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Case study

Pseudo-ventricular tachycardia—Diagnostic enigma in the emergency department: A case report

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ABSTRACT

Ventricular tachycardia (VT) is a type of broad complex tachycardia originating from a focus in the ventricle. It is one of the four important rhythms which can lead to cardiac arrest. Accurate and timely diagnosis of true VT is the cornerstone for proper management in the emergency department (ED).

We present an interesting case of an electrocardiographic artifact mimicking VT, which led to a diagnostic dilemma in the ED.

Keywords: arrhythmias, ventricular tachycardia, pseudo-ventricular tachycardia, EKG

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INTRODUCTION

Ventricular tachycardia (VT) accounts for more than 16% of cases of broad complex tachycardias presented to the emergency department (ED).[1] Diagnosis of VT has always been challenging, especially when it comes to differentiating it from supraventricular tachycardia with aberrancy or electrocardiograph (EKG) artifact. Multiple algorithms have been devised to cope with these challenges.[2]

We are presenting a puzzling case in which the patient avoided an unnecessary intervention due to his concerning EKG findings under the pretext of syncope.

CASE PRESENTATION

A 62-year-old male with a known history of essential hypertension, type 2 diabetes mellitus, and chronic kidney disease secondary to diabetic nephropathy was referred to our ED from a private hospital due to a recorded polymorphic VT in his Holter monitoring trace, which was initially done as a workup for syncope.

A couple of days earlier, he experienced a witnessed syncopal episode during his wife's blood extraction that lasted for a few seconds, followed by complete recovery. A point-of-care venous blood gas analysis did not show any acid–base abnormality, electrolyte imbalance, anemia, or hypoglycemia. 12-lead EKG done at that time showed sinus bradycardia with a heart rate of 58 beats per minute (bpm) and T-wave inversion in leads I, aVL, and V2-6 (Figure 1). Both PR and corrected QT (QTc) intervals were within normal limits. There were no other significant changes in the EKG. For comparison, no previous EKG was available. He was discharged with Holter monitoring for 24 hours. The next day, his 24-hour Holter monitoring trace showed a broad complex tachycardia with a heart rate of almost 300 bpm (polymorphic VT) that lasted for about 15 minutes, followed by self-termination (Figure 2). In addition, there were a few premature atrial complexes (PACs) with a heart rate ranging from 58 bpm to 73 bpm. Despite being asymptomatic, he was referred to the tertiary care ED for further evaluation.

In the ED, when the patient was inquired about the concerned period, he reported watching a football match on television at home and declined any symptoms like dizziness, palpitation, shortness of breath, chest pain, or diaphoresis. At presentation, he was vitally stable with a heart rate of 68 bpm, blood pressure of 168/82 mmHg, and respiratory rate of 19 breaths per minute. His oxygen saturation was 99% at room air with an oral temperature of 36.8°C. General physical and systemic examinations were unremarkable. Laboratory investigations were done, and they revealed normal electrolytes, including serum magnesium, with raised troponin-T level (50 ng/L). Renal function tests were at his baseline (Creatinine of 309 µmol/L) (Table 1). He was monitored for almost

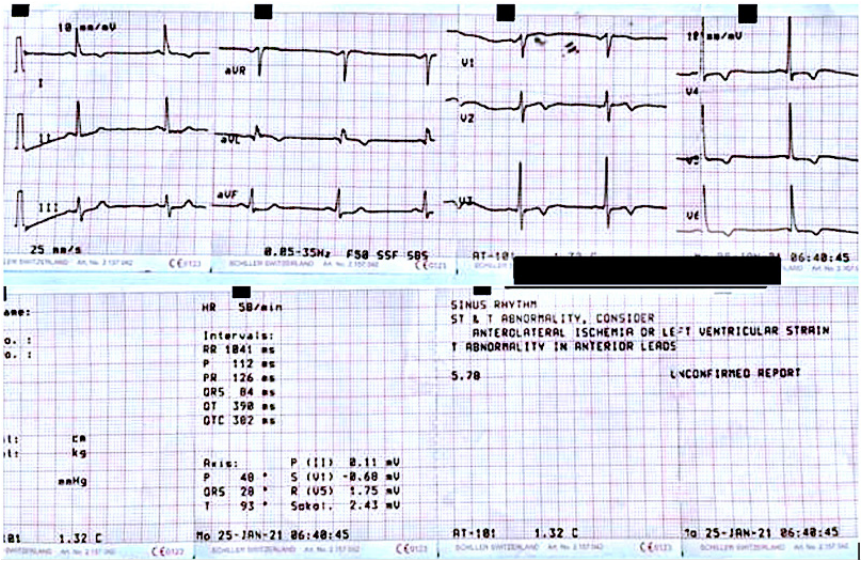

Figure 1. 12-lead EKG after first syncopal episode showing sinus bradycardia (heart rate of 58 bpm) with T-wave inversion in leads I, aVL, V2, V3, V4, V5, and V6.

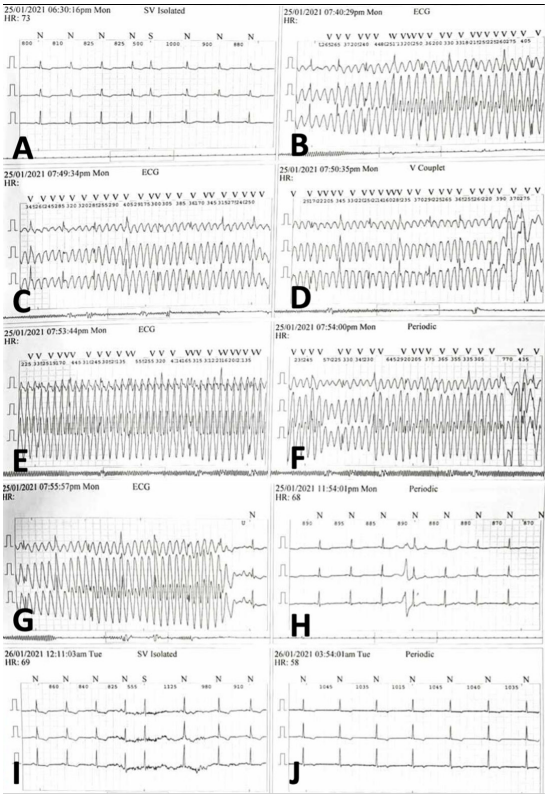

Figure 2. 10-second captures from a 24-hour Holter monitoring trace showing a broad complex tachycardia with a heart rate of almost 300 bpm (B–G), followed by self-termination to sinus rhythm with a heart rate ranging from 58 to 73 bpm (H–J).

Table 1. Laboratory investigation results.

Blood investigations	Day 0 (presentation at ED)	Day 3	Day 4	Normal range
WBC (x103 /µL)	5.2	6.9	8.12	4.0–10.0
Hb (gm/dL)	11.9	11.8	13.4	13.0–17.0
Platelets (x103/µL)	208	278	297	150–400
Urea (mmol/L)	20.1	15.7	–	2.5–7.8
Creatinine (µmol/L)	309	288	–	62–106
Magnesium (mmol/L)	0.91	–	0.94	0.70–1.00
Phosphorus (mmol/L)	1.41	–	–	0.80–1.50
Potassium (mmol/L)	4.2	4.4	4.7	3.5–5.3
Sodium (mmol/L)	138	136	137	133–146
Troponin-T HS (ng/L)	50	52	51.2	3–15

5 hours, and a 12-lead EKG showed normal sinus rhythm and normal QTc interval with T-wave inversion in Lead I, aVL, and V4-6 (Figure 3).

The provisional diagnosis of self-limited, sustained polymorphic VT was made based on the abnormal Holter monitoring trace and elevated serum troponin-T level with a history of syncope. On the other hand, it was physiologically implausible for someone to be asymptomatic for about 15 minutes with a ventricular rate of 300 beats/min; hence there existed a diagnostic quandary at the time. Because the patient was asymptomatic when he arrived at the ED, a cardiology consultation was planned in view of this diagnostic conundrum before proceeding with any intervention.

After the cardiologist assessed the serial 12-lead EKG and EKG trace from Holter monitoring carefully, it was established that the rhythm was a pseudo-VT as the “spike and notch signs” were

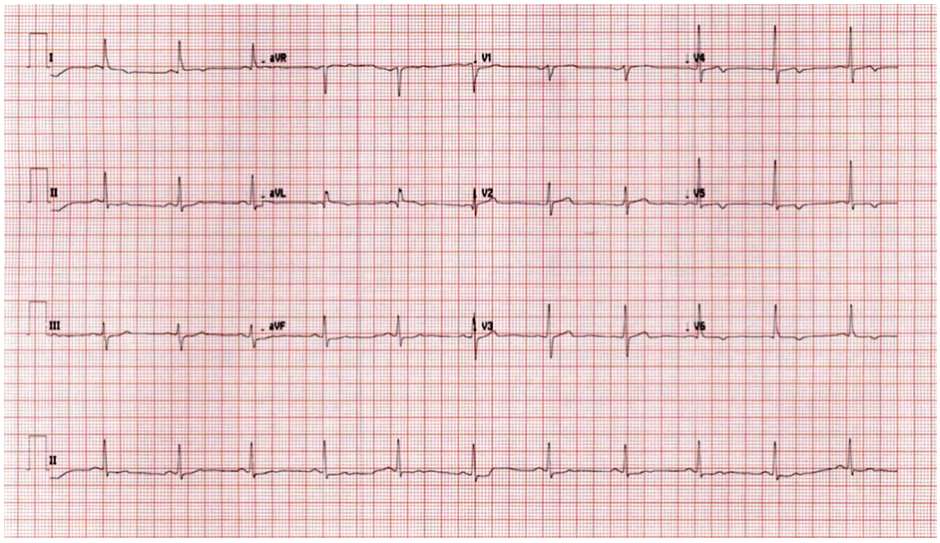

Figure 3. Tertiary care ED 12-lead EKG showing normal sinus rhythm, normal QTc interval, and T-wave inversion in Lead I, aVL, V4, V5, and V6.

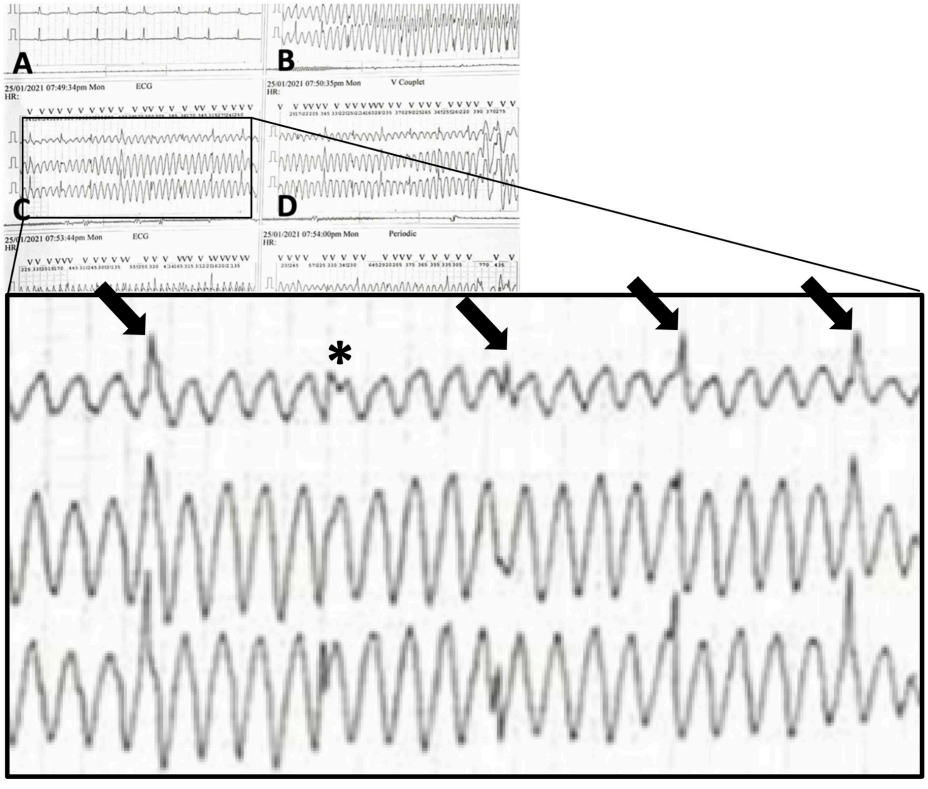

Figure 4. Zoomed image of Figure 2C. Spikes and notches are highlighted by arrows and asterisks, respectively.

quite overt (Figure 4), which represents the superimposition of the patient's normal sinus rhythm QRS complexes on pre-existing wide complex tachycardia. The raised troponin level was attributed to the patient's baseline chronic kidney disease. When the cardiologist probed further, the patient disclosed his habit of shaking his legs when anxious. "As it was a close contest, I might be shaking my legs at that time," he added. The patient was discharged as a case of pseudo-VT with reassurance on the cardiologist's advice.

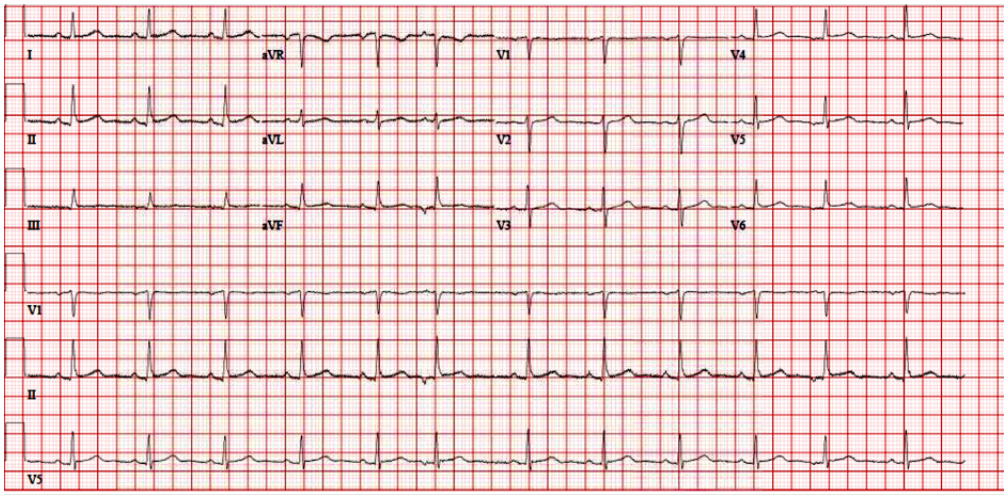

Figure 5. 12-lead EKG a week apart from the initial event. It showed normal sinus rhythm with occasional PACs but no more T-wave inversion.

We followed him up for one more month. During this period, he had COVID-19 pneumonia and remained admitted to a designated COVID-19 facility with continuous monitoring but never had another episode. A 12-lead EKG was repeated after a week of the initial event. It showed a normal sinus rhythm with occasional PACs but no more T-wave inversion (Figure 5). Additionally, there had been no evidence of atrioventricular block, accessory pathways, or QTc prolongation in any of the EKGs. We obtained three more troponin levels at day 3, 4, and 6, which were 52, 51.2, and 48.7 ng/L, respectively, coinciding with his initial values.

This affirmative justification consolidates our initial diagnosis of pseudo-VT.

DISCUSSION

European society of cardiology attributes syncope as a transient loss of consciousness due to cerebral hypoperfusion, characterized by a rapid onset, short duration, and complete spontaneous recovery.[3] 13% of elderly patients presenting to the ED and chest pain units with syncope actually have cardiac disorders as an underlying cause and arrhythmias account for more than 60% of it.[4]

VT is a life-threatening arrhythmia, which can also cause syncope and needs immediate intervention. A careful literature review shows that in non-ED-based studies, VT is the most common (80%) cause of broad complex tachycardia,[5,6] while a purely ED-based study reveals otherwise and shows only 16% of patients who presented to the ED with BCT were having VT.[1]

There are multiple reported cases where an EKG artifact was mistakenly labeled as VT, leading to unnecessary interventions.[7,8] ED physicians should correctly and timely differentiate VT from pseudo-VT, as misinterpretation might end up with some adverse outcomes for the patient.

EKG artifacts can be of two different etiologies: physiological and non-physiological. Physiological sources include muscle fasciculation, tremors, or body movements. Non-physiological causes are device malfunction, broken wires, loose connections, inadequate gel, or high-frequency electricity flow (60 Hz) in the vicinity of the EKG machine.[9]

Huang et al. devised an algorithm to differentiate VT from tremor-induced pseudo-VT. It consists of three steps: sinus sign, spike sign, and notch sign.[10] To look for a sinus sign, we need a 12-lead EKG, where one of the frontal leads (leads I, II, and III) will be showing normal P, QRS, and T waves at the time of suspected VT. This was not available for the questioned EKG trace in our case; however, we can clearly recognize a spike sign, where there are clear multiple spiked peaks at regular or irregular intervals, consistent with the patient's baseline rhythm. Similarly, a notch sign can also be appreciated, which is represented by the presence of notches just adjacent to the wide complexes (Figure 6). All of these signs have 100% specificity, with the notch sign being the most sensitive, with a sensitivity of up to 93%. The overall sensitivity, specificity, negative predictive value, and positive predictive value will be 97.3%, 100%, 98.4%, and 100%, respectively, if all three signs are used according to Huang et al.'s algorithm.[10]

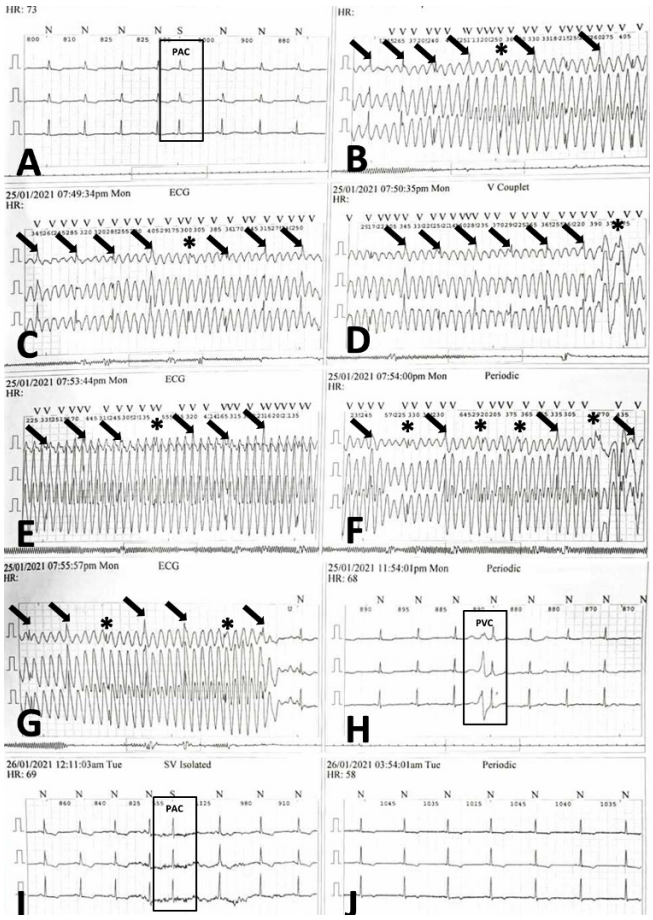

Figure 6. 10-second captures from a 24-hour Holter monitoring trace. Spikes and notches can be seen clearly represented by arrows and asterisks, respectively (B–G), scattered in the broad complex tachycardia (rate of almost 300 bpm) coinciding with the baseline normal sinus rhythm with occasional premature atrial (A, I), and ventricular complexes (H). Baseline heart rate ranges from 58 to 73 bpm.

As mentioned in the case above, the patient did not show any high-risk features and the history was very suggestive of vasovagal syncope with a clear triggering event. We opine that Holter monitoring was not indicated in this patient in the first place. Furthermore, the rhythm discovered by the Holter monitor was undoubtedly a pseudo-VT supported by positive spike and notch signs without any relevant symptoms.

CONCLUSION

In evaluating patients presenting with wide complex tachycardia, the possibility of artifacts should always be considered whenever history and physical examination do not correlate with EKG findings. That is why, an expert opinion is always warranted in challenging cases. Moreover, emergency physicians are also encouraged to familiarize themselves with recommended algorithms to differentiate between VT and tremor-induced pseudo-VT, as requesting unnecessary investigations outside of the clinical context might add more confusion and delay the diagnosis and disposition of such cases.

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AUTHORS' CONTRIBUTIONS

Muhammad Abd Ur Rehman: involved in data curation and wrote the original draft. Bilal Albaroudi: validated, wrote, reviewed, and edited the manuscript. Hina Akram: conceptualized the study and did a literature review. Shabbir Ahmad: supervised the study.

CONFLICT OF INTEREST

In compliance with the ICMJE uniform disclosure form, all authors declared no conflict of interest.

CONSENT STATEMENT

An informed written consent was obtained from the patient, and we took approval from the online platform of Hamad Medical Corporation "www.abhath.hamad.qa" for the publication of this article after anonymizing all the personal details of the patient.

ETHICAL APPROVAL

The initial manuscript of this article was submitted to the medical research center in Hamad Medical Corporation, and an approval letter was obtained from the ethical committee as well as institutional review board with approval number MRC-04-21-276.

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