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CASE REPORT

Wavy Triple an Electrocardiographic Sign (Yasser Sign) in Hypocalcemia with Tented T-Wave and Chronic Renal Failure Diverse Management – A Case Report

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ABSTRACT
Rationale: Electrolyte disorders is a noteworthy entity in clinical practice. Hypocalcemia is a well-known serious electrolyte disorder. A Wavy triple sign of hypocalcemia (Yasser sign) is a novel an electrocardiographic sign linked to calcium deficiency. Tented T-wave is frequently identified in chronic kidney disease. Hyperkalemia is a lethal metabolic disturbance that may be a result of kidney failure.
Patient concerns: An old-aged male carpenter patient presented to the emergency department with hyperventilation syndrome, chest pain, and tetany.
Diagnosis: Chronic renal failure-induced hypocalcemia with subsequent a Wavy triple an electrocardiographic sign or Yasser sign.
Interventions: Electrocardiography, arterial blood gases, oxygenation, and echocardiography.
Lessons: The dramatic reversal of the wavy triple sign of hypocalcemia (Yasser sign) after calcium gluconate injection interpret that these signs were due to hypocalcemia of chronic renal failure. Tented T-wave is not necessary occurs by hyperkalemia.
Outcomes: There was a dramatic improvement of both clinical and electrocardiographic wavy triple sign of hypocalcemia after calcium injection.

KEYWORDS: Hypocalcemia ; Calcium ; Wavy triple sign of hypocalcemia ; Yasser sign ; Tented T-wave ;Hyperkalemia.

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INTRODUCTION

Various qualitative changes due to electrolyte imbalance may be seen on an electrocardiogram (ECG) **[1]**. The prolongation of the QTc-interval is an old non-specific ECG finding in hypocalcemia **[2]**. Wavy triple an electrocardiographic sign (Yasser Sign) is a new innovated diagnostic sign in hypocalcemia **[3]**. The analysis for this sign in the author interpretations are based on the following :

1. Different successive three beats in the same lead are affected.
2. All ECG leads can be implicated.
3. An associated elevated beat is seen with the first of the successive three beats, depressed beat with the second beat, and isoelectric ST-segment in the third one.
4. The elevated beat is either accompanied with ST-segment elevation or just an elevated beat above the isoelectric line.
5. Also, the depressed beat is either associated with ST-segment depression or just depressed beat below the isoelectric line.
6. The configuration for depressions, elevations, and isoelectricities of ST-segment for the subsequent three beats are variable from case to case. So, this arrangement non-conditional.
7. Mostly, there is no participation among the involved leads. The author intended that is not conditionally included in an especial coronary artery for the affected leads **[3]**.

Abnormal T-wave changes are seen on the ECG is either benign or severe, lethal conditions [4]. The most serious complications of T-wave abnormalities are both a

misdiagnosis of a serious T-wave pathology and delay in treatment intervention [4]. Tall T-waves or hyperacute T-waves are considered an early sign of ST-elevation myocardial infarction (MI). It may be the earliest sign of MI on the ECG. The T-waves will be widened and peaked in the leads corresponding to the artery occlusion [5]. However, tall T-waves can also be seen in both ventricular hypertrophies, depending on the distribution in the precordial leads. Furthermore, T-waves may be tall as a normal variant. Serial ECG is essential to compare all tracing with elevations in T-wave morphologies. Indeed, elevated T-waves can be seen as a normal variant in young athletes, typically in the precordial V2-V4 leads [6]. Hyperkalemia is a condition that can cause peaked T-waves. According to the degree of hyperkalemia, the tented T-waves may range on the ECG from a low amplitude to a tall peak and a sinusoidal form. The pathogenesis of the T-wave morphologies will thorough inhibition of the positively charged extracellular potassium on the repolarization of the myocardium. In initial ECG changes in hyperkalemia, the T-waves become narrow, pointed, and tall; these changes will be seen in all leads on the ECG. As the hyperkalemia deteriorates, other ECG findings may happen: decreased P-wave amplitude, a broad QRS, PR prolongation, and finally, the ECG may become sinusoidal [7,8].

CASE REPORT

A 60-year-old married, male, Carpenter, Egyptian patient presented to the emergency department with rapid difficult breathing, carpopedal spasm, chest pain, and dizziness. He had recently given a recent history of acute deterioration in renal functions and past prostatic management. The patient denied a history of cardiac or other relevant diseases. Upon examination, the patient appeared tachypneic and tetany. His vital signs were as follows: blood pressure of 140/80 mmHg, the pulse rate of 64/bpm and regular, the respiratory rate of 28/min and regular, the temperature of 37.1°C, and the pulse oximeter of oxygen (O2) saturation of 96%. No more relevant clinical data were noted during the clinical examination. The patient was admitted to the intensive care unit (ICU) as tetany, chest pain, and severe hypocalcemia. An Urgent initial ECG tracing on the emergency room showing normal sinus rhythm with Wavy triple sign of hypocalcemia (Yasser sign) in four leads of ECG V1, 2, 5, and 6) with VR; 66 bpm. There are hyperacute T-waves in V4 and 5 leads (**Figure 1**). A 100% O_2 inhalation using nasal cannula at the rate of 5 *L/min was administered.* The patient was advised to be calm and try to rest his breath. The immediate ABG showed compensated respiratory alkalosis (PH;7.39 mmHg, PCO2;33.3 mmHg, HCO3;21.1 mmHg, and PaO2; 96 mmHg). Measured random blood sugar was 98 mg/dl. Full blood count (FBC); Hb was 10.1 *g/dl*, RBCs; $4.9*10^3/mm^3$, WBCs; $8.5*10^3/mm^3$ (Neutrophils; 57.6 %, Lymphocytes: 36.1%, Monocytes; 6.3%), Platelets; $154*10^3/mm^3$. SGPT;28 U/L, SGOT;39 U/L, serum creatinine;8.4 mg/dl, blood urea; 148 mg/dl. Plasma sodium was (144 mmol/L). Serum potassium was (5.4 mmol/L). Serum calcium showing hypocalcemia with ionized calcium; 0.43 mmol/L. The troponin test was negative (less than 2 ng /L). Later echocardiography was normal with EF 55%. No more workup was done. Two calcium gluconate ampoules (10 ml 10% over IV over 20 minutes) were given as an emergency dose. Maintenance therapy with IVI calcium gluconate ampoules (10% with the rate; 0.5 mg/kg/hour over IV over 6 hours) was the infused. The patient as discharged within 24 hours of clinical and electrocardiographic improvement. The second ECG tracing was taken within 10 minutes after 2 amp of IV calcium therapy showing the disappearance of above Wavy triple sign of hypocalcemia (Yasser sign) but still, there are hyperacute T-wave in V4-6 leads with VR; 68 bpm (**Figure 2**). Serum ionized calcium after the correction was 0.87 mmol/L). The patient was discharged within 12 hours after relieving, and electrocardiographic normalization. Oral calcium and vitamin-D preparation were prescribed on discharge. Future serial ionized calcium and nephrologist consultation was advised.

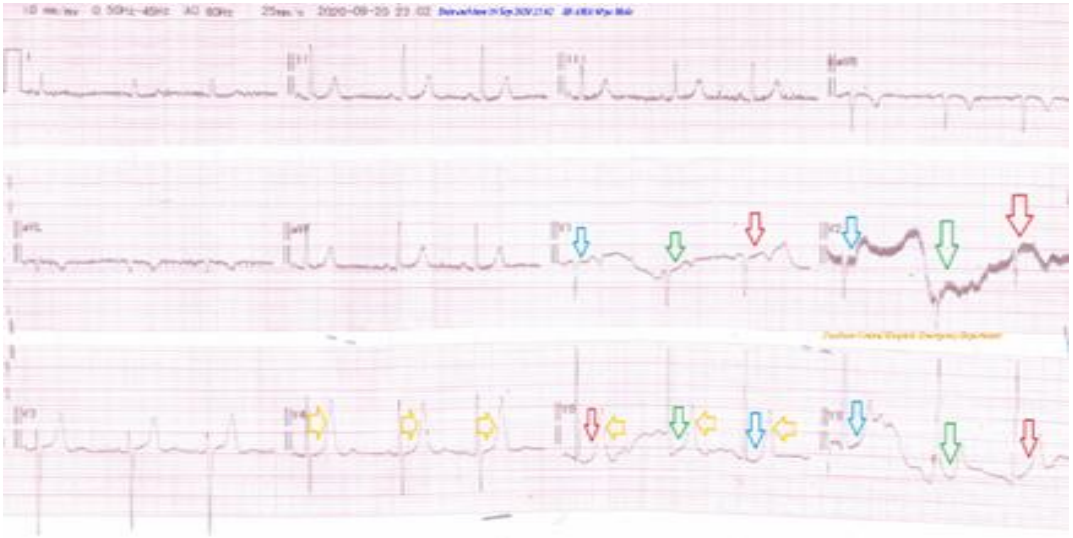

Fig.1 : An initial ECG tracing on emergency arrival showing normal sinus rhythm with Wavy triple sign of hypocalcemia (Yasser sign) in four leads of ECG V1, 2, 5, and 6) with VR; 66 bpm. Red arrows indicate an elevated beat. Green arrows indicate a depressing beat. Blue arrows indicate isoelectric beat. There are hyperacute T-wave in V4 and 5 leads (gold arrows).

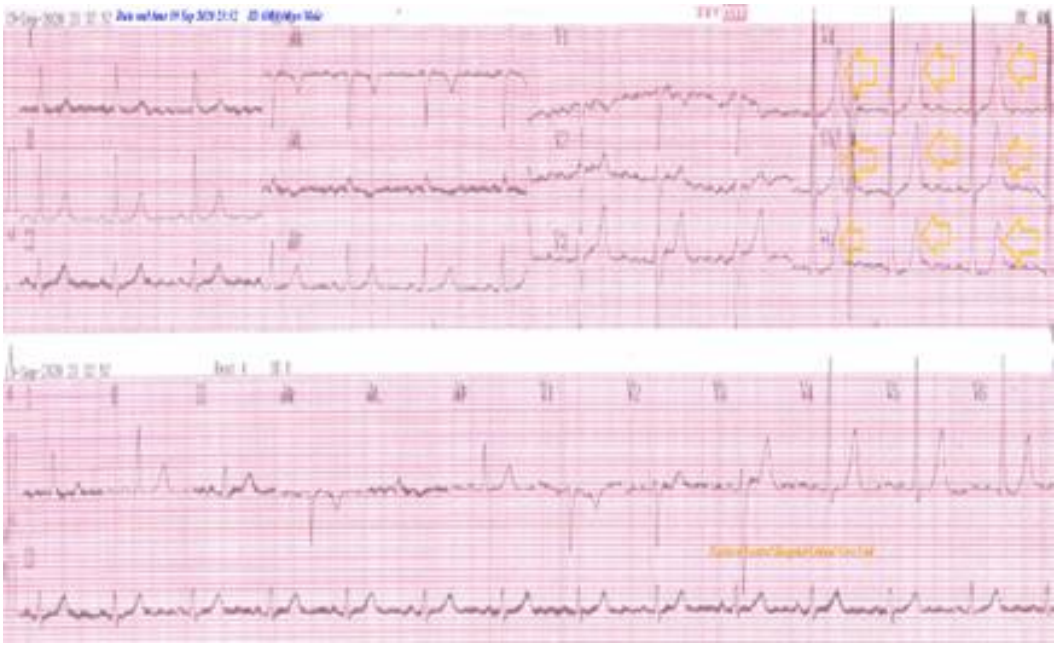

Fig.2: The second ECG tracing was taken within 10 minutes after 2 amp of IV calcium therapy showing the disappearance of above Wavy triple sign of hypocalcemia (Yasser sign) but still, there are hyperacute T-wave in V4-6 leads (gold arrows) with VR;68 bpm.

DISCUSSION

Overview: An old-aged male carpenter patient presented to the emergency department with hyperventilation syndrome and tetany.

Study Objective: The primary objective for my case study was the presence of hyperventilation syndrome, tetany, and severe hypocalcemia.

The secondary objective for my case study was the priority in the management of hyperventilation syndrome, tetany, and severe hypocalcemia.

Results : The dramatic reversal of Wavy triple sign of hypocalcemia (Yasser sign) after calcium gluconate injection interpret that these signs were due to hypocalcemia.

The negative troponin test with non-conclusive ECG changes for ischemic heart disease (IHD), and normal echocardiography will quietly exclude the presence of acute myocardial infarction (AMI).

The etiology of the hypocalcemia in the current case is unknown. Hyperventilation syndrome which causing respiratory alkalosis was the possible cause.

Tented T-wave in the current case is limited to V-6 ECG leads is mostly not caused by hyperkalemia due to chronic kidney disease. Tented T-wave ECG leads due to hyperkalemia is usually affect all ECG leads.

The differential diagnosis of Peaked T-waves are The AMI, vasospastic angina, normal variant, hyperkalemia, acute pericarditis left bundle branch block (LBBB), and left ventricular hypertrophy (LVH) [8].

The present case is not comparable with similar conditions. There are no similar or known cases with the same management for near comparison.

Study Questions: How did you manage the current case? What are the possible causes of a tented T-wave in the ECG changes?

Study Limitations: There are no known limitations in the study.

Recommendations: It is recommended to widening the research in clearing the simultaneous presence of Wavy triple sign (Yasser sign) with hypocalcemia in chronic renal failure.

CONCLUSION

The dramatic disappearance of Wavy triple sign (Yasser sign) is meaning that this sign was lonely due to severe hypocalcemia in chronic renal disease. However, the tented T-wave is not essentially due to hyperkalemia.

ABBREVIATIONS

ABG: Arterial blood gases .
ECG: Electrocardiogram.
IHD: Ischemic heart disease.
MI: Myocardial infarction .
O2: Oxygen.
VR: Ventricular rate.

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COMPETING INTERESTS

The author declares no competing interests with this case.

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PATIENTS CONSENT

Written informed consents were obtained from the patients for the publication of this case report.

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