A 50-year-old man presents to the emergency department (ED) with midsternal chest discomfort. The pain started at 3:00 AM and was 6/10 in intensity; it was relieved by 2 sublingual nitroglycerin tablets. The patient had another episode of chest pain at 11:00 AM, this time with radiation toward his left arm. The pain was 10/10 in intensity and was associated with shortness of breath, nausea, and diaphoresis but no vomiting.

The patient was brought to the ED by ambulance. On the way, bradycardia (40 beats per minute) developed; it was treated with 0.5 mg of intravenous atropine.

The patient has a history of coronary artery disease, type 2 diabetes mellitus controlled with oral medications, hypertension, and hyperlipidemia. He is a current smoker (3 packs per day).

His ECG in the ED is shown here.

Which of the following is the most likely diagnosis?

A. Acute pericarditis
B. Inferior myocardial infarction (MI) with the culprit artery being the left anterior descending artery
C. Inferior MI with the culprit artery being the left circumflex artery
D. Inferior MI with the culprit artery being the right coronary artery proximal to the right ventricular branch
E. Inferior MI with the culprit artery being the right coronary artery distal to the right ventricular branch

(Answer and discussion begin on next page.)

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DISCUSSION

Acute MI of the inferior wall results from occlusion of the right coronary artery in 80% of cases or the left circumflex coronary artery in 20%. Patient outcome depends largely on which artery is affected; thus, the presumptive identification of the culprit artery on the basis of the ECG recorded at admission is of clinical importance.\(^1\)

ST-segment elevation greater in lead III than in lead II and ST-segment depression of more than 0.5 to 1 mm in leads I and aVL suggest involvement of the right coronary artery rather than the left circumflex artery with a sensitivity of 79%, specificity of 100%, negative predictive value of 88%, and positive predictive value of 100%. The association with ST-segment elevation in lead V\(_1\) suggests proximal occlusion (before the right ventricular branch) of the right coronary artery with associated right ventricular infarction.\(^1,6\)

If the ST segment is more elevated in lead III than in lead II and a 0.5 mm ST segment elevation is noted in lead I, this will favor the left circumflex artery as the culprit vessel. If the ST segment is isoelectric in lead I, the \((\sum \downarrow ST \text{ in leads V1 to V3}) / (\sum \uparrow ST \text{ in leads II, III, and aVF})\) should be done. A ratio greater than 1 implies that the left circumflex is the culprit vessel. Conversely, a ratio less than 1 indicates that the culprit vessel is likely to be the right coronary artery.\(^1,6\)

On the other hand, if the ST-segment elevation in lead II is greater than that in lead III, with an isoelectric or elevated ST segment in lead aVL, the left circumflex artery is likely to be the culprit vessel with a sensitivity of 83%, a specificity of 96%, a negative predictive value of 93%, and a positive predictive value of 91%.\(^1,6\)

HOSPITAL COURSE

The patient was treated with 50 mg of tenecteplase and started on heparin, intravenous metoprolol, nitroglycerin infusion, and aspirin. After 1 hour, the following ECG shown in Figure 1 was recorded.

![ECG](image)

**Figure 1** – This ECG was recorded 1 hour after the patient was treated with 50 mg of tenecteplase and started on heparin, intravenous metoprolol, nitroglycerin infusion, and aspirin.

**What is the diagnosis?**

A. Ventricular tachycardia with atrioventricular (AV) dissociation

B. Junctional escape rhythm with complete heart block

C. Normal sinus rhythm with premature ventricular complexes

D. Accelerated idioventricular rhythm, with fusion beats and normal sinus rhythm

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**Answer: E**
What would be the best therapy for the patient at this time?

A. Amiodarone, 300 mg IV by rapid infusion, then 0.5 mg/min IV for the next 24 hours

B. Amiodarone, 15 mg/min IV for 10 minutes (150 mg), then 1 mg/min IV for 6 hours (360 mg), then 0.5 mg/min IV for 18 hours (540 mg)

C. Lidocaine, 50 to 100 mg IV over 2 to 3 minutes (loading dose), 0.02 to 0.05 mg/kg at a rate of 1 to 4 mg/min IV (maintenance)

D. Continue with the current medical management with the addition of a glycoprotein IIb/IIIa inhibitor and take the patient emergently for a percutaneous coronary intervention

E. Continue with the current medical management because the thrombolytic therapy was effective

Answer: E

The patient’s repeated ECG a few hours later is shown in Figure 2.

DISCUSSION

Accelerated idioventricular rhythm. An accelerated idioventricular rhythm (AIVR), also called “slow VT,” arises below the atroioventricular (AV) node and has a rate between 50 and 100 or 120 beats per minute. It may be the result of sinus pacemaker failure, and therefore be an escape rhythm, or it may represent an abnormal ectopic focus in the ventricle that is accelerated by sympathetic stimulation and circulating catecholamines. The arrhythmia usually begins with a fusion beat. The onset is gradual (nonparoxysmal). It can be regular or irregular and occasionally can show sudden coupling, suggesting the presence of exit block. Additional characteristics of AIVR are listed in the Table.

Several conditions, including myocardial ischemia (especially inferior wall ischemia or infarction), digoxin toxicity, electrolyte imbalance (eg, hypokalemia), and hypoxemia may accentuate the phase-4 depolarization in the subordinate pacemaker tissues of the AV junction or His-Purkinje system, thus increasing the rate of impulse generation. Frequently, when inferior wall ischemia is present, the subordinate pacemaker acceleration coexists with sinus node depression. The latter permits escape and domination of the non-sinus pacemaker func-
tion. AIVR occurs in up to 50% of patients with acute MI. Some studies have suggested an association with reperfusion following thrombolytic therapy. However, AIVR is neither a sensitive nor very specific marker for successful reperfusion.7,9

Fusion beats often develop at the onset and termination of arrhythmia, which occurs when the cardiac pacemaker foci are competing for control of ventricular depolarization. Because of the slow rate, capture beats also are common. As a result of the slow rate and nonparoxysmal onset, precipitation of more rapid ventricular arrhythmias rarely is observed.

Most episodes are transient and require no treatment. Furthermore, treatment is contraindicated if AIVR is an escape rhythm, since suppression of the pacemaker focus will result in asystole. Rhythm termination generally occurs gradually, while the underlying sinus rhythm accelerates or the AIVR slows down.9

Capture beats versus fusion beats. If the AV node and the His-Purkinje system are not refractory to atrial impulses, AV conduction can occur. This results in a capture beat whereby ventricular conduction occurs over the normal conduction system, resulting in a normal-appearing (narrow) QRS complex. A capture beat occurs at a shorter RR interval than the RR interval of the AIVR. AV conduction also may occur simultaneously with depolarization of the ventricular focus. In this instance, the ventricle will be depolarized in part over the normal pathway and in part from the ventricular focus. The resulting QRS complex will be intermediate in morphology between a normal QRS and a QRS of ventricular origin. In this instance, the RR interval will not change. This is called a fusion beat (Figure 3).

**Table – Characteristics of accelerated idioventricular rhythm**

- Similar to ventricular tachycardia with slower rate
- P waves are present, with no association to the QRS complex
- P waves become closer and closer to the QRS complex, until they disappear in the QRS complex; at the same time, the QRS complex becomes wide and its shape changes
- Rate of wide QRS complexes is between 60 and 120 beats per minute
- It typically follows reperfusion in patients with acute myocardial infarction

HOSPITAL COURSE

The patient underwent a left heart catheterization that showed normal left anterior descending and left circumflex vessels, with a long eccentric area of 80% to 90% narrowing in the right coronary artery. This was treated by angioplasty with a drug-eluting stent. His left ventriculogram showed an ejection fraction of 30% to 35% with akinesis of the apex and severe hypokinesis of the basal and inferior walls. The patient tolerated the procedure well and was discharged home in stable condition.

**REFERENCES:**