Delays

Christopher Granger, MD, a professor of medicine at Duke University School of Medicine and chairman of the AHA's Mission: Lifeline program to improve heart attack care nationally, states that a key roadblock for investigators in this area is the highly competitive nature of America's healthcare system.

"Individual hospitals do a really good job with healthcare, but they are not really working together because part of their job in terms of administration is to be a successful business, and that means being better than the competition in terms of margin-generating patient care," he explains. "It is not really in their best interests to do things that will help their competition, especially with something like cardiovascular care, which is fairly lucrative."

Another challenge is the fragmentation between different healthcare services.

"In some states, like Maryland, there is one EMS system and it is pretty organized. In other states, like North Carolina and Georgia, EMS is not really part of the medical system. It is funded through state and local government in a fragmented way, and it is part of the highway transportation system," he says. "In North Carolina, we have 640 different EMS agencies."

Granger notes that this competition and fragmentation can slow down care when there are not common approaches or protocols to follow regarding what a paramedic, emergency physician, and an interventional cardiologist will do when someone is having a heart attack.

"The paramedic goes to the scene and diagnoses that the patient is having a heart attack, and then he or she has to decide where to go. Maybe there are two PCI-capable hospitals that are equidistant or the paramedic doesn't know if one hospital is ready or not," he says. "In the past, the paramedics have ended up having to call around or even to drive to the nearest ED before anything is really done, and then that ED might call in the catheterization lab, and that results in an additional delay. Pretty soon you've got these long delays, and every minute of delay after a heart attack begins there is irreversible injury to the heart muscle and greater likelihood of death and heart failure."

Given that outcomes in the case of STEMI are highly dependent on providing rapid care, investigators have seized on a big opportunity to improve care by doing a better job

CME/CE QUESTIONS

To earn credit for this module, log in to _____ to take the post-test.

1. The happy heart syndrome is an unusual variant of:
   a. the holiday heart syndrome.
   b. takotsubo syndrome.
   c. stress cardiomyopathy.
   d. acute cerebral bleed associated left ventricular dysfunction.

2. A study in non-ST elevation myocardial infarction patients comparing initial complete revascularization by percutaneous coronary intervention (PCI) vs. culprit lesion PCI followed at another time by complete revascularization by PCI showed initial complete PCI:
   a. reduced target vessel revascularization.
   b. reduced mortality.
   c. increased stroke.
   d. increased bleeding.

3. The goal of the Mission: Lifeline STEMI Accelerator project is to increase the number of STEMI patients who receive percutaneous coronary intervention (PCI) within the time parameters recommended by guidelines: within _____ minutes of first medical contact with emergency responders if patients are taken to a hospital that is capable of fully handling the PCI, or within ____ minutes if the patients must be transferred to a second hospital for PCI.
   a. 20 minutes, 40 minutes
   b. 30 minutes, 50 minutes
   c. 50 minutes, 70 minutes
   d. 90 minutes, 120 minutes

4. Christopher Granger, MD, a professor of medicine at Duke University School of Medicine and chairman of the AHA's Mission: Lifeline program to improve heart attack care nationally, states that a key roadblock for investigators in this area is:
   a. lack of data.
   b. the highly competitive nature of America's healthcare system.
   c. financial constraints.
   d. provider inertia.

Mimics of ST Elevation Myocardial Infarction (STEMI)

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Dr. Velez, Dr. Hockstein, Dr. Furmaga, and Dr. Smith report no financial relationships relevant to this field of study.

Early in my training, I remember reading an article that concluded 25% of myocardial infarctions (MIs) were missed. I know that we physicians are not perfect, but are we that bad? We miss a quarter of patients with an acute infarction who present to the ED? When I delved into this literature about missed MIs, I found that this "miss" rate was being determined by analysis that had nothing to do with ED patients. What were being determined were silent infarctions — those that did not produce enough clinical symptoms to prompt patients to come to the ED. Interestingly, a May 16, 2016, publication in Circulation *found that of all MIs observed in about 9,400 individuals initially free of cardiovascular disease and followed for almost nine years, more than 45% were silent.*

When I reviewed the ED literature on missed MIs, the observed miss rate varied between 2% and 4%. Thankfully, not a quarter, but also not zero. There were multiple factors associated with missing an MI, including patient and physician factors. Two ancillary testing factors were also associated with missing an infarction: the insensitive cardiac biomarker tests available in that era and physician misinterpretation of the initial ECG. I believe the miss rate has decreased further with the widespread adoption of sensitive and specific cardiac biomarkers used with a serial testing protocol. One factor that still bedevils emergency physicians is the appearance and potential for misinterpretation of the initial ECG.

The opposite of a missed infarction is a false-positive overcall of infarction, primarily due to disorders that can mimic an acute infarction by producing ST segment elevation. This article discusses several of these conditions that are important for the emergency physician to recognize to appropriately manage patients in the ED and to better distinguish acute infarction from other conditions on the ECG.

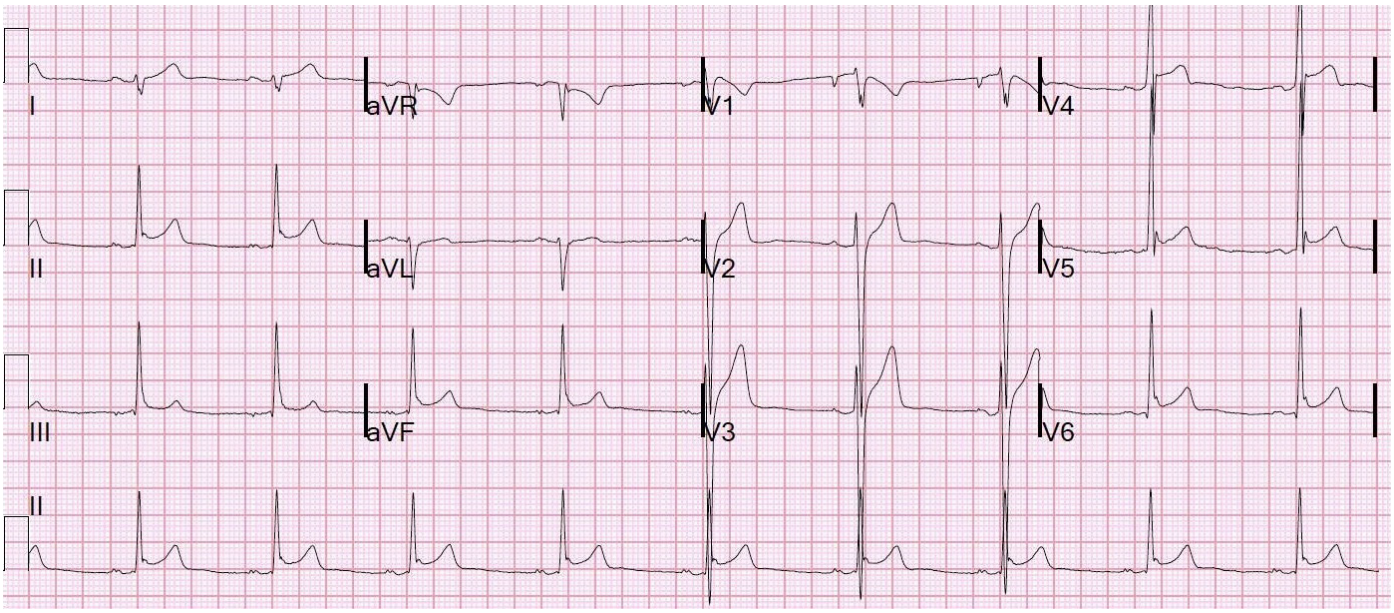
— J. Stephan Stapczynski, MD, FACEP

One common axiom in emergency medicine is that ST segment elevation in an emergency department (ED) patient with chest pain should be assumed to be an acute myocardial infarction (AMI) until proven otherwise. Like many axioms in emergency medicine, it embodies an "assume the worst and rule it out" approach to evaluating acute symptoms. While such a principle concerning ST segment elevation may be cautious and prudent, it is only one factor to consider when assessing patients with chest pain. The physician is also aware of the patient's history, physical exam findings, and other features of the electrocardiogram (ECG) when determining the potential cause of ST segment elevation. In fact, the majority of ST segment elevation seen in ED patients with chest pain is not due to AMI.[1] Thus it is important for emergency physicians to have an understanding of the differential diagnosis of ST segment elevation.

In 2013, the American College of Cardiology Foundation and the American Heart Association (ACCF/AHA) revised the electrocardiographic definition of ST elevation myocardial infarction (STEMI) to: "new ST elevation at the J point in at least 2 contiguous leads of ≥ 2 mm (0.2 mV) in men (≥ 2.5 mm in men under 40 years old) or ≥ 1.5 mm (0.15 mV) in women in leads V2–V3 and/or of ≥ 1 mm (0.1 mV) in other contiguous chest leads or the limb leads."[2] In the updated guidelines, a presumably new left bundle branch block (LBBB) in isolation is no longer considered STEMI equivalent. Moreover, the American College of Cardiology (ACC) emphasized that AMI is a syndrome, a constellation of clinical findings, including but not limited to findings on the 12-lead ECG that are concerning for an acute infarct, but also including the subsequent release of biomarkers indicative of myocardial necrosis.

The mechanism by which ST segment elevation occurs in an AMI is not completely elucidated;[3] however, it is clear that ST elevation occurs reliably with transmural and subepicardial myocardial infarctions. In a classic study conducted in 1960, ST segment elevation was described as an "injury current,"[4] after observing its presence in a canine myocardium after ligating its supplying coronary artery. In this experiment, the injured myocardium displayed simultaneous areas of depolarized and repolarized tissue, which

Figure 1. Early Repolarization

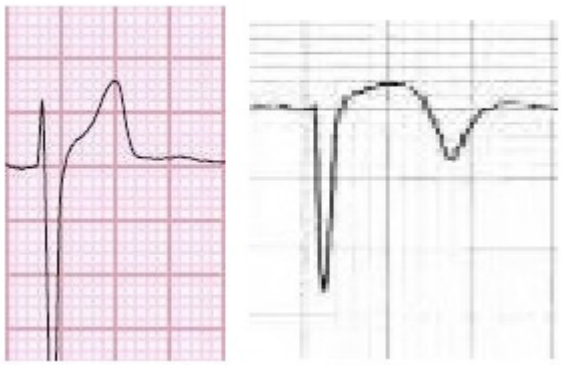


Early repolarization showing J-point elevation in multiple contiguous leads and slurring and/or notching on the downstroke of the R wave in leads II, III, aVF, V5, and V6.

LBBB, but a variant also is encountered in a right ventricular paced pattern (as both of these cause ventricular depolarization to happen from right to left). A new LBBB was once regarded as a STEMI equivalent; however, after observing a relatively low frequency of acutely obstructing coronary lesions in ED patients with chest pain and a new LBBB, that recommendation was removed from the 2013 ACCF/AHA STEMI Management Guidelines.[2] In hemodynamically stable patients with a presumed new LBBB, evaluation of their symptoms requires both measurement of cardiac biomarkers and observation. In patients with hemodynamic compromise (including acute heart failure), revascularization should be emergently considered.[13]

The Sgarbossa criteria[14] can help guide the decision for emergent catheterization and coronary intervention in the presence of both new and old LBBBs. The normal state of LBBB is described by the "rule of appropriate discordance."[15] This idea is that ventricular repolarization (ST-T) occurs in the opposite direction of the majority of the ventricular depolarization (QRS), which manifests itself as the net polarity of the QRS and T wave being opposite from each other. Thus an ST segment in the same direction as the QRS (also known as "concordant") is indicative of ischemia/infarction. Conversely, in LBBB, the QRS in V1-V3 is always negative; therefore, the normal condition of the ST segment in these leads is ST elevation. Thus, excessive discordant ST segment elevation in leads V1-V3 is indicative of an anterior MI. The modified Sgarbossa criteria[16] determine "excessive discordance" by a proportion rather than an absolute number (these criteria have been validated[17]):

Figure 2. ST Segment Concave and Convex



Concave or sagging ST segment on the left and convex (or non-concave) ST segment on the right.

1) Concordant ST elevation ≥ 1 mm in any single lead (*see Figure 4*);

2) Concordant ST depression ≥ 1 mm in just one of leads V1-V3;

3) Proportionally excessive discordant ST elevation as defined by a ratio of ST elevation at the J-point, relative to the depth of the S wave (ST/S ratio), of ≥ 0.25 (this has replaced the original third criterion of ST elevation, which was an absolute number [≥ 5 mm]). (*See Figure 4.*)

The original Sgarbossa decision tool assigned points to each criterion: 5 points for concordant ST elevation > 1 mm in any lead; 3 points for concordant ST depression > 1 mm in leads V1 to V3; and 2 points for discordant ST elevation

also increase myocardial oxygen demand. Chest pain is a common complaint of patients who present to the ED after using cocaine. The COCHPA (Cocaine Associated Chest Pain) study group determined that there was no clinical parameter to predict which patients were at very low risk for cocaine-induced MI.[50] As cocaine-associated MI is a well-established entity, and cocaine users are at an increased risk for accelerated atherosclerosis,[51] all patients with cocaine use and chest pain should be treated as potentially having acute coronary syndrome.[52]

Conclusion

Emergency physicians evaluate patients with chest pain every day. In patients with ECG changes consistent with a STEMI, the primary goal is to salvage myocardium by rapid reperfusion by an interventional cardiologist (when available). However, in many clinical scenarios, non-ischemic causes of ST segment elevation need to be considered. A clinical context and a thorough evaluation of the ECG will often provide clues to some of these STEMI mimics.

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EXECUTIVE SUMMARY

- Classic findings of aortic dissection are uncommon.
- Consider dissection when there is sudden onset of chest pain associated with visceral symptoms.
- Intravenous contrast-enhanced CT is the imaging modality that is often readily available and highly accurate.
- Intravenous beta-blockers should be the initial therapy in patients with the goal heart rate of around 60 beats per minute and systolic blood pressure of less than 120 mmHg.

and a delayed diagnosis. For example, women are more likely than men to present with congestive heart failure or altered mental status.[7,8] As a result, women have a higher mortality rate.

Etiology and Pathophysiology

The aorta is a large, elastic artery composed of three layers: (*see Figure 2*)

- Intima: the inner layer covered in a single layer of endothelium;
- Media: the thickest layer composed of elastic tissue, smooth muscle cells, and collagen;
- Adventitia: the outer layer composed of loose connective tissue, which contains the vasa vasorum (the blood supply to the aortic wall).

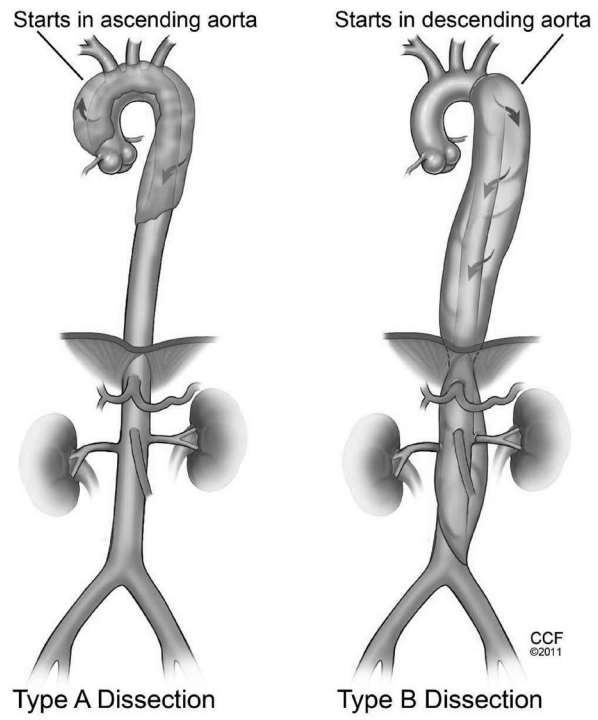
Aortic dissection is the result of a tear between the aortic intima and the inner layer of aortic media that allows blood to enter and split the layers of the aortic media. This leads to blood accumulation in the potential space called a false lumen. Pulsatile blood tears through the length of the aorta and disrupts flow in the true lumen, resulting in hypoperfusion of critical organs including brain, spine, gut, heart, and kidneys. Blood also may accumulate freely within the pericardium, stifling cardiac output. Free aortic wall rupture into the pleural space may result in blood loss and hypotension, along with respiratory compromise. This injury to the aortic wall can occur as a result of multiple factors: abnormal aortic wall stress, structural irregularities of the aortic media, or injury to the aortic wall.

Cystic medial necrosis, or cell death of the smooth muscle cells of the media, is described as a "prerequisite" for aortic dissection.[9] Connective tissue disease, such as Marfan syndrome, as well as chronic hypertension are both associated with this cell death process that weakens the aortic structure and predisposes patients with these conditions to aortic dissection. Aortic dissection also can occur via spontaneous hemorrhage of the vasa vasorum due to hypertension. This results in an intramural hematoma within the wall of the aorta and weakening of the media. Intramural hematomas are associated most commonly with Type B dissections in elderly patients.[10]

Genetic disorders are associated with aortic dissection. Marfan syndrome, Ehlers-Danlos syndrome, and aortoannular ectasia are all associated with increased risk of aortic dissection due to weakness of the aortic wall. Marfan syndrome is present in 50% of those presenting with aortic dissection who are younger than 40 years of age, and most patients with Marfan syndrome who present with dissection also have a family member who suffered a dissection.[11] Collagen vascular disease affects the mechanical properties of the aortic wall and the flow of blood through the aortic lumen, which contributes to weakness in the aortic wall and increased risk of dissection. Patients with bicuspid aortic valves also have a 5-18 times greater risk of aortic dissection compared to the general population, due to abnormal wall stress and presence of less elastic tissue in the aortic wall.[12,13]

Hypertension is considered one of the most significant risk factors for aortic dissection. About 70-90% of patients with aortic dissection also have a diagnosis of hypertension.

Figure 1. Aortic Dissection Type A and Type B



patients with diabetes, prior diagnosis of aortic disease, or dissection of iatrogenic origin, and in older patients. There is increased mortality in these patients, often related to delay in diagnosis.[20]

Physical exam findings described as "typical" in textbooks often are not present in patients with aortic dissection, and their absence cannot be relied upon to adequately exclude a patient with a high pretest probability of aortic dissection. Systolic blood pressure difference of > 20 mmHg previously has been reported as convincing evidence for aortic dissection,[21] but new evidence suggests that up to 20% of unaffected people may have this finding.[10] Similarly, a 2002 meta-analysis demonstrated that only 31% of patients with aortic dissection had pulse deficits or blood pressure differentials, and although the presence of these findings increased the possibility of aortic dissection, absence of the finding did not adequately exclude aortic dissection.[22]

High-risk physical findings include:

- Pulse deficits or systolic blood pressure differential: both associated with worse outcomes;
- New-onset aortic regurgitation (especially if complicated by pericardial tamponade): new diastolic murmur, jugular venous distention, tachycardia and muffled heart sounds, hypotension;
- Syncope: present 13% of the time in aortic dissection, resulting from acute cardiac dysfunction or vascular outflow obstruction; associated with increased rates of stroke or other neurologic deficits and higher rates of death in the hospital;[23]
- Hypotension or shock state: associated with worse outcomes, especially if related to aortic rupture or pericardial tamponade, the most common causes of death in Type A dissections;
- Focal neurologic deficits: *stroke symptoms plus chest pain should raise a high level of concern for aortic dissection.* Proximal arch dissections may cause intracranial deficits, while distal arch dissections can cause spinal cord and lower extremity weakness. It is critical to identify the presence of aortic dissection during stroke, as thrombolytic medications are contraindicated in this circumstance and can result in death;
- Mesenteric ischemia is the most common gastrointestinal complication of aortic dissection and also the most frequent cause of death in Type B dissection.[8]

Seemingly benign presentations, such as sore throat, hoarseness, or dysphagia, can be associated with mass effect from compression of the proximal aorta and aortic arch. (*See Table 2.*)

Table 2. Symptoms of Aortic Dissection

Symptom	Structure Being Compressed by Aortic Dissection
Dyspnea or stridor	Trachea
Hoarseness	Recurrent laryngeal nerve
Horner's syndrome (miosis, ptosis, anhidrosis)	Sympathetic chain (ipsilateral)
Dysphagia	Esophagus

Diagnosis

Routine screening tests available to evaluate patients with chest pain in the ED are usually of limited utility in the diagnosis of aortic dissection. The primary value is to confirm the presence of another disease process, although aortic dissection can coexist with many other etiologies of chest, back, or abdominal pain. Electrocardiography, serum lab markers, and chest radiography are tests that can be obtained rapidly and usually routinely. CT angiography is the most useful imaging test to detect aortic dissection in the ED, but transesophageal echo also has some utility in the management of unstable patients.

Electrocardiography

The IRAD investigators found that the electrocardiogram (ECG) was normal in about 30% of cases. Another 2016 study retrospectively analyzed ECGs from patients with acute type A aortic dissections and found that 38% of the time, acute ischemic changes were present, with 16% of patients having a concurrent ST-elevation myocardial infarction (STEMI). This may cause some clinicians concern, since the treatment for a STEMI is significantly different than the treatment of aortic dissection. The 2010 American Heart Association (AHA) guidelines, however, state that patients with a low-risk history and physical exam for aortic dissection who present with ECG changes consistent with an acute coronary syndrome should be managed with coronary reperfusion therapy. Further imaging of the aorta should be undertaken only if no culprit lesion is detected on angiography.

Chest Radiography

Chest radiography is neither specific nor sensitive for aortic dissection, and is of limited value to make a diagnosis in most patients. A completely normal chest radiograph reduces the probability of aortic dissection in low-risk patients, and is a recommended study in low- and intermediate-risk patients. Contrast-enhanced CT of the chest is the diagnostic test of choice in those at high risk. A normal chest X-ray should not be used to rule out the disease in high-risk patients. A widened mediastinum or abnormal aortic contour increases the likelihood of aortic dissection, but was found to have a sensitivity of 64% in one prospective study.[24]

Figure 3. Contrast-enhanced CT of Type A Dissection at Aortic Root

The images show the true (B) and false (A) lumens. The "bird's beak sign" is an acute edge of the hematoma at the distal end of the dissection where the dissection is growing longer. The left main coronary is seen arising from the true lumen and is filled with contrast.

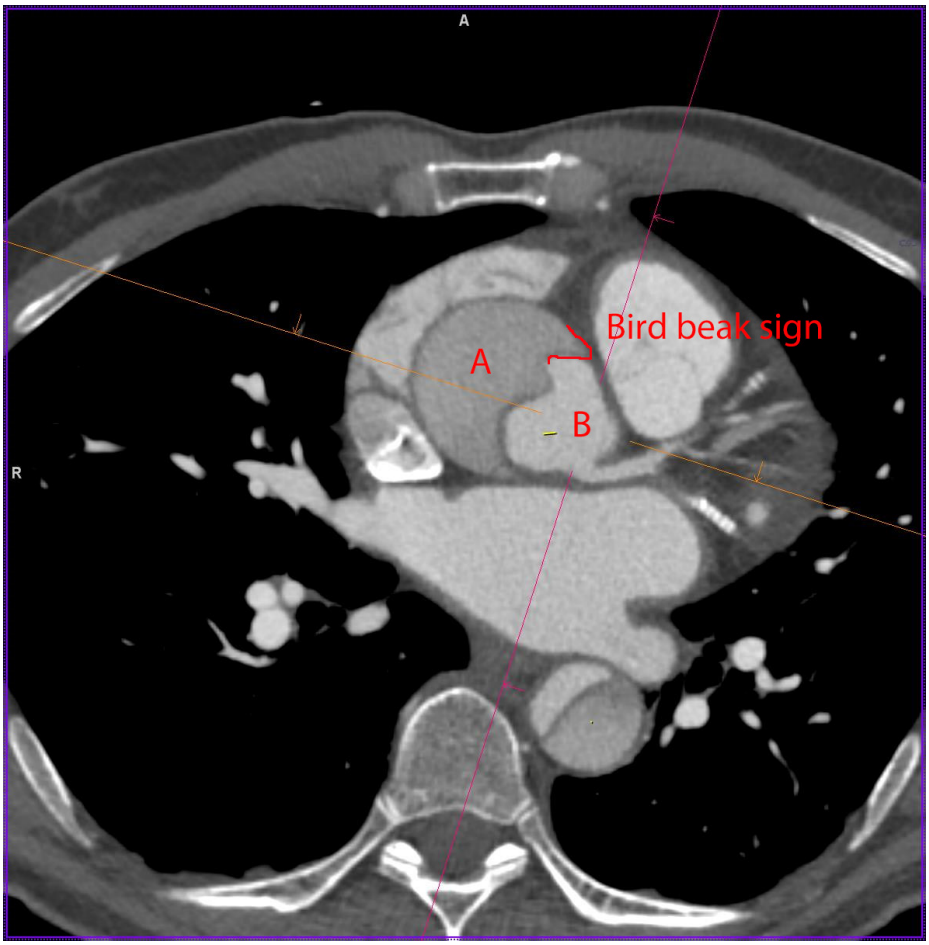


Image courtesy of Paul Schoenhagen, MD, Imaging Institute, Cardiovascular Imaging, Cleveland Clinic

Laboratory Analysis

Use of the D-dimer as a screening test to exclude aortic dissection in low-risk patients has not been evaluated in a large prospective trial. The AHA guidelines recommend against the use of D-dimer as a diagnostic tool in patients being evaluated for aortic dissection because of a lack of evidence supporting its use.[8] The American College of Emergency Physicians (ACEP) guidelines state that although there are limitations to use of the D-dimer, using the lab test potentially may reduce unnecessary radiation exposure to some patients. Nonetheless, the ACEP guidelines also counsel "do not rely on D-dimer *alone* to exclude the diagnosis of aortic dissection."[25]

Computed Tomography

Newer multidetector CT (MDCT) scanners with 64 or more detector rows have nearly perfect specificity and sensitivity and are the recommended advanced imaging test for high-risk patients. They are available at most institutions and provide relatively rapid results. MDCT has the advantage over the other imaging modalities with the ability to image and reconstruct the entire aorta in three dimensions.[26] Evaluation for aortic dissection should be performed with triphasic CT angiography of the aorta (CTAA), which consists of unenhanced, arterial, and venous images. The initial noncontrast images should image from the lung apex to the upper abdomen. The arterial scans are obtained by administering a bolus-tracked set of images from lung apices to the groin. The delayed images (1-2 minutes later) assess for late filling of a false lumen, detect abdominal organ malperfusion, and contrast extravasation from a ruptured aorta. Electrocardiographic gating reduces overdiagnosis of aortic dissection due to motion artifact, and is becoming the standard of care. It also better detects the presence of complications of the proximal