



CASE REPORT

SERIAL ELECTROCARDIOGRAMS SHOW ACUTE ONSET OF INVERTED P WAVES IN A 62-YEAR-OLD MALE WITH CHEST PAIN: A CASE REPORT

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Recommended Citation: Faber B. (2024). Serial ECGs show acute onset of inverted P-waves in a 62-year-old male with chest pain: A case report. *International Journal of Paramedicine*. (5), 133-140. <https://doi.org/10.56068/ZMXE2675>. Retrieved from <https://internationaljournalofparamedicine.com/index.php/ijop/article/view/2728>.

Keywords: junctional rhythms, retrograde P waves, iatrogenic events, emergency medical services, EMS, paramedicine

Received: March 26, 2023
Revised: August 6, 2023
Accepted: September 18, 2023
Published: January 5, 2024

Funding: External funding was not used to support this work.

Disclosures: The author declares no conflicts of interest or disclosures.

Acknowledgements: The author wishes to thank Ken Grauer, MD for his assistance with the manuscript. Acknowledgement also given to Matt Morgan, MD and Aaron Johnson, AEMT-CC.

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ABSTRACT

Iatrogenic events are common causes of EMS encounters. This report presents the case of a 62-year-old male who presented with new-onset chest pain after his primary care provider increased his diltiazem dose to better control atrial fibrillation. The patient presented with nausea, vomiting, diaphoresis, and 8/10 sub-sternal chest pain. Sequential ECGs captured the initiation of a junctional rhythm indicated by negative (inverted) P waves and a shortened PR interval. The patient had also been prescribed dofetilide (Tikosyn), which can raise diltiazem levels. The case emphasizes the importance of recognizing arrhythmias associated with retrograde A/V conduction and raises awareness of potential iatrogenic events as causes for prehospital encounters.

INTRODUCTION

Iatrogenic events, defined as harm resulting from the actions of healthcare providers, are common reasons for emergency medicine encounters (Peer & Shabir, 2018). Schwendimann et al. (2018) found that up to 10% of hospital inpatients have experienced at least one iatrogenic event, with half preventable and 7.3% fatal. In their study of admissions to 11 Massachusetts hospitals, Bates, Levine, and Salmasian (2023) found that adverse events occurred in 23.6% of admissions, and 32.3% of these events caused harm that required intervention or recovery.

Medication errors are a common and often serious cause of patient harm (Baumgartner et al., 2018; Wittich et al., 2014; Kohn et al., 2000). Simpson and Kovach (2021), in a retrospective study of 126 older patients in a post-acute rehabilitation center, found that of 578 new reported problems, 41.7% were iatrogenic adverse events, and medication-related events were common. Insani, Whittlesea, Alwafi, et al. (2021), in their systematic review of adverse drug reactions

(ADR) in primary care, found a pooled prevalence of 8.32%, with cardiovascular system drugs being the most commonly reported medication class. Similarly, Wong, Lee, Sarkar, and Sharma (2022), in their study characterizing ambulatory-care-related adverse events, found that the largest group of events were medication-related (17%). Sheikh & Bates (2014) report that pediatric and geriatric patients, as well as patients who are physiologically compromised, have complex underlying medical conditions, and have behavioral health issues are at greatest risk for iatrogenic events.

This case report presents a prehospital encounter wherein an increase in a patient's daily diltiazem dose resulted in the acute development of an accelerated junctional rhythm. As we show below, the change in conduction was captured by serial prehospital ECGs. The case emphasizes the importance of recognizing the arrhythmias associated with retrograde A/V conduction, maintaining a high index of suspicion for potentially iatrogenic reasons for patient complaints, and conducting serial ECGs in acute onset chest pain. Patient identifiers have been changed or redacted from the case.

CASE REPORT

A 65-year-old male called 911 for new onset sub-sternal chest pain, shortness of breath, nausea, and diaphoresis. He had a history of atrial fibrillation and hypertension and his medications included pantoprazole for GERD, hydrochlorothiazide and lisinopril for hypertension, and dofetilide and diltiazem (Cardizem) for atrial fibrillation. He had no history of myocardial infarction. The 911 dispatcher advised the patient to take 325 mg of aspirin. A few hours before this event, his primary care provider increased his daily diltiazem dose from 180mg to 240mg to better control his atrial fibrillation.

CLINICAL FINDINGS

On EMS arrival, the patient appeared anxious. He was alert and oriented, had a GCS of 15, and could answer questions appropriately and in full sentences. He stated that he began to feel his symptoms approximately one hour ago and was now experiencing 8/10 chest pain. The pain was continuous, increased with exertion, and was associated with increased work of breathing.

He had regular radial pulses with a heart rate of 72 and a blood pressure of 120/75 mmHg (MAP 90). His lungs sounds were clear bilaterally with a respiratory rate of 16 breaths per minute and an SpO₂ of 97% on room air. The patient did not use oxygen at home. His temperature was 96.7 F. His blood glucose was 104 mg/dL.

The first of three 12-lead ECGs, recorded before the ambulance left the scene (Figure 1), showed a normal sinus rhythm, a premature ventricular complex (PVC), and slight straightening of the ST segment in V2 and V3 but no ST elevations greater than 1 mm. A second ECG recorded 6 minutes later (Figure 2) showed a regular rhythm at 68 bpm and negative (inverted) P waves. The PR interval was shortened to 108 ms (normal 120-200 ms), and the QRS duration was 107 ms. The ST straightening in V2 and V3 was resolved, and no leads showed ST elevation greater than 1 mm. A third ECG recorded

13 minutes later (Figure 3) showed a regular rhythm with a rate of 71 bpm, negative P waves, and a PVC. The PR interval remained short at 112 ms, and the QRS duration was 106 ms.

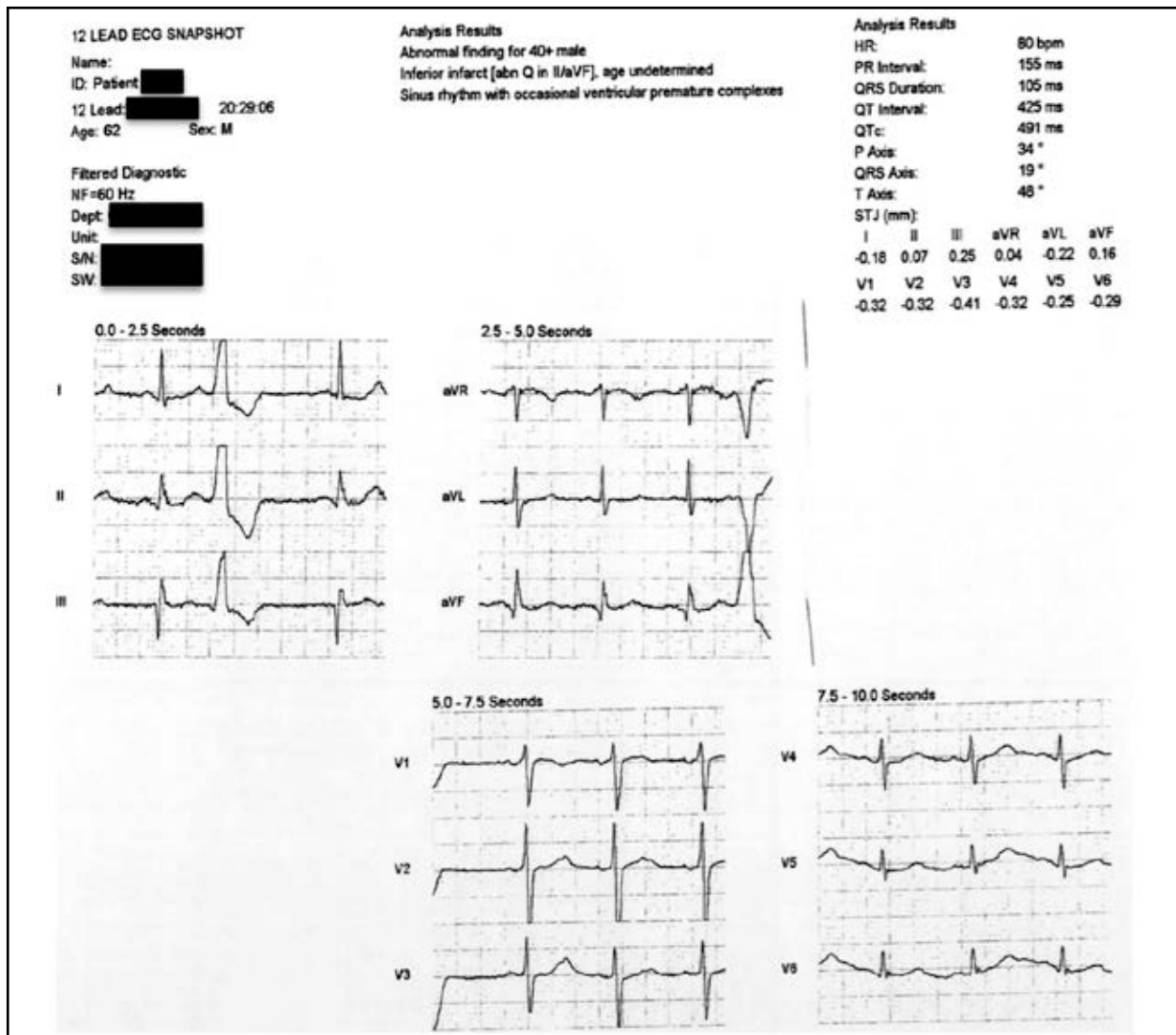


Figure 1. Twelve-lead electrocardiogram #1 performed at 20:29. The EKG shows a normal sinus rhythm with a rate of 80 bpm. The PR interval is 155 ms (normal range 120-200 ms). The QRS complex is normal, if slightly extended, in duration at 105 ms (normal range 70-100 ms). There is a premature ventricular complex (PVC).

The patient remained anxious throughout the encounter, and his GCS remained 15. Repeated vitals recorded blood pressures of 123/84 mmHg (MAP 97) and 131/85 mmHg (MAP 87). His SpO2 remained 96% on room air, and his respiratory rate remained 16 breaths per minute. His chest pain remained unchanged.

He was treated prehospitally with 4 mg of ondansetron IM and 250 ml of intravenous normal saline, and patient care was transferred to the Emergency Department (ED) of

a rural community hospital. The ED course was uneventful, and all the patient's labs returned normal. The initial hospital EKG also showed a junctional rhythm. The patient converted to a normal sinus rhythm after several hours. After 3.5 hours, the patient was discharged to home. The discharge diagnosis was atypical chest pain. It was determined that the increased diltiazem dose had slowed atrial conduction and instigated a junctional rhythm. The hospital's cardiology department recommended reducing the diltiazem dose back to 180 mg daily.

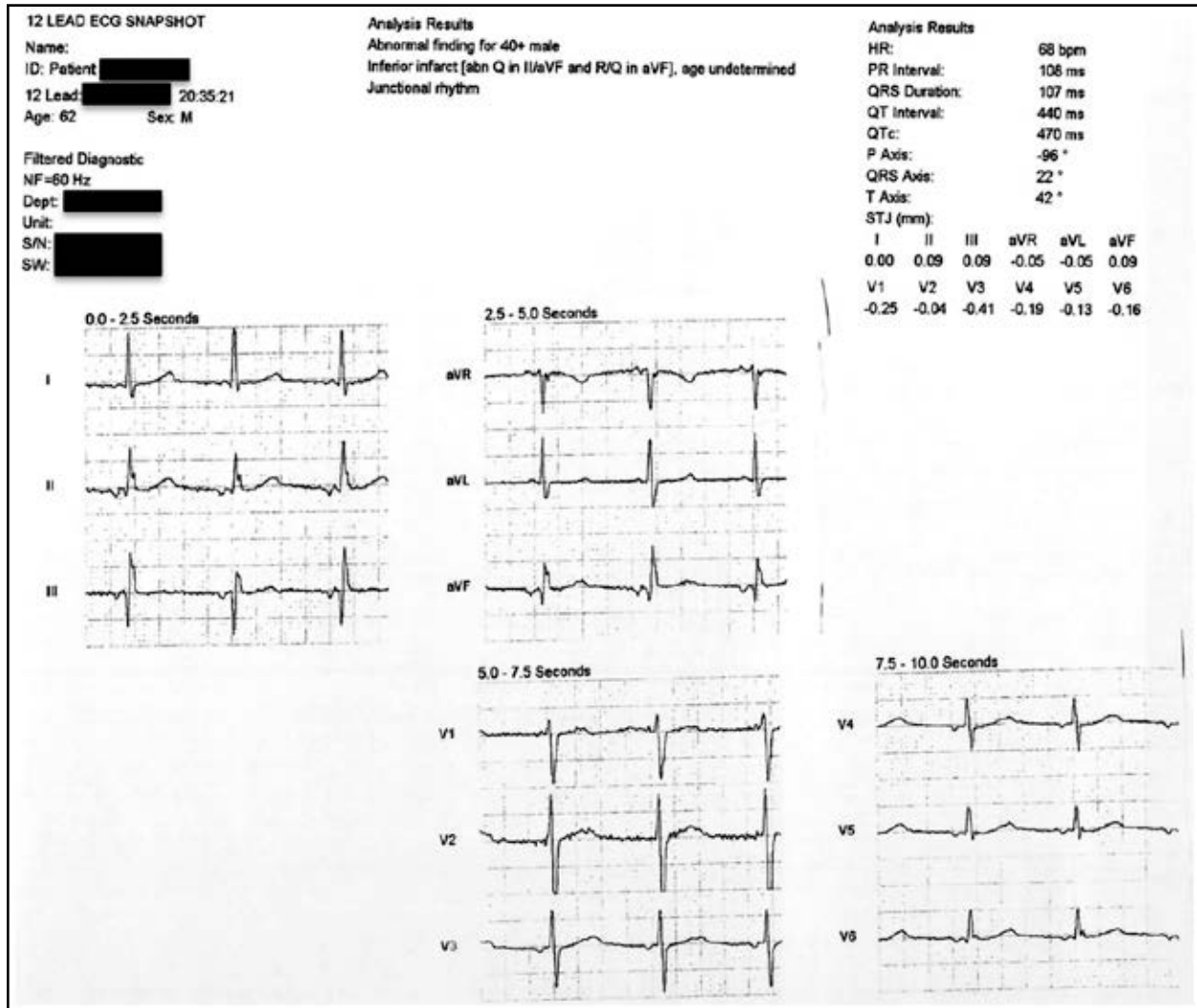


Figure 2. Twelve lead electrocardiogram #2 performed at 20:35. The second 12-lead EKG shows a regular rhythm at 68 bpm. P-waves are now negative (inverted) and the PR interval is shortened to 108 ms. The QRS duration is 107 ms. No leads show ST elevation greater than 1 mm.

DISCUSSION

The heart's electrical conduction system normally initiates an electrical stimulus at the sinoatrial (SA) node. This electrical stimulus traverses through the atria to the atrioventricular (AV) node, where it is briefly slowed before continuing to the ventricles via the

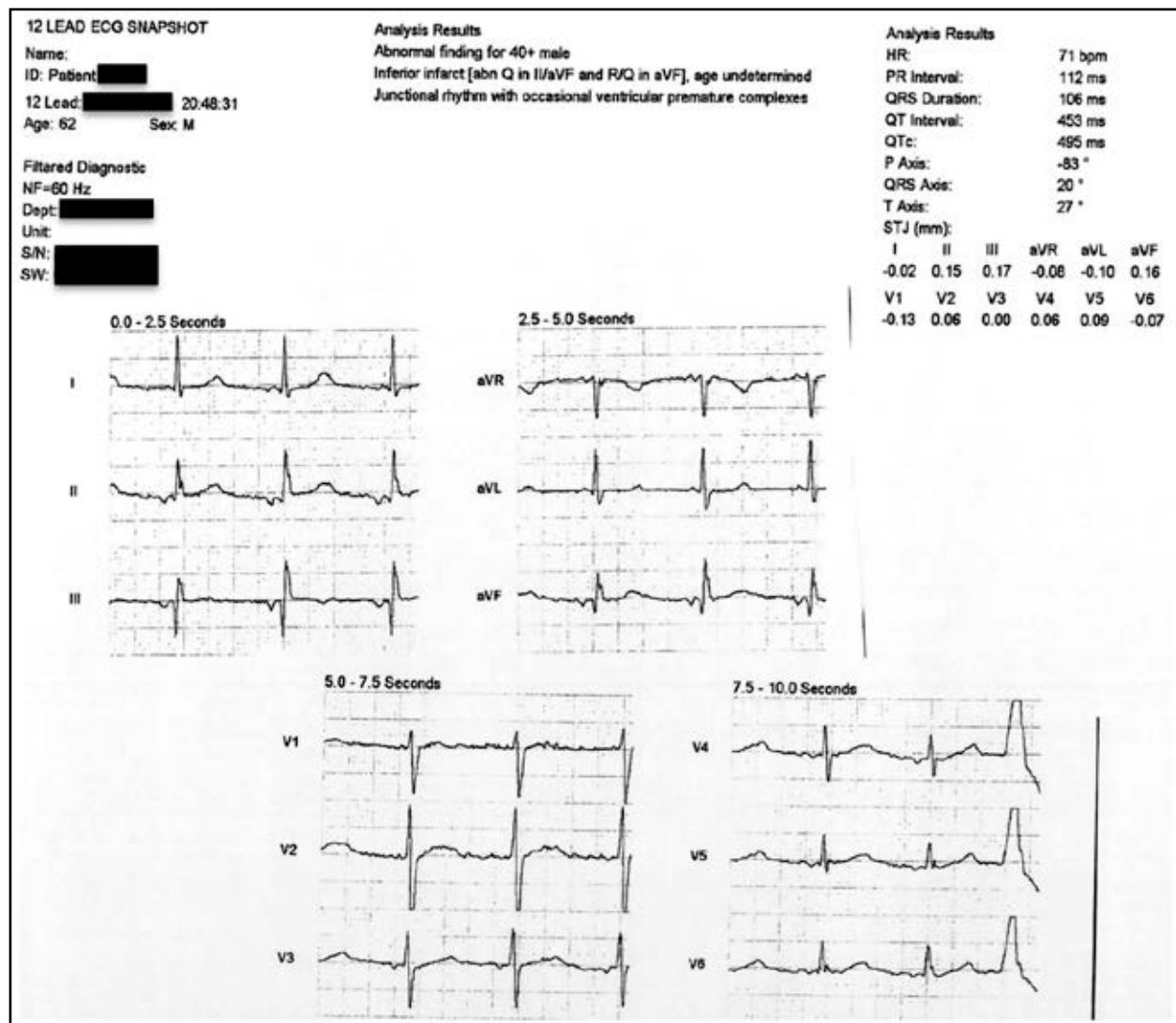


Figure 3. Twelve lead electrocardiogram #3 performed at 20:48. The third 12-lead EKG shows a regular rhythm with a PVC at a rate of 71 bpm. P-waves remain negative (inverted) and the PR interval remains short at 112 ms. The QRS duration is 106 ms. No leads show ST elevation greater than 1 mm.

bundle of HIS, where it diverges into the left and right bundle branches of the ventricles.

The AV node filters impulses from the atria, enabling regular and optimum conduction to the ventricles (Billette & Tadros, 2019). The more frequently the AV node is stimulated, the slower the node conducts an impulse (Fogoros, 2022). This feature provides natural protection from diseases like atrial fibrillation (AF), which can conduct hundreds of impulses per minute to the AV node (Fogoros, 2022).

If the SA node is damaged or blocked, the AV node, the bundle of HIS, or a site low in the atria can take over as the heart's primary pacemaker (Grauer, 2019;

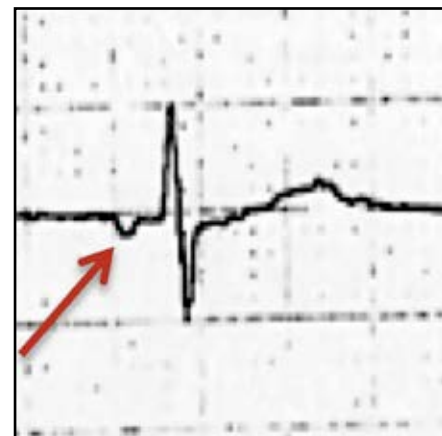


Figure 4. Negative (inverted) P-wave (red arrow)

Hafeez & Grossman, 2022). The AV node can conduct electrical impulses bidirectionally (Adams & Pelter, 2004). If the SA node fails to depolarize (activate) the atria, the AV node can take over and initiate what is called retrograde conduction (Adams & Pelter, 2004). Retrograde conduction will appear as negative (inverted) P waves in an EKG tracing (Figure 4) and a shortened PR interval (less than 120 ms). In some cases, the inverted P waves may be found after the QRS. In these patients, atrial depolarization occurs after ventricular depolarization, and there is no atrial-ventricle synchronicity (Adams & Pelter, 2004).

Junctional rhythms are not rate-dependent but are described as junctional bradycardia (<40 bpm), junctional escape rhythm (40-60 bpm), accelerated junctional (60 to 100 bpm), and junctional tachycardia (>100 bpm) (Burns & Buttner, 2021). A junctional rhythm may also be indicated if the PR interval is less than 120 ms. A reduced PR interval may also suggest accessory pathways between the atria and ventricles or a low atrial rhythm (Grauer, 2019).

Diltiazem (Cardizem) is a commonly used calcium channel blocker prescribed for rate control in patients with AF (Jafri et al., 2021). Diltiazem is used interchangeably with metoprolol, a non-selective beta blocker, depending on physician preference, availability, and patient responsiveness (Jafri et al., 2021). Diltiazem acts by slowing conduction between the SA and AV nodes, reducing the electrical stimulation in atrial cardiac tissue associated with AF (Jafri et al., 2021). Diltiazem also inhibits cardiac and smooth muscle contractions and increases vasodilation in coronary and systemic arteries (Jafri et al., 2021; Morrow & de Lemos, 2019). Side effects are associated with vasodilation and are typically seen at dosages exceeding 240mg daily (Morrow & de Lemos, 2019).

The patient was also prescribed dofetilide (Tikosyn), a class III antiarrhythmic agent prescribed for atrial fibrillation or flutter. Dofetilide can raise diltiazem levels (Eprocrates). By blocking potassium channels, Dofetilide slows repolarization and increases the refractory period of atrial tissue (Ibrahim, Kerndt, & Tivakaran, 2023).

While this patient's condition was attributed to the increase in his diltiazem prescription, digitalis toxicity is the classic cause of accelerated junctional rhythms (Burns & Buttner, 2021). Other causes of junctional rhythms include cardiac surgery or acute myocardial ischemia.

Fortunately, this patient remained stable throughout the encounter. Had the patient's condition deteriorated, unstable calcium channel overdose treatment focuses on airway, breathing, and circulation. Chakraborty and Hamilton (2023) recommend continuous cardiac monitoring and IV crystalloids during initial resuscitation, and they suggest considering endotracheal intubation for rapidly deteriorating patients with hemodynamic compromise. They note that providers should watch for fluid overload if the patient experiences cardiac slowing or weak contractions due to the drug's negative inotropic effects. Pharmacological therapies for unstable patients can include calcium, insulin, glucagon, and catecholamines (dopamine, norepinephrine, epinephrine) to improve vascular resistance and hypotension (Levine, 2018). As always, work within local protocols and consult medical control or poison control centers when needed.

CONCLUSION

The presence of inverted P waves and a shortened PR interval (< 120 ms) indicate a junctional or low-atrial rhythm. Absent congenital issues, recognition of an accelerated junctional rhythm should prompt suspicion of digitalis toxicity or iatrogenic causes. Other causes of junctional rhythms include calcium channel blocker toxicity, ischemia or infarction, and recent cardiac surgery. The case also raises awareness of iatrogenic events, such as prescription drug complications, as causes of prehospital encounters. Finally, the case reminds us that serial ECGs should be a regular part of any chest pain work-up in the field.

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