Accelerated idioventricular rhythm as anginous substrate in elderly: Report of an unprecedented case

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Introduction

Accelerated idioventricular rhythm (AIVR) results from the abnormal action of an ectopic ventricular pacemaker. AIVR is characterized as a regular rhythm with 3 or more ventricular complexes (QRS complex >120 ms) and a heart rate up to 100 beats per minute,[1] although ranges up to 110[2]–120[3] have been described, varying according to the source consulted. The pathophysiology involves hyper-automatism and triggered activity, particularly in cases of ischemia and digoxin toxicity.[3,4]

Classically, AIVR is observed in the reperfusion phase of an acute myocardial infarction or post thrombolysis, being a well-tolerated, benign, and self-limited arrhythmia.[5] Cases in patients with hyperkalemia, digitalis intoxication, and myocarditis, as well as in patients post resuscitation, have also been described.[5–7]

Although most of the time it is a benign and asymptomatic event, AIVR can also arise with various symptoms; palpitations, cough, syncope, and lightheadedness are some of the possibilities.[7] We describe the case of a patient who presented with anginous chest pain as a symptom of AIVR. This case prompts a consideration of a potential pathophysiology that parallels painful left bundle branch block syndrome (PLBBBS), but with a ventricular arrhythmic substrate, and also raises additional diagnostic hypotheses for the case.

Case report

A 68-year-old male patient, previously diagnosed with hypertension and on a daily regimen of losartan 100 mg, presented for an outpatient consultation at a tertiary cardiology hospital. His chief complaint was episodes of intermittent,

KEY TEACHING POINTS

- Although accelerated idioventricular rhythm (AIVR) is commonly seen as a benign rhythm after myocardial reperfusion, clinicians should remain alert to possible rare symptomatic presentations, such as anginous chest pain, as observed in the described case.
- Probably we are talking about an extension of the enigmatic phenomenon of painful left bundle branch block syndrome (PLBBBS). In this sense, we propose a presentation similar to PLBBBS, but with a different substrate (AIVR), which is characterized by angina concurrent with the sudden appearance of an LBBB pattern on the electrocardiogram (ECG). Key diagnostic hallmarks include pain alleviation with the resolution of the AIVR rhythm, absence of myocardial ischemia, and a prior and postblock normal ECG.
- Given AIVR's rare potential to be the primary cause behind anginal pain, especially when other usual triggers are absent, a broad differential approach becomes essential for clinicians when confronted with prevalent symptoms such as chest pain.
- The varied clinical manifestations of AIVR, as shown in this case, underline the importance of comprehensive patient evaluation, even when presented with rhythms traditionally viewed as benign.
- We propose a reflection on our limited knowledge from understanding the exact mechanisms behind the varied presentations of one of the most common symptoms in daily clinical practice, ie, chest pain. Further analyses are required, reinforcing the importance of scientific publication of more related cases.

KEYWORDS Accelerated idioventricular rhythm; Chest pain; Angina pectoris; Left bundle branch block; Painful left bundle branch block syndrome
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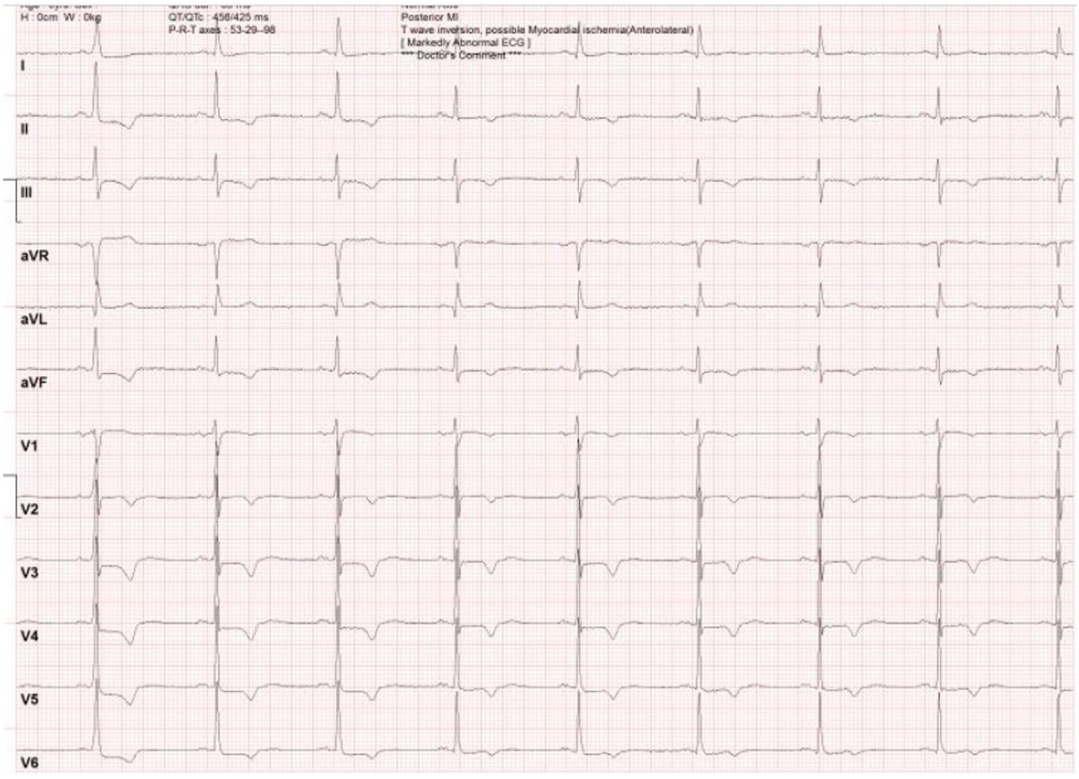

Figure 1 First electrocardiogram showing regular sinus rhythm with an inverted, diffuse, and asymmetric T wave, without other findings.

nonradiating retrosternal chest pain, described as squeezing in nature, lasting less than 5–10 minutes and occasionally occurring with physical exertion. The episodes manifested both at rest and following moderate-to-intense physical activity. The patient mentioned that the pain was self-limited, with no relieving factors, and until that point no associated symptoms were reported.

An electrocardiogram (ECG) performed prior to the consultation demonstrated a sinus rhythm with diffuse and nonspecific secondary changes in ventricular repolarization (Figure 1). The physical examination revealed no abnormalities. However, during the consultation, he experienced one of the before-mentioned chest pain episodes.

Concurrently, a new ECG (Figure 2) was captured, which unveiled a ventricular arrhythmia with a morphology consistent with a left bundle branch block (LBBB) morphology with inferior axis. This episode of AIVR was notably synchronized with his anginous symptoms, prompting hospitalization for a comprehensive investigation.

In earlier investigations of chest pain at the same hospital, a treadmill test was conducted wherein the patient experienced a sudden episode of angina. During this episode, the ECG illustrated the onset of an arrhythmia consistent with AIVR, which was again self-limited.

Notably, this ECG (Figure 3) exhibited an AIVR with a LBBB morphology, but this time with a superior axis and a discordant transition in the precordial leads with a positive QRS in V_2 and rS morphology in the lateral leads. This arrhythmic pattern was distinct from the ECG captured during the office consultation (Figure 2).

While this variation in the arrhythmic pattern suggests the possibility of different foci within the right ventricle as the origin of AIVR, the exact location and nature of these foci could be more precisely determined through a comprehensive electrophysiological study and 3-dimensional mapping. Additionally, the possibility of an electrode reversal during the examination may also be considered.

To rule out obstructive coronary artery disease during this recent hospitalization, the patient underwent a coronary angiography, which did not reveal the presence of atherosclerotic disease. Troponin levels, evaluated both prior to the procedure and throughout the duration of the hospital stay, remained consistently within normal limits. Thyroid hormones and serum electrolyte levels were normal.

A transthoracic echocardiogram revealed an ejection fraction of 65% by the Simpson method, an interventricular septum of 9 mm, a posterior wall measuring 8 mm, and cardiac valves without structural abnormalities. A 24-hour Holter monitor and a new treadmill test were executed and demonstrated no discernible abnormalities. In this context, for structural evaluation, cardiac magnetic resonance imaging with contrast was performed. This imaging technique identified a minor diverticulum in the inferobasal segment of the left ventricle and late gadolinium enhancement in the

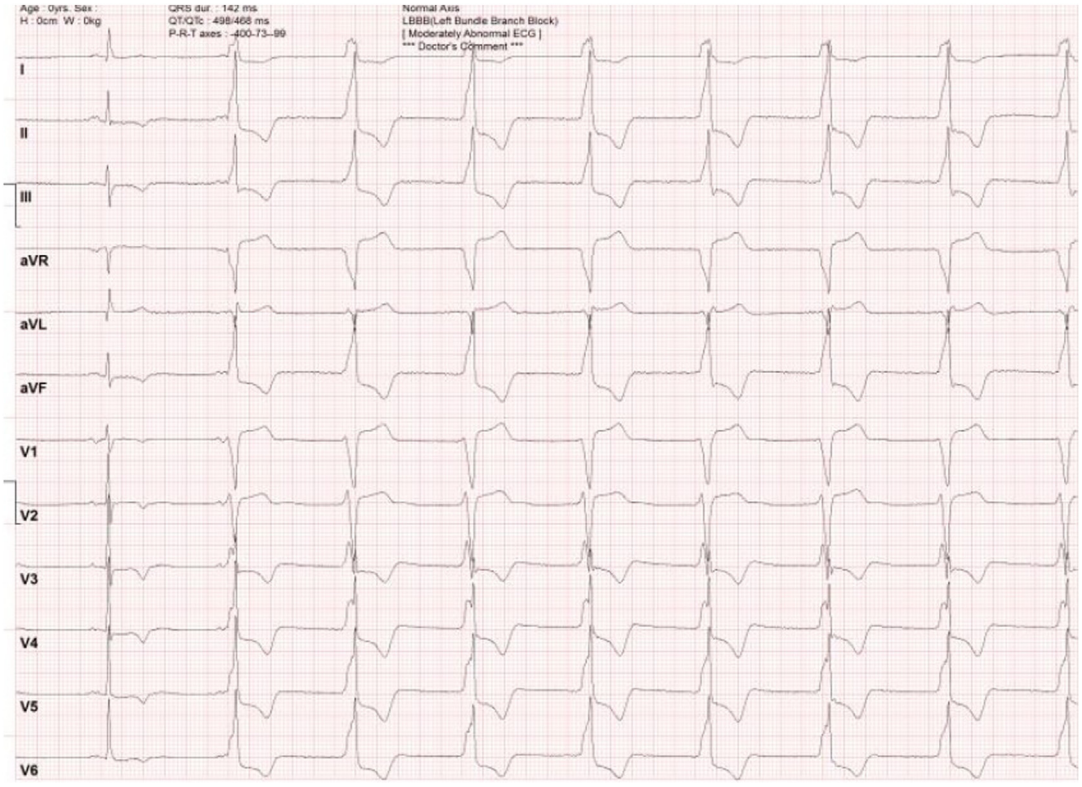

Figure 2 Second electrocardiogram, at the time of pain, showing, in the second beat, the onset of a wide QRS rhythm and, from the third complex onward, also not preceded by a P wave. The heart rate is 55 beats per minute.

inferoseptal region of the right ventricle (Supplemental Figure 1A and 1B).

The introduction of an oral beta-blocker, metoprolol 25 mg twice a day, proved effective as an antianginal strategy. Subsequent follow-ups with 3 and 6 months, including a new 24-hour Holter and exercise testing (treadmill test), revealed that the beta-blocker led to a resolution in the occurrence of the AIVR episodes. At this point, the most notable benefit was alleviating the anginous chest pain. The patient did not experience new events during hospitalization, and the pain was attributed to intermittent episodes of arrhythmia. He was discharged in good clinical condition for follow-up in a specialized outpatient clinic.

Discussion

There are scarce reports that relate episodes of AIVR as the etiology of acutely symptomatic events. This is a rhythm that, most of the time, is described as a consequence of another process (such as acute coronary syndrome, hyperkalemia, or digitalis intoxication).[6]

In this sense, Wang and colleagues[8] studied 27 patients with an average age of 32 years who had symptomatic episodes of AIVR on 24-hour Holter and were followed for electrophysiological study and evaluation of the need for catheter ablation. Most of the patients had normal ventricular function, and the hypotheses of acute coronary syndrome, hydroelectrolytic disorders, and thyrotoxicosis were ruled out after admission. In this case series, only 2 patients presented with chest discomfort (anginal events not described). Those considered for ablation were those who had syncope, persistent AIVR (>70%), or ventricular dysfunction—considering the possibility of arrhythmia-induced cardiomyopathy; metoprolol treatment was performed for the others, with good response.[9]

The pathophysiological analysis by which ventricular arrhythmia episodes can cause anginal pain are certainly little known when not associated with other events, such as acute coronary syndrome. Our case presents a significant challenge in elucidating the mechanism of the pain. One possibility we propose is a mechanism akin to that described for PLBBBS. In this syndrome, with fewer than 100 published cases, patients present with angina concurrent with the acute appearance of an LBBB and by diagnostic criteria there is improvement in pain with the resolution of the bundle branch blockage.[10]

In the proposed criteria by Shvilkin and colleagues[11] (see sidebar for complete list of criteria), the absence of myocardial ischemia and normal left ventricular function must be proven. Normal ECGs before and after the LBBB are also a diagnostic criterion. The exact mechanism of pain associated with LBBB remains elusive. However, the most plausible explanation is that the dyssynchronous ventricular contraction caused by LBBB may be the origin of the pain.[11]

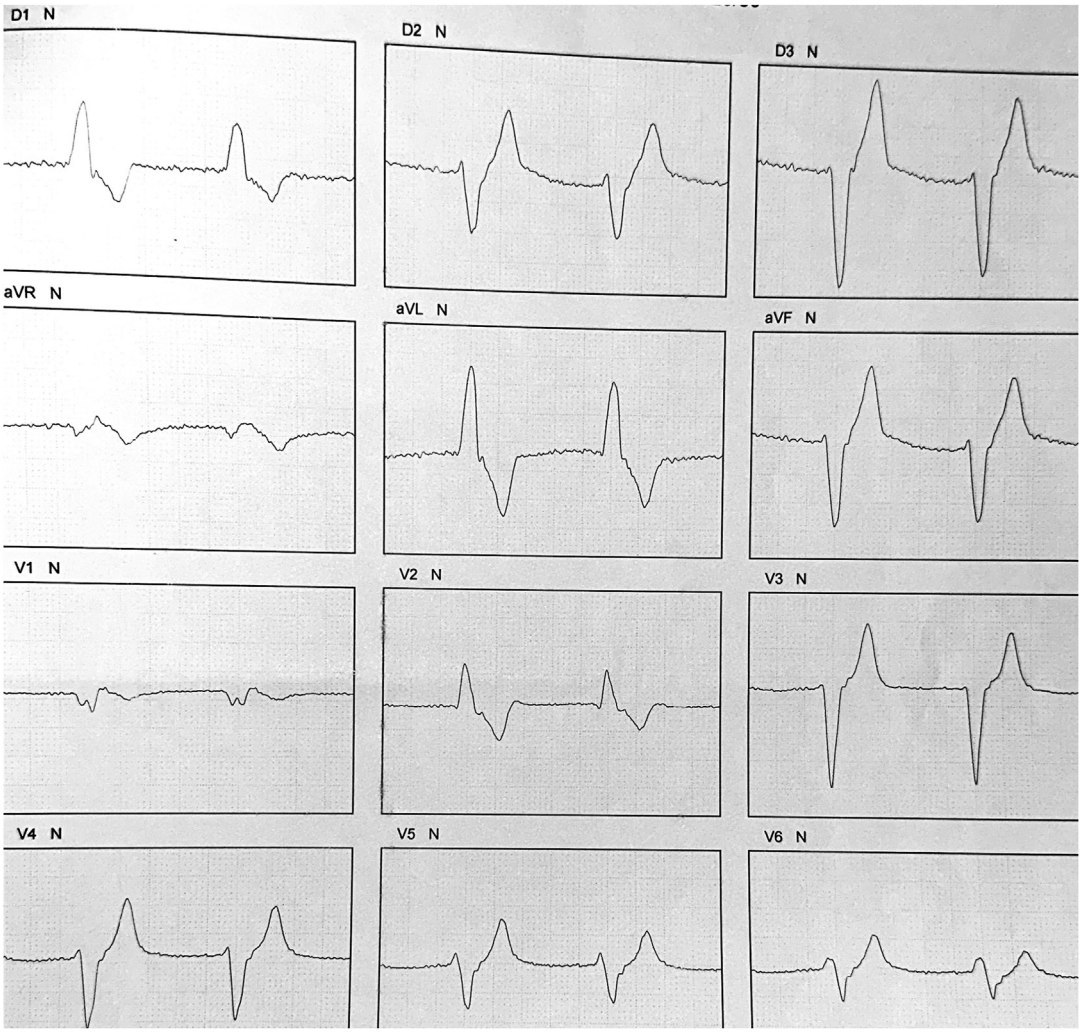

Figure 3 Twelve-lead electrocardiogram in the third minute of the treadmill test, which was interrupted owing to a complaint of chest pain, revealing ventricular arrhythmia that ceased concurrently with the end of the anginous episodes.

Painful left bundle branch block syndrome criteria proposed by Shvilkin and colleagues[11]

(1) Abrupt onset of chest pain with the development of LBBB
(2) Concomitant resolution of symptoms with the resolution of LBBB (occasionally absent)
(3) Normal 12-lead ECG before and after the LBBB
(4) Absence of myocardial ischemia during functional stress testing
(5) Normal ventricular function and absence of other conditions that may explain the symptoms
(6) S/T ratio <1.8 in precordial leads and lower axis

Our patient does not present an intermittent LBBB pattern but rather a rhythm that we believe to be idioventricular. In this case, although the patient presents 2 probable different foci for his AIVR's episodes, we propose that, during at least some of these episodes, the ventricles may be electrically activated in a pattern resembling LBBB. This is indicated by the RR′ activation pattern with a mid notch in I and the LBBB-like morphology observed in V_1. From this perspective, one hypothesis is that there might be a mechanism of pain related to ventricular dyssynchrony similar to that observed in PLBBBS.

We propose that the T-wave inversions in the baseline ECG may occur owing to the presence of "Memory T waves," given that the patient frequently changes heart rhythm from sinus to what we call AIVR. Memory T wave is a known phenomenon characterized by the persistence of T-wave inversion after a period with a wide QRS complex rhythm, such as bundle branch blocks, accessory pathways, ventricular pacemakers, and ventricular arrhythmias.[12] An alternative explanation for the diffuse T-wave inversion in our patient is the presence of a discrete diverticulum in the left ventricle. In a recent literature review, T-wave inversion was the most prevalent alteration among those with this abnormality.[13]

In reflecting upon the anginal symptoms observed in our case of AIVR, it is prudent to recognize various potential differential diagnoses without extensively focusing on a singular pathophysiological explanation. Consequently, our case opens the door to other plausible explanations for the

symptoms associated with AIVR. These include AV dyssynchrony, which could lead to chest discomfort akin to conditions such as AV node reentrant tachycardia, and other etiologies like pericarditic-type pain or coronary spasms. Understanding these various possibilities is crucial, emphasizing the need for a broader perspective and more comprehensive research in this domain.[14,15]

In our report, we observed that the patient exhibited 2 distinct arrhythmic patterns of AIVR, 1 with a superior axis and another with an inferior axis, both presenting an LBBB morphology in V_1. The distinct morphological features in these ECGs suggest the possibility of different foci within the right ventricle as the origins of the AIVR episodes. However, without the conduct of an electrophysiological study and 3-dimensional electroanatomic mapping, the exact location and nature of these foci remain speculative. Additionally, the potential for a limb lead reversal during the ECG recording cannot be definitively ruled out.

The absence of an electrophysiological study in our case represents a logistical limitation, preventing us from determining the ventricular nature and the exact location of the arrhythmia foci. However, as the patient is clinically well after starting medication therapy, recommending this procedure, even if minimally invasive, is not in our plans. Furthermore, the absence of a P wave preceding the complexes makes it unlikely that there is another mechanism to explain this arrhythmia other than an ectopic ventricular focus.

We do not credit the presence of the diverticulum in the left ventricle as a possible etiology of angina, owing to pathophysiological implausibility and the unequivocal causality correlation between the moment the arrhythmia begins and the onset of pain, as well as the termination of both. We also do not credit the diverticulum as the cause of the patient's ventricular arrhythmia, as it is in the left ventricle, and therefore, if there were reentrant mechanisms related to the diverticulum, the arrhythmia would have a right bundle branch block pattern, which is not the case for the reported patient.

Moreover, the arrhythmia does not behave consistently with a reentrant mechanism. As observed in Figure 2, the onset is not preceded by an extrasystole, as is common in reentrant arrhythmias, but appears more like an automatic or triggered activity rhythm. This also rules out the possibility of this arrhythmia being caused by perifibrotic reentry around the late enhancement found in the patient's right ventricle.

Conclusion

In our judgment, the patient undergoes paroxysmal episodes of AIVR with a ventricular activation pattern similar to that of a left bundle branch block. This phenomenon may be responsible for the patient's anginal symptoms, even though he does not have coronary artery disease. The patient in question also has a slight diverticulum in the left ventricle and late enhancement in the right ventricle. In our view, however, the diverticulum and late enhancement found in the cardiac resonance probably have no relation to the patient's anginal episodes.

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Verbal consent was obtained from the patient granting permission that data concerning his case could be submitted for publication. The patient and responsible researchers signed informed consent and responsibility forms, respectively. The present study was evaluated by the ethics committee in research of the responsible institution and was approved with CAAE 72712523.6.0000.5192. The study did not involve experiments on humans or animals.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.12.014.

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