Review Article

Critical Management of Accelerated Idioventricular Rhythm in Emergency Sitting, Review Article

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ABSTRACT

The terminology "accelerated idioventricular rhythm" refers to an ectopic ventricular rhythm with three or more consecutive premature ventricular monomorphic beats with slow onset and end. That is slower than ventricular tachycardia but faster than the typical intrinsic escape rate of the ventricles, which is 30 to 40 beats per minute. Accelerated idioventricular rhythm differentiates from ventricular tachycardia in addition to having a positive outcome, a long coupling period at the beginning, a gradual drop-in ventricular rate at the conclusion, and an increase in sinus rate. After thrombolysis upon acute myocardial infarction, its presence is a sign that reperfusion was successful. Since malignant ventricular tachycardias are not commonly linked with accelerated idioventricular rhythm and the condition is typically hemodynamically well tolerated, no special therapy is typically required beyond managing the underlying heart condition. Accelerated idioventricular rhythm (AIVR) is usually benign, transitory, and untreatable. The goal of this observational study is to learn more about the clinical signs, prognosis, and therapy of frequent AIVR.

Keyword: Ventricular, Tachycardia, Ectopic, Infarction, Ventricular perfusion.

Introduction

Sir Thomas Lewis introduced accelerated idioventricular rhythm (AIVR) for the first time in 1910 [1]. Idioventricular tachycardia, lethargic ventricular tachycardia, and nonparoxysmal ventricular tachycardia are some of the descriptive terms used to describe this ectopic ventricular rhythm. Later, Marriott and Menendez [2], introduced the simply descriptive phrase, accelerated idioventricular rhythm, which is now widely used to address the semantic issues. The clearest method to define an accelerated idioventricular rhythm is characterised by three or more ectopic beats, successive, slower-paced premature ventricular beats than ventricular tachycardia but quicker than the 30 to 40 bpm ventricular intrinsic escape rate that is common [3]. A faster than normal idioventricular rhythm (AIVR) is frequently temporary, regarded as benign, and does not need to be treated [4]. In addition, accelerated idioventricular rhythm displays several typical characteristics that further set it apart from ventricular tachycardia, such as how the arrhythmia begins and

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Finishes and how it typically takes a benign course. Ectopic rates frequently coincide with the dominant sinus rate, resulting in isorhythmic dissociation. Although acute myocardial infarction [5] and acute myocarditis [6] have both been associated with multiform accelerated idioventricular rhythm, there often is only one accelerated ventricular focus. The most prevalent arrhythmia associated with reperfusion in humans is accelerated idioventricular rhythm. However, with spontaneous restoration of antegrade flow, the two main causes of sudden death, ventricular tachycardia and ventricular fibrillation, are still present [7]. Notably, there is no widespread agreement on the maximum rate that constitutes as an accelerated idioventricular rhythm. The adoption of 120 bpm as an upper rate cut-off for this arrhythmia was advised because ectopic ventricular rhythms exhibiting the characteristic signs of accelerated idioventricular rhythm can occasionally occur with rates over 100 bpm [8,9]. It's important to note that the rate criterion alone cannot diagnose accelerated idioventricular rhythm; additional characteristics, such as how the arrhythmia starts and terminates, must be examined [9].

Causes

There are three or more idioventricular beats in an accelerated rhythm (AIVR). Successive monomorphic beats with progressive beginnings and ends [10, 11]. AIVR is seldom observed in people with structural heart disease or fully normal hearts, and is typically observed under the context of reperfusion after a sharp myocardial infarction (when thrombolytic treatment or invasive coronary procedures has ended). Clinicians working in emergency rooms have observed AIVR decreased compared to the thrombolytic period since percutaneous coronary intervention (vs thrombolytic therapy) is now being used increasingly frequently to treat patients who have acute myocardial infarction [11]. Additionally, AIVR has been linked to a number of medicines (such as aconitine, halothane, Drugs like digitalis, cocaine, with desflurane), electrolyte abnormalities (severe hyperkalemia with hypokalemia), heart diseases, with following a cardiac arrest in the postresuscitation phase [11-14]. There are two potential explanations for this rhythm that could coexist. First, nodal automaticity may be depressed, which is potentiated by increased vagal tone, causing structural damage to the sinoatrial or atrioventricular node. Second, the dominant pacemaker may be an aberrant ectopic focal within the ventricle [15]. The ventricular rate increases when the ectopic focus accelerates by thirty to forty bpm over the intrinsic rate or when the sinus rate slows down (below that of the ectopic focus). It is possible to experience isorhythmic dissociation, fusion beats, and capture beats when the sinus and ectopic focal discharge rates are comparable [14].

Diagnosis and Clinical manifestations

ECG and Holter monitoring are the two basic methods used for recurrent AIVR diagnosis. It is crucial from a clinical standpoint to screen and stratify this patient population [16, 17]. Patients with structural heart disease or other predisposing factors who have undergone acute myocardial infarction are more likely to experience transient AIVR, which is typically acceptable and has no negative effects on prognosis [18, 19]. However, based on the observations, the clinical signs and prognosis of recurrent AIVR appear to be distinct from those of temporary AIVR. Some authors did come across a DCM patient whose clinical outcome was deteriorated by persistent AIVR, despite the fact that it most commonly happens in people with a structurally normal heart. Arrhythmia-induced cardiomyopathy (AIC) may be brought on by persistent AIVR [20, 21]. Four patients in one study had their compromised LVEF totally restored to normal following ablation. These are similar to early ventricular complexes (PVCs), this may result in an AIC with a significant ectopic load [22, 23]. After a successful ablation, the DCM sufferer who is heavily burdened by AIVR displayed modest clinical improvement, but it is hypothesised that patients with structural heart disease may experience worse clinical outcomes as a result of repeated AIVR due to an increased clinical risk associated with this condition. Previous research discovered separate associations between PVC-induced cardiomyopathy and the length lack of indications with symptoms as well as PVCs' epicardial origins [24, 25]. Furthermore, preliminary research did not discover an association between reduced LVEF and additional factors including the occurrence and progression of frequent AIVR, together the exception of AIVR burden and QRS width. All of the patients in our group had symptoms, unlike those with PVCs, which could be attributed to the following factors:(1) Patients with symptoms were more likely to see a cardiologist than patients with no symptoms; (2) Because AIVR lasts for at least three beats, it is more uncomfortable than PVCs. So far, it has not been determined whether epicardial origin might impact LV function in individuals with AIVR. AIVR has a reasonably constant RR interval in contrast to PVCs, which frequently have brief coupling intervals, also, the heartbeat is virtually normal when there is an AIVR. This could be the possible explanation for why AIVR predicts poor LV function more accurately than PVCs. Future research should, however, incorporate more cases to improve prediction precision. Due to its ectopic and competing character, frequent AIVR frequently causes palpitations, similar to frequent PVCs. Nevertheless,

particularly an overwhelming reaction to the understanding-based tenor of with AIVR patients with structural heart disease may bring about hemodynamic uncertainty with perhaps sudden cardiac arrest [25].

Management

Management is done basically using catheter ablation and electrophysiological study (EPS). Under conscious sedation, EPS and catheter ablation procedures are carried out. For mapping and ablation, in the intended chamber, 4 mm tip quadripolar irrigation catheter is introduced. A digital electrophysiology device is used for capturing intracardiac electrograms utilising a filter of 30–300 Hz [17]. For complete electroanatomic mapping, In the course of the AIVR, bipolar electrograms (100–150 points) are recorded, using a system such as CARTO3 or CARTO XP. When a Right bundle branch block (RBB) origin is present, by employing data from endocardial activity measured by a decapolar catheter along the HIS-RBB axis and/or 3D mapping is suspected, the origin of the arrhythmia is confirmed [16]. An irrigated catheter is used to provide, as soon as possible, radiofrequency energy triggering factor; the infusion rate, maximum temperature, and maximum power are all set at 35 W, 17 mL/min, and 45 °C, respectively. The termination of AIVR during ablation, as well as post-operation non-inducibility and non-provocation with isoproterenol, are all considered the procedure endpoints [18]. Traditionally, an arrhythmia with a low risk of injury is AIVR [11]. The majority of patients of this dysrhythmia won't require immediate therapy due to the fact that AIVR frequently self-limits and ceases when the sinus rate is higher than the ventricular foci. Antiarrhythmic medication should not be given to individuals with AIVR since it may quickly worsen their hemodynamic condition [15]. Keep in mind to address the root cause of AIVR: For instance, thrombolytics or percutaneous coronary intervention can be used to reestablish myocardial perfusion, or incorrect electrolytes can be corrected. The restoration of atrioventricular synchronisation to restore atrial kick may be beneficial for individuals with diseases causing decreased cardiac output (for example, severe biventricular failure). Atropine can be administered to boost sinus rate and atrioventricular conduction when AIVR has resulted in decreased cardiac output [17].

Follow up

After successful ablation, patients are off anti-arrhythmic drugs (AADs), with the exception of patients known to suffer from dilated cardiomyopathy (DCM). AADs are started in other ways. The following procedures are carried out: TTE, 24-hour Holter monitoring, and ECG. Third, sixth, and twelve-month marks as well as annually following discharge. If patients had any symptoms, an isolated ECG is carried out. Patients with DCM, those who undergo failed ablation, and those who do not have ablation are all given metoprolol [18].

Discussion

At the top of the right atrium is where the sinoatrial node is located. The action potential that drives the rest of the myocardium to depolarize is first produced by sinoatrial node, which is the area of the heart that depolarizes first. The slower intrinsic rates of the heart's inherent lower-order pacemakers are inhibited by sinoatrial depolarization and subsequent electrical impulse propagation. An accelerated idioventricular rhythm develops when a typically suppressed focus depolarizes faster than "greater order" emphasises (the sinoatrial node and the atrioventricular node) [22]. When sinus bradycardia is present, this is most typical. After spontaneous antegrade flow restoration, the heartbeats of ventricular tachycardia and fibrillation remain the primary factors in sudden death. Prior to the current method of treating acute coronary syndrome with percutaneous coronary intervention, the use of pharmacologic thrombolysis was more common and idioventricular rhythms were used as a sign of successful reperfusion. Despite the possibility that atrioventricular dyssynchrony will cause hemodynamic instability, with regard to STEMI in particular, it is a benign arrhythmia that doesn't need to be treated (Where it is typically viewed as a sign of reperfusion). Atropine or overdrive pacing are two treatments for this [23]. A significant QRS complex and a regular rhythm, similar to ventricular tachycardia, are indicators of accelerated idioventricular rhythm. Because the rate is less than 120 bpm, and frequently less than 100 bpm, it can be separated from VT. Depending on whether ventricular escape or AV block is the underlying reason, AV dissociation may or may not take place. Any type of structural heart disorder can cause an accelerated idioventricular rhythm, additionally in infants and adults without structural cardiac disease. Cardiovascular disease patients are more prone to experience an accelerated idioventricular rhythm [24]. Effective reperfusion is indicated by its existence during thrombolysis in an acute myocardial infarction. Aside from treating the underlying heart condition, rapid idioventricular rhythm is typically well tolerated hemodynamically and is not associated with malignant ventricular tachycardias. Patients with acute myocardial infarction frequently have arrhythmias as a side effect (MI) [25]. Although increased idioventricular rhythm is only marginally beneficial as a marker of reperfusion, when paired with other non-invasive measures, its presence is linked to a high likelihood of successful reperfusion (ST-segment resolution). Myocardial infarction's potentially fatal

side effect is early ventricular arrhythmias. However, they don't seem to be a bad prognostic indicator if they are identified and treated in a timely manner. A larger myocardial infarction is more often indicated by later ventricular fibrillation or VT [26]. AF does not immediately endanger the patients' lives, but it frequently affects patients with larger myocardial infarction and is a standalone indicator of these patients' bad long-term prognosis. Early and efficient reperfusion therapy is the best anti-arrhythmic therapeutic method for myocardial infarction patients [19]. Recurrent symptomatic accelerated idioventricular rhythm, in contrast to transient asymptomatic idioventricular rhythm, has distinct clinical characteristics and is associated with a poor prognosis. Patients with rapid idioventricular rhythms who have an arrhythmia prevalence of greater than 70%, a reduced LVEF, or both may consider catheter ablation. Any signs of presyncope or syncope brought on by an excessive response to sympathetic tone are advised to undergo ablation [26].

Conclusion

Regardless of the cause, there is a different clinical presentation and prognosis for frequent AIVR with a significant burden. People with a high load, reduced LVEF, or presyncope or syncope symptoms brought on by an excessive sympathetic tone are advised to undergo ablation. Metoprolol can be used to lessen symptoms to some extent. Such AIVR patients should be given long-term follow-up.

Conflict of Interest

None

Funding

None

References

1. Zhao YT, Zhou H, Cui Y. Accelerated idioventricular rhythm degenerating into bidirectional ventricular tachycardia following acute myocardial infarction. Am J Emerg Med. 2018;36(4):735.e1-735.e3. doi:10.1016/j.ajem.2018.01.030

2. Marriott HJ, Menendez MM. A-V dissociation revisited. Prog Cardiovasc Dis. 1966;8(6):522-538. doi:10.1016/s0033-0620(66)80014-2

3. Castelli G, Ciaccheri M, Cecchi F, Troiani V, Nannini M, Marconi P, et al. Nonsustained ventricular tachycardia as a predictor for sudden death in patients with idiopathic dilated cardiomyopathy. The role of amiodarone treatment. G Ital Cardiol. 1999;29(5):514-523.

4. Riera AR, Barros RB, de Sousa FD, Baranchuk A. Accelerated idioventricular rhythm: history and chronology of the main discoveries. Indian Pacing Electrophysiol J. 2010;10(1):40-48.

5. Baher AA, Uy M, Xie F, Garfinkel A, Qu Z, Weiss JN. Bidirectional ventricular tachycardia: ping pong in the His-Purkinje system. Heart Rhythm. 2011;8(4):599-605. doi:10.1016/j.hrthm.2010.11.038

6. Koene RJ, Adkisson WO, Benditt DG. Syncope and the risk of sudden cardiac death: Evaluation, management, and prevention. J Arrhythm. 2017;33(6):533-544. doi:10.1016/j.joa.2017.07.005

7. Moens AL, Claeys MJ, Timmermans JP, Vrints CJ. Myocardial ischemia/reperfusion-injury, a clinical view on a complex pathophysiological process. Int J Cardiol. 2005;100(2):179-190. doi:10.1016/j.ijcard.2004.04.013

8. Ferrari R. Ischaemic heart disease: clinical improvement with metabolic approach. Rev Port Cardiol. 2000;19 Suppl 5:V7-V20.

9. Akdeniz C, Gul EE, Celik N, Karacan M, Tuzcu V. Catheter ablation of idiopathic right ventricular arrhythmias in children with limited fluoroscopy. J Interv Card Electrophysiol. 2016;46(3):355-360. doi:10.1007/s10840-016-0133-6

10. Gildea TH, Levis JT. ECG diagnosis: Accelerated idioventricular rhythm. Perm J. 2018;22:17-173. doi:10.7812/TPP/17-173

11. Glancy DL, Mills TA. ECG of the Month: Wide-QRS Tachycardias. J La State Med Soc. 2016;168(6):215-217.

12. Bonnemeier, Ortak, J., Wiegand, U. K., Eberhardt, et al. Accelerated idioventricular rhythm in the post-thrombolytic era: incidence, prognostic implications, and modulating mechanisms after direct percutaneous coronary intervention. Ann Noninvasive Electrocardiol. 2005;10(2):179-187. doi:10.1111/j.1542-474X.2005.05624.x.

13. Moroz VM, Lipnitskii TN. Study of pathogenesis of ventricular arrhythmia in experimental rats by separation of sinus and ventricular substitutional rhythms. Bull Exp Biol Med. 2006;141(4):400-403. doi:10.1007/s10517-006-0182-4

14. Tsai MS, Huang CH, Chen HR, Hsieh CC, Chang WT, Hsu CY, et al. Postresuscitation accelerated idioventricular rhythm: a potential prognostic factor for out-of-hospital cardiac arrest survivors. Intensive Care Med. 2007;33(9):1628-1632. doi:10.1007/s00134-007-0633-z

15. Grimm W, Hoffmann J, Menz V, Schmidt C, Müller HH, Maisch B. Significance of accelerated idioventricular rhythm in idiopathic dilated cardiomyopathy. Am J Cardiol. 2000;85(7):899-A10. doi:10.1016/s0002-9149(99)00892-9

16. Chen M, Gu K, Yang B, Chen H, Ju W , Zhang F, et al. Idiopathic accelerated idioventricular rhythm or ventricular tachycardia originating from the right bundle branch: unusual type of ventricular arrhythmia. Circ Arrhythm Electrophysiol. 2014;7(6):1159-1167. doi:10.1161/CIRCEP.114.002112

17. Doppalapudi H, Yamada T, McElderry HT, Plumb VJ, Epstein AE, Kay GN. Ventricular tachycardia

originating from the posterior papillary muscle in the left ventricle: a distinct clinical syndrome. Circ Arrhythm Electrophysiol. 2008;1(1):23-29. doi:10.1161/CIRCEP.107.742940
18. Jaromin J, Markiewicz-Łoskot G, Szydłowski L, Kulawik A. Diagnostic value of the TpTe interval in children with ventricular arrhythmias. Int J Environ Res Public Health. 2021;18(22):12194. doi:10.3390/ijerph182212194
19. Gopinathannair R, Etheridge SP, Marchlinski FE, Spinale FG, Lakkireddy D, Olshansky B. Arrhythmia-induced cardiomyopathies: Mechanisms, recognition, and management. J Am Coll Cardiol. 2015;66(15):1714-1728. doi:10.1016/j.jacc.2015.08.038
20. Simantirakis EN, Koutalas EP, Vardas PE. Arrhythmia-induced cardiomyopathies: the riddle of the chicken and the egg still unanswered? Europace. 2012;14(4):466-473. doi:10.1093/europace/eur348
21. Penela D, Fernández-Armenta J, Aguinaga L, Tercedor L, Ordoñez A, Bisbal F, et al. Clinical recognition of pure premature ventricular complex-induced cardiomyopathy at presentation. Heart Rhythm. 2017;14(12):1864-1870. doi:10.1016/j.hrthm.2017.07.025
22. Larsen TR, Huizar JF, Tan AY. How pure is the honey? Distinguishing premature ventricular complex-induced from premature ventricular complex-worsened cardiomyopathy. Heart Rhythm. 2017;14(12):1871-1872. doi:10.1016/j.hrthm.2017.08.026
23. Yokokawa M, Kim HM, Good E, Good E, Chugh A, Jr FP, et al. Relation of symptoms and symptom duration to premature ventricular complex-induced cardiomyopathy. Heart Rhythm. 2012;9(1):92-95. doi:10.1016/j.hrthm.2011.08.015
24. Latchamsetty R, Yokokawa M, Morady F, Kim HM, Mathew S, Tilz R, et al. Multicenter outcomes for catheter ablation of idiopathic premature ventricular complexes. JACC Clin Electrophysiol. 2015;1(3):116-123. doi:10.1016/j.jacep.2015.04.005
25. Ban JE, Park HC, Park JS, Nagamoto Y, Choi JI, Lim HE, et al. Electrocardiographic and electrophysiological characteristics of premature ventricular complexes associated with left ventricular dysfunction in patients without structural heart disease. Europace. 2013;15(5):735-741. doi:10.1093/europace/eus371
26. Tan AY, Hu YL, Potfay J, Kaszala K, Howren M, Sima AP, et al. Impact of ventricular ectopic burden in a premature ventricular contraction-induced cardiomyopathy animal model [published correction appears in Heart Rhythm. 2016;13(5):1171]. Heart Rhythm. 2016;13(3):755-761. doi:10.1016/j.hrthm.2015.11.016