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Original research

UNDERSENSING, R-ON-T PHENOMENON, AND HYPOKALEMIA: HIDDEN DANGERS OF TEMPORARY TRANSVENOUS PACING IN A STEMI PATIENT

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ABSTRACT

Background: *Temporary transvenous pacing is a critical intervention for managing bradyarrhythmias in acute myocardial infarction, particularly in patients with complete A-V block. However, this procedure carries three hidden dangers: inadequate sensing by the pacemaker, the subsequent R-on-T phenomenon, and the presence of severe hypokalemia. These factors can collectively precipitate life-threatening ventricular fibrillation.*

Case report: *We report the case of a 68-year-old male with an inferior ST-elevation myocardial infarction complicated by complete A-V block. The patient underwent emergency percutaneous coronary intervention with simultaneous temporary transvenous pacing. Two days post-procedure, he experienced a sudden syncopal episode accompanied by ventricular fibrillation, which required multiple defibrillation attempts. Careful analysis of the cardiac monitor revealed that the pacemaker failed to sense intrinsic ventricular activity, leading to pacing during the vulnerable repolarization phase (R-on-T phenomenon). Additionally, severe hypokalemia (serum potassium 2.2 mmol/L) was identified, further lowering the threshold for arrhythmia. The temporary pacemaker was deactivated, and prompt electrolyte correction stabilized the patient's rhythm, preventing further episodes. The patient remained stable and was later discharged in good condition.*

Conclusions: *This case highlights three hidden dangers associated with temporary transvenous pacing in STEMI patients: undersensing of intrinsic cardiac activity, the resulting R-on-T phenomenon, and severe hypokalemia. Recognizing and addressing these risks through meticulous device programming and vigilant electrolyte monitoring is essential for improving patient outcomes.*

Key words: *Temporary transvenous pacing, undersensing, R-on-T phenomenon, ventricular fibrillation.*

I. INTRODUCTION

Temporary transvenous cardiac pacing is a life-saving intervention for bradyarrhythmias in the setting of acute myocardial infarction, particularly for patients with high-grade atrioventricular block or hemodynamically significant bradycardia. The procedure is generally safe, and most reported complications relate to mechanical issues (lead dislodgement, perforation), infection, or vascular injury [1]. Malignant arrhythmias triggered by pacing are uncommon, accounting for a small minority of complications [2]. Among these, VF resulting from an "R-on-T" pacing stimulus is exceedingly rare but can be catastrophic if it occurs. The concept of the vulnerable period of ventricular repolarization was first described by Wiggers and Wegria in 1940. In their experiments, a stimulus delivered during the T wave could induce VF, but it required an amplitude several hundred times greater than that needed to capture the ventricle in diastole [3]

Whereas, hypokalaemia is the most frequently encountered electrolyte imbalance in hospitalized patients, making it a significant contributor to arrhythmias and related mortality in clinical

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settings. It often occurs in patients with underlying heart conditions. Hypokalaemia typically results, in descending order of probability, from (i) increased potassium loss, (ii) a transcellular shift of potassium into cells, or (iii) insufficient dietary potassium intake. Increased potassium loss is commonly a result of diuretic or laxative use, or due to diarrhea. The transcellular shift of potassium into cells may be triggered by certain medications like β2 receptor agonists, hormonal imbalances, or metabolic alkalosis. Reduced potassium intake can occur in cases of anorexia, dementia, or appetite loss associated with malignancy [4, 5]. On ECG, hypokalemia characteristically produces ST-segment depression, T-wave flattening, the appearance of U waves, and increases the propensity to ventricular ectopy. There is an inverse relationship between serum potassium level and ventricular irritability: for each 1.0 mmol/L decrease in potassium, the frequency of ventricular premature beats has been shown to increase significantly (on the order of a 20–30% higher risk) [6]. Moreover, hypokalemia predisposes to early afterdepolarizations and torsades de pointes, a form of polymorphic ventricular tachycardia often associated with a prolonged QT interval [6, 7]. Torsades can degenerate into VF, especially if there are additional triggers.

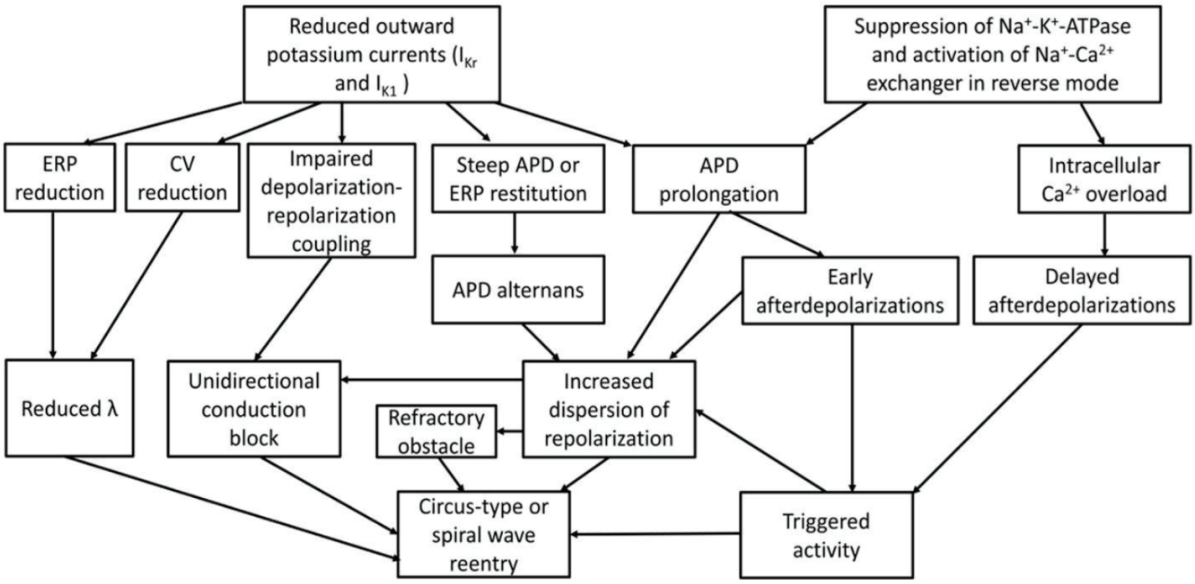

Figure 1. Overview of various electrophysiological mechanisms responsible for triggered activity and re-entry in hypokalaemia [8].

Nevertheless, in the context of myocardial ischemia or electrolyte disturbances, the threshold for fibrillation can be significantly lowered. The "R-on-T" phenomenon, wherein an ectopic beat or pacing spike occurs during the T wave, is a known trigger for VF in vulnerable hearts. We report a case of an inferior STEMI in which an undersensed beat during temporary pacing led to an R-on-T induced VF, exacerbated by severe hypokalemia.

II. CASE PRESENTATION

A 68-year-old man was hospitalized to our unit because of chest pain that had started one hour before. In admission, the blood pressure in the upper limbs was 130/80 mmHg, the pulse was 67 beats per minute (bpm). The ECG showed complete A-V block, and ST elevated in DII, DIII and aVF. Echocardiography showed severe hypokinesia in the inferior of his heart, and cardiac function of 50%. He was diagnosed with acute inferior STEMI with complete A-V block and heart failure, and his subclinical tests were done when he was waiting for emergency PCI.

This patient was taken to the digital subtraction angiography room. The doctors determined that this patient had complete A-V block and decided to insert the temporary pacing electrodes before coronary

angiography and ventricular demand pacing started. The patient's coronary angiography went smoothly, with LAD stenosis of 70% proximal segment, 80% mid-segment, LCx stenosis of 80% proximal segment, and RCA stenosis of 50% proximal segment and complete occlusion from the distal which was completely stented (Figure 2).

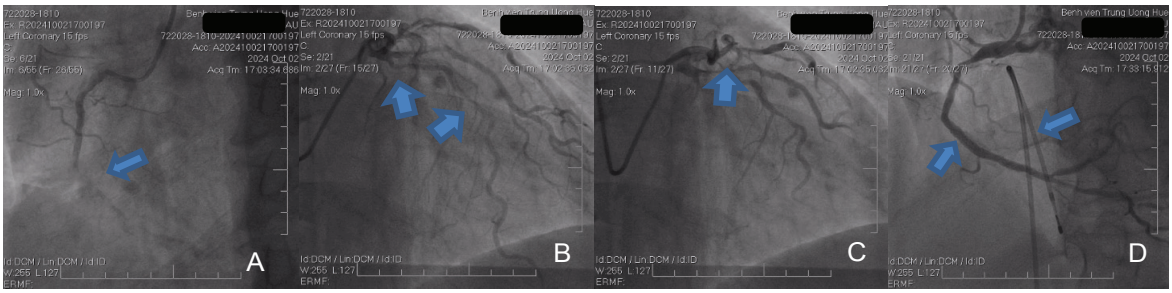

Figure 2: This patient's coronary angiography results. (1A: RCA stenosis of 50% proximal segment and complete occlusion from the distal segment; 1B: LAD stenosis of 70% proximal segment; 1C: LCx stenosis of 80% proximal segment; 1D: temporary pacemaker lead and stent deployment in RCA)

But 2 day following - up in our unit after stenting, he manifested a syncopal episode, characterized by loss of consciousness, respiratory arrest, generalized muscular rigidity and cyanosis. Ventricular fibrillation was observed on monitoring during the attack (Figure 3, 4). Defibrillation was achieved with a 270J shock after two shocks at 200J were ineffective. He was given an intravenous loading dose of amiodarone followed by a continuous infusion. His subclinical test results were given below:

A potassium infusion was started. The doctors reviewed the ECG after electric shock. It seemed to be triggered by inadvertent pacing during the repolarization phase of a non-sensed sinus rhythm. The temporary pacemaker was turned off. A vary third degree atrioventricular block (again) was noted, but the ventricular fibrillation stopped immediately and did not recur (Table 1).

Table 1: The laboratories test results.

Subclinical tests	Value	Normal	Unit
1. Blood:			
WBC	14.3	4 - 10	K/µL
NEU%	81.0	40 - 80	%
RBC	4.92	4.0 - 5.8	M/µL
HGB	15.2	12 - 16.5	g/dL
PLT	s	150 - 450	K/µL
2. Serum:			
Glucose	2.39	3.9 - 5.6	mmol/L
Urea	7.77	2.8 - 8.0	mmol/L
Creatinine	91.3	45 - 84	µmol/L
3. Electrolytes:			
Na+	143.9	136 - 146	mmol/L
K+	2.20	3.4 - 5.1	mmol/L
Cl-	93.2	101 - 109	mmol/L

Subclinical tests	Value	Normal	Unit
4. Immune:			
Troponin T-hs	> 10	< 0.014	ng/mL
CK	6082	0 - 171	U/L
CKMB	233.0	1.35 - 4.94	ng/mL

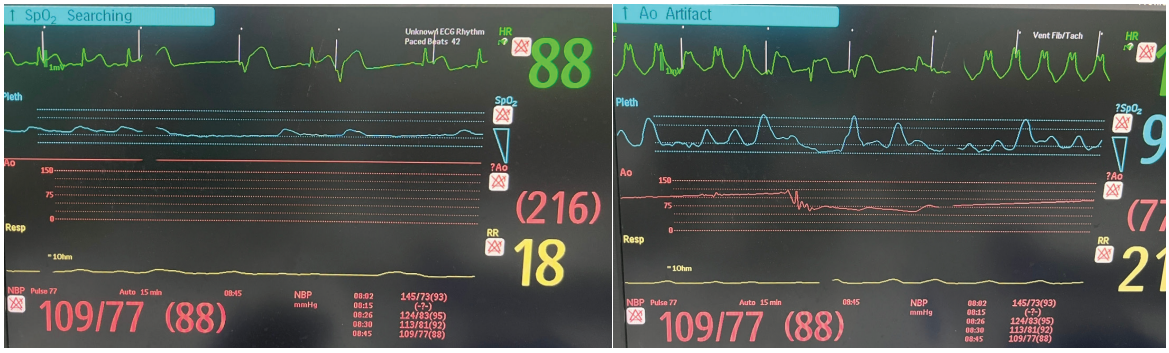

Figure 3: Ventricular arrhythmia due to undersensing in this patient

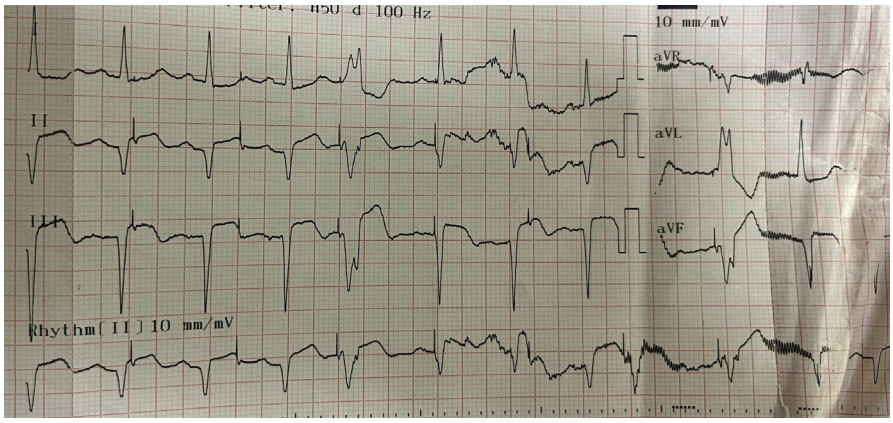

Figure 4: ECG after electric shock. It appears that the temporary pacemaker failed to sense the patient's sinus rhythm.

During following up in our unit after turning off the temporary pacemaker, the hospital course was uneventful and his ECG showed that sinus rhythm and no atrioventricular blocks. The patient was indicated for stenting in the remaining two branches, LAD and LCx. The patient's condition gradually improved. The patient was discharged in relatively good condition and treated medically with Aspirin and Ticagrelor.

III. DISCUSSION

While the phenomenon described in this case is rare, it's possible that some instances of pacemaker-induced ventricular fibrillation following temporary pacing for acute myocardial infarction go unrecognized, even with continuous ECG monitoring. Recognizing this phenomenon is crucial because the resulting arrhythmia can be fatal, although it is treatable. Most instances of ventricular fibrillation following cardiac pacing in acute myocardial infarction occur during the positioning of the temporary pacing electrode in the right ventricle, especially after inferior or right ventricular myocardial infarction [9]. Poor electrode contact with the endocardium often results in both inadequate sensing of the underlying local action potential and suboptimal pacing. This issue is generally identified, prompting electrode

repositioning. Our case report exemplifies both inadequate pacing and sensing. In cases of poor sensing, stimulation (even at a low voltage) during the repolarization phase of the cardiac cycle is recognized as a potential trigger for ventricular fibrillation (the "R on T" phenomenon) and has been observed in patients receiving pacemaker treatment [3, 10].

Several electrophysiological factors can contribute to undersensing by a pacemaker. Variability in local action potential duration and electrical inhomogeneity in the myocardium surrounding the lead can prevent the pacemaker from detecting a depolarization. The sensing electrode of a temporary pacemaker, especially if placed on or in infarcted tissue, may register only a fraction of the cardiac electrical activity. Unlike a surface ECG, which reflects the integrated electrical activity of the whole heart, a pacemaker lead detects signals only from the immediate vicinity of the lead tip [11]. If fibrotic or necrotic tissue surrounds the lead, the intrinsic QRS may not generate sufficient local voltage to cross the device's sensing threshold, even though a QRS complex is evident on the surface ECG. In our patient, the temporary pacing lead was positioned in the anterior right ventricle (RV), a commonly used location due to ease of access. The likely cause of undersensing in this case is the total occlusion of the right coronary artery, which perfuses the right ventricle. Consequently, the pacing lead may have been positioned adjacent to infarcted myocardial tissue. However, one study indicated that this anterior RV placement may offer less - than - ideal sensing and stimulation thresholds [1, 12]. The sensitivity setting for detecting the underlying cardiac rhythm is typically around 2-3mV, which is sufficient to pick up most normal or ectopic ventricular depolarizations without being overly sensitive to extraneous artifacts that could lead to pacemaker inhibition. In some cases, the sensitivity level can be adjusted, potentially reducing the issues described here. It is evident that poor sensing often coincides with inadequate pacing. The usual corrective approach is to reposition the electrode within the right ventricle [13]. However, as demonstrated in this case, the best approach is often to either turn off the pacemaker or remove it entirely.

The question of why a small electrical stimulus can trigger such serious ventricular arrhythmias is important for both clinical practice and academic study. As noted earlier, the strength of a stimulus needed to provoke this effect is hundreds of times greater than the threshold necessary to initiate a single ventricular contraction during diastole. However, this threshold can be altered in specific ways. Hoffman et al demonstrated that if a stimulus is accurately timed during a period of relatively increased excitability, the required strength to trigger repetitive firing can be reduced to roughly 20 times the diastolic threshold [14]. Additionally, Palmer et al found that a stimulus of about 10 times the diastolic threshold could initiate repetitive firing when the stimulus location was such that it activated early repolarizing tissue while adjacent myocardium remained partially refractory. In Palmer's studies, this response was associated with QRS interruption of the T wave, abnormal ventricular conduction, and repetitive firing. The fact that the induced ventricular response interrupted the T wave suggests that excitation occurs while some myocardial tissue is still incompletely repolarized. This situation likely promotes response fragmentation and possible re-entry, ultimately leading to fibrillation. Palmer achieved T wave interruption and fibrillation with small stimuli by taking advantage of asynchronous repolarization under certain conditions: stimulating early-depolarizing areas (like the septum), targeting the origin site of a preceding ectopic ventricular complex, or stimulating an ischemic ventricular area after the passage of a conducted supraventricular complex. Ischemia, known to shorten action potential duration, results in earlier recovery and a lowered threshold for fibrillation. Palmer also demonstrated that hypokalemia, by prolonging ventricular conduction, increased T wave interruption and elevated the likelihood of fibrillation [7].

In our case, the patient's potassium of 2.20 mmol/L likely also created a milieu favoring VF through prolonged repolarization and increased dispersion of refractoriness. His frequent premature ventricular complexes were noted on telemetry prior to his VF arrest, which is consistent with hypokalemia. A recent meta-analysis has highlighted a strong

inverse correlation between serum K+ channel levels and the occurrence of ventricular arrhythmias in patients with myocardial infarction. Hypokalemia is recognized not only as a risk factor for VT or VF during the acute phase of STEMI but is also linked to VF occurring before primary percutaneous coronary intervention (PPCI). An increased risk of VF prior to PPCI in patients with low potassium levels (< 3.5 mmol/L) and high potassium levels (> 5.0 mmol/L). A U-shaped relationship between potassium levels and VF risk before PPCI has been identified, with the highest VF risk seen in patients with severe hypokalemia (2.1 - 2.7 mmol/L). In emergency situations, serum K+ levels measured upon admission, either alone or alongside the Thrombolysis in Myocardial Infarction (TIMI) risk score, have been shown to provide a more accurate prediction of both short- and long-term risks for malignant ventricular arrhythmias [8, 15]. In this patient, it was not entirely clear what caused the potassium to fall to such a low level; he was not on diuretics prior to the event. We suspect that a combination of factors (possibly catecholamine-driven intracellular shifts in the setting of acute stress, and reduced oral intake during illness) contributed to his severe hypokalemia. Along with the marked decrease in serum potassium levels, this patient also exhibited hypoglycemia, with a blood glucose level of only 2.39 mmol/L. This significant drop in glucose may have contributed to the decline in serum potassium levels through mechanisms involving cardiac ion channels - particularly by inhibiting the rapid component of the cardiac delayed rectifier potassium current (IKr), one of the major potassium repolarization channels in cardiomyocytes [16]. IKr blockade leads to prolonged action potentials, resulting in QT interval prolongation and an increased risk of EADs, DADs, and the onset of ventricular tachyarrhythmias [17]. Another proposed mechanism is that hypoglycemia significantly increases levels of epinephrine and norepinephrine, which further contributes to potassium depletion [18].

In summary, our patient's VF arrest was the result of an unusual convergence of factors: a pacing system malfunction (undersensing) leading to an R-on-T premature stimulus, combined with a pro-arrhythmic internal environment caused by hypokalemia (and acute ischemia). This case illustrates that even low-output pacing stimuli can precipitate VF when they occur at precisely the wrong time in a vulnerable myocardium. It also emphasizes that electrolyte optimization are fundamental to preventing iatrogenic arrhythmias

IV. CONCLUSION

Ventricular arrhythmias in acute inferior MI patients requiring temporary pacing are a significant challenge. This case illustrates that pacemaker undersensing combined with severe hypokalemia can trigger VF via an R-on-T phenomenon. Vigilant monitoring of device function and prompt correction of electrolyte abnormalities are essential to mitigate this risk and improve patient safety.

Declaration of conflicting interests

The authors declare no conflicts of interest with respect to the research, authorship, and/or publication of this article.

Ethical approval

All details, medical records and figures were used with the written consent for publication from the patient. This case report was approved by the Research Ethics Committee of Hue Central Hospital

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