Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

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Wide complex tachycardia in a COVID-19 patient: What is the mechanism?

Vivek Reddy *, Vickram Reddy, Satwant Mangat, Mohamed Shokr, Shanker Kundumadam, Hansini Laharwani

Introduction

Cardiac manifestations of COVID-19 have included but are not limited to atrial arrhythmias, ventricular arrhythmias, atrial or ventricular extra systole, myocarditis, bradyarrhythmias, troponin elevations and other conduction system disturbances [1]. In this case we present an interesting case of a COVID-19 positive patient with a wide complex rhythm.

Case description

A 72-year-old male with a medical history significant for hypertension and debility and no known cardiac history presented to the hospital with hypotension, fevers and tachycardia. A recent prior EKG is shown in Fig. 1. The patient's presentation was presumed to be secondary to COVID-19, which was confirmed during the hospital course and he was appropriately managed for concomitant septic shock. Pertinent cardiac laboratory findings included an elevation of Troponin T to 370, which later increased to 780 and acute renal failure without electrolyte derangements. No echocardiogram was performed. Cardiology was called due to a wide complex tachycardia as noted in Fig. 2, which was present on day 1 of admission. The patient's rhythm was suspected to be atrial tachycardia or atypical atrial flutter with 2:1 conduction with a left bundle branch block pattern. After administering amiodarone, the subsequent EKG revealed normal sinus rhythm with an intermittent left bundle branch block pattern as shown in Fig. 3. The patient was continued on amiodarone maintenance therapy and is currently being managed for septic shock and hypoxia secondary to COVID-19.

Discussion

In this case we describe an interesting atrial arrhythmia likely atrial tachycardia or atypical atrial flutter with 2:1 conduction and a concomitant wide QRS morphology in a COVID-19 positive patient. Owing to the history of COVID-19, no echocardiogram was performed so the overall left ventricular function is unknown. The etiology of the wide QRS is likely a transiently refractory left bundle secondary to rate dependency with recovery at slower heart rates. Interestingly, the wide QRS axis is superior with an extremely delayed R wave transition zone across the precordium in Fig. 3. In Fig. 2, the R wave transition zone is the same however there appears to be an inferior axis. This however is due to right arm and left leg limb lead reversal in Fig. 2. After accounting for this error, the morphology of the wide QRS complex in both EKG's is the same. These findings suggest that the RV apex is the earliest site of depolarization with the left bundle being activated transeptally from the apex to the base as seen in a right ventricle pacing lead located in the apex [2]. Another less likely explanation would include two independent tachycardias. This would imply that there is an independent atrial arrhythmia and an independent ventricular tachycardia origination from the RV apex. In order for a dual tachycardia to be a possible explanation, the EKG in Fig. 3 would require a complex interplay of the conduction system that is worth noting. In Fig. 3 we note sinus P waves occurring at a regular rate followed by intermittent wide QRS complexes with a LBBB pattern and a superior axis. If a dual tachycardia was present in Fig. 2, given that the same wide complex morphology is present in Fig. 3, this would imply that there was transient antegrade block of the AV node, followed by a PVC exiting from the RV apex with subsequent retrograde block in the AV node thus not affecting the timing of the subsequent P wave resulting in an unchanged P wave cycle length. However, given that the wide QRS morphology in Fig. 3 is the same as Fig. 2, transient antegrade block of the left bundle followed by transeptal activation of the left bundle from the apex to the base is the most likely explanation. The role of COVID-19 on the conduction system still remains to be completely unraveled and our case illustrates a complex pathologic effect that the virus has on the conduction system in a patient with no previously known conduction system disease.

Conclusion

We describe a COVID-19 patient with no known cardiac history who presented with an atrial arrhythmia and concomitant wide QRS, which is likely secondary to antegrade block of the left bundle,

* Corresponding author.
E-mail address: vreddy@med.wayne.edu (V. Reddy).

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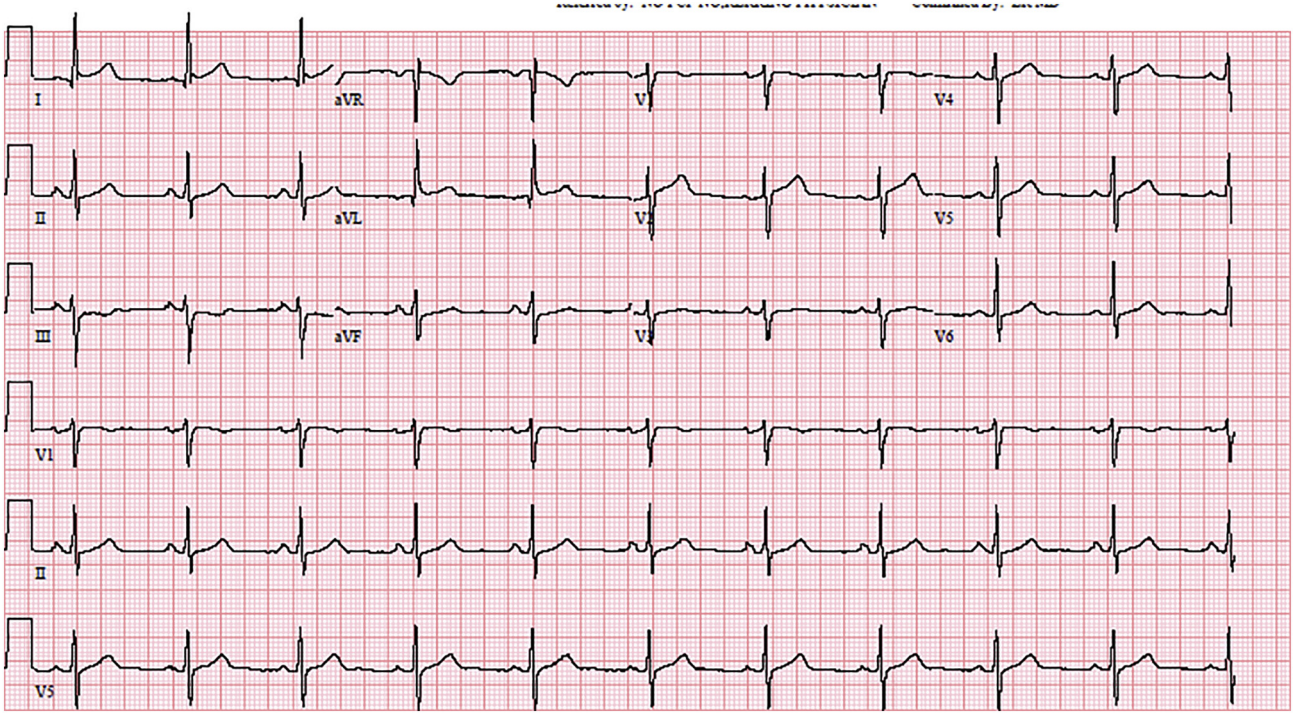

Fig. 1. Normal sinus rhythm with narrow QRS complex.

followed transeptal activation of the left bundle from the apex to the base. The full effect of COVID-19 on the conduction system is unclear but our case illustrates the complex effect it may have on the conduction system.

Author statement

All authors state that they were involved in this work. No author has any disclosures.

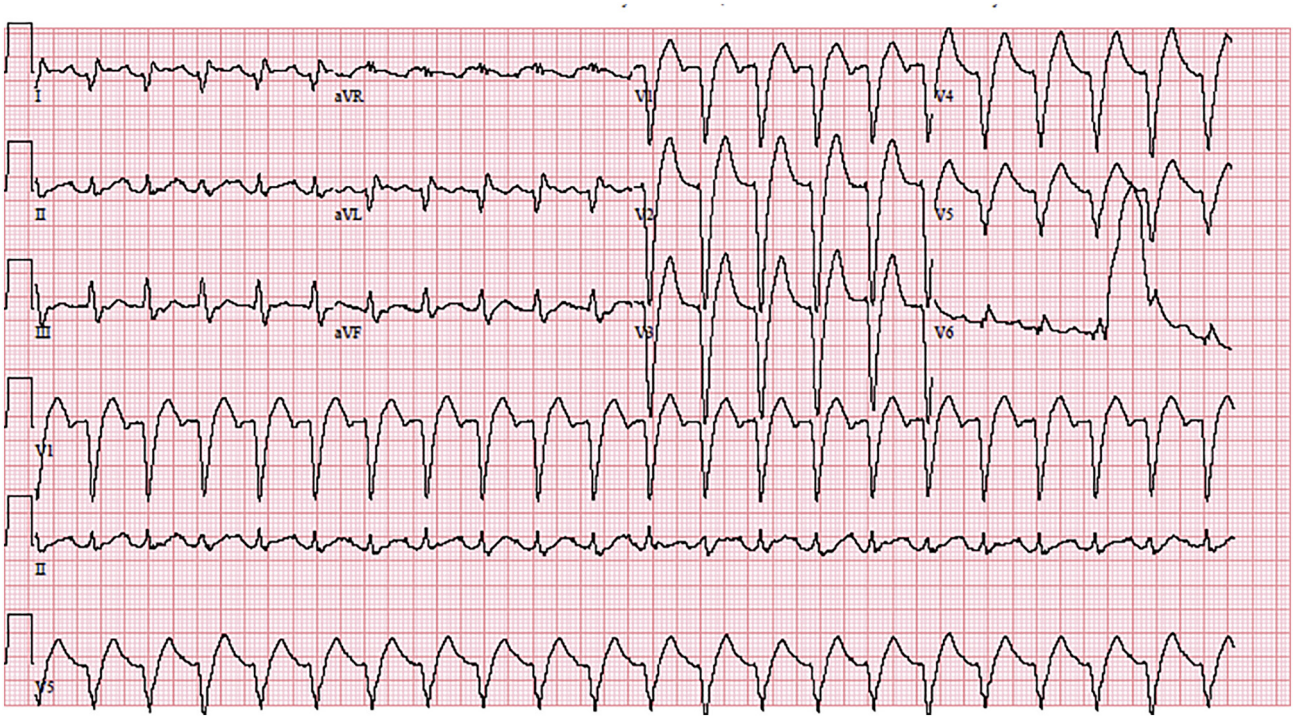

Fig. 2. Atrial tachycardia/atrial flutter with 2:1 conduction and left bundle branch block with RA-LL limb lead reversal.

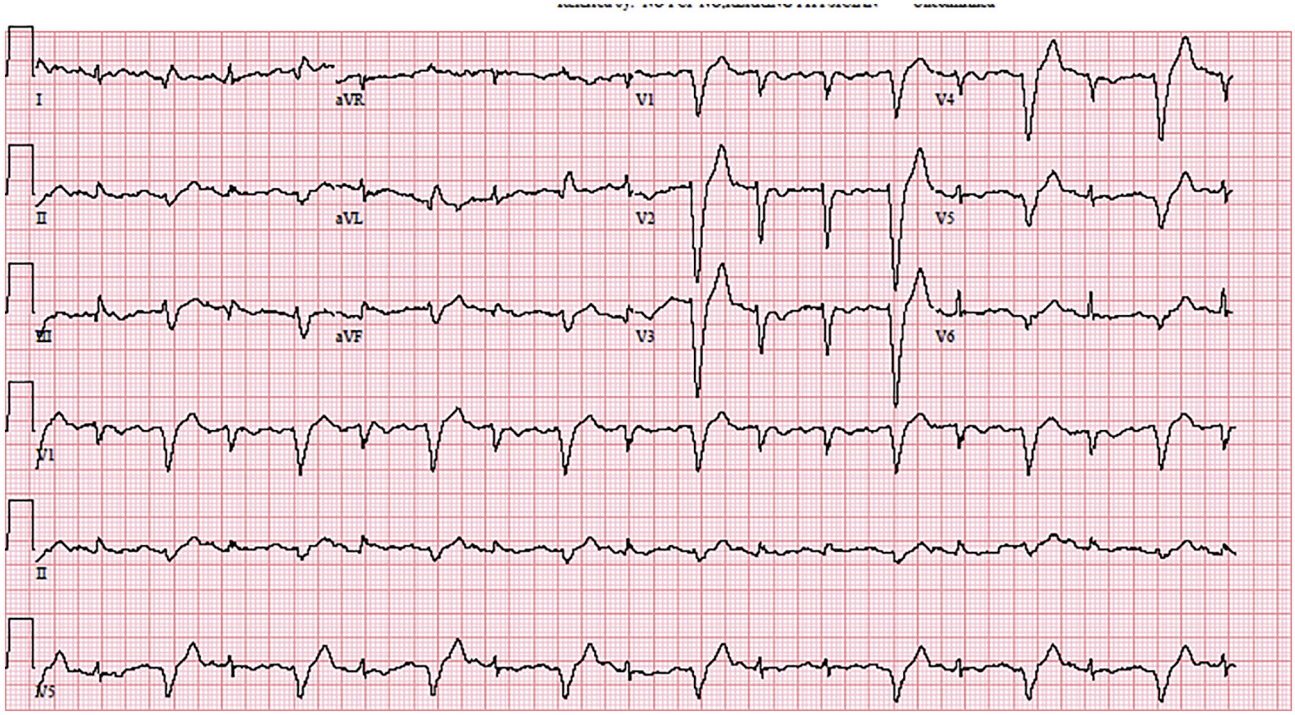

Fig. 3. Normal sinus rhythm with intermittent left bundle branch block and inferior axis.

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