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Case Report

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Electrical Injury-Inducing Fleeting Claw Hand Mimic-Tetany Sign, Yasser's Cardiac Contracture, Fading Mosaic Palm Sign, and ECG Haphazard Yasser's Syndrome-Oxygen Reversal and Medical Discoveries

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Abstract

Introduction: Electrical injuries (EIs) are a common health problem affecting the general population and may carry high morbidity and mortality. Electrical injuries may induce lethal ventricular arrhythmias. An early and quick recognition and diagnosis of frequent, serious, and late ECG manifestations of EIs is an essential step. Arrhythmias on the ECG represent the leading and most serious complications after electrical injury. Oxygen (O2) is a commonly neglected nutrient because of its entrance inside the human body through the lungs. Household alternating current (AC) at frequencies between 15 and 150 Hz repetitively stimulates muscle contraction, causing skeletal muscle tetany.

Case presentation: A 17-year-old single adolescent Egyptian male student was admitted to the intensive care unit (ICU) after EI with chest pain and variant ECG changes. The case was diagnosed as electrical injury-inducing Fleeting claw hand mimic-tetany sign, Yasser's cardiac contracture, Yasser's fading palm sign and haphazard ECG changes. The interventions were oxygenation, ABG, ECG, and echocardiography. Gradual, dramatic clinical and electrocardiographic improvement occurred.

Conclusion: Fleeting claw hand mimic-tetany, Fading mosaic palm sign, Yasser's cardiac contracture, and ECG haphazard Yasser's syndrome are innovations in clinical medicine. Oxygen is a vital therapy to counteract the devastating deep-tissue destruction caused by electrical injuries. Differentiation between tetany and contracture is inevitable.

Keywords: Fleeting claw hand, Mimic-tetany sign, Yasser's cardiac contracture, Fading mosaic palm sign, and ECG haphazard Yasser's syndrome, Oxygen reversal, Discoveries.

Introduction

Electrical injuries (EIs) are a common health problem affecting the general population and may carry high morbidity and mortality.[1] EIs are likely fatal.[2] Most cases cause no injury. Electricity can cause injury in a variety of mechanisms, which may still hidden from the unsuspecting physician until it is too late.[3] They include electrocution, electric shock, burns, and secondary injuries. There are two types of EIs: high- or low-voltage contact. Low voltage injuries often happen in the home, but high voltage injuries are usually work-related. Both types may be fatal. External manifestations often belie the severity of deeper wounds.[4] EIs can directly affect the heart, brain, cell membranes, and vascular smooth muscle, and they may pass through body tissues. magnitude of energy delivered, voltage, resistance to current flow, type of current, duration of contact with the current source, and current pathway are factors that determine the site and severity of electric trauma.[5] Arrhythmias represent the leading and most serious complications after electrical injury. Immediate arrhythmia after electrical exposure is evidence of cardiac injury.

However, spontaneous recovery of arrhythmias due to electrical injury may occur. Sometimes, there is a delayed presentation of arrhythmias post-electrical injury. Both sinus bradycardia and sinus tachycardia are frequent ECG manifestations. A newly diagnosed atrial fibrillation (AF), frequent multifocal atrial complexes, sinus node arrest with atrial escape rhythm, prehospital-resolved ventricular fibrillation (VF), ventricular bigeminy, and recurrent non-sustained ventricular tachycardia (VT) may be presented findings. Generally, EI-inducing arrhythmias are mostly functional, benign, and resolved within a few hours post-hospitalization. VF represents the most common serious arrhythmia, occurring in 60 % of patients with electrical injuries.[1] Tetany may lead to sustained contact, and serious arrhythmia serious tissue destruction and secondary injuries which result from short contact.[4] A 24-hours hospitalization monitoring for arrhythmia is an essential. Even with a short ECG monitoring period.[6] Superficial, partial-thickness, and full-thickness thermal burns are probable to be after EI. Dry skin (DS) show a higher resistance (about 100,000 ohms), generating heat when exposed to a current, causing skin burns, and even to adjacent tissue. So, DS is protective to internal organs because it prevents passage of current. But the protective mechanisms are lost with wet skin, show a much lower resistance (less than 2500 ohms) and generates less heat but passes more current to internal organs for an equivalent voltage.[7] Household alternating current (AC) at frequencies between 15 and 150 Hz repetitively stimulates muscle contraction, causing skeletal muscle tetany. When the site of exposure is the palm, the victim may actually grasp the source because the flexors of the hand and arm are stronger than the extensors, prolonging the duration of contact and perpetuating tissue injury (ie, "can't let-go current" or "let-go threshold"). It is for this reason that a 20mA current may not be perceptible at 10 Hz, but the same current may cause respiratory paralysis or ventricular fibrillation at a higher frequency.[3,7] Household AC is a common cause of more serious injury because of its arrhythmogenic frequency that can cause prolonged exposure from hand tetany.[7] Oxygen (O2) is a commonly neglected nutrient because of its entrance inside the human body through the lungs. O2 is ideal of all other nutrients.[8] O2 is essential for human cells to maintain and support aerobic respiration, which takes place in the mitochondria, which are considered a nutrient. Without O2, mitochondrial respiration and oxidative phosphorylation cannot take place.[9]

In this manuscript, I report a case of a single adolescent Egyptian male student who was admitted to the intensive care unit (ICU) after an electrical injury with chest pain and variant ECG changes. The case was diagnosed as an electrical injury, inducing the innovations "Fleeting claw hand mimic-tetany sign, "Yasser's cardiac contracture", "Fading mosaic palm sign, and "ECG haphazard Yasser's syndrome" in a middle-aged male patient.

Case presentation

A 17-year-old single adolescent Egyptian male student was admitted to the intensive care unit (ICU) after an electrical injury with chest pain and variant ECG changes. Tremors, palpitations, dizziness, and fatigue were associated symptoms. He gave a recent history of unintended catching of bared electric cable 20 minutes ago. The patient gave no history of medical diseases, drug history, substance of abuse, and like attack. Upon general physical examination; generally, the patient had tachypnea, fine tremor, anxious, showed a severe pallor, yellowish face, and distressed. A photo of the patient's right hand of the patient on the initial ICU presentation showed a claw-hand mimic contracture and tetany with brownish and whitish discoloration spots (Figure 1).

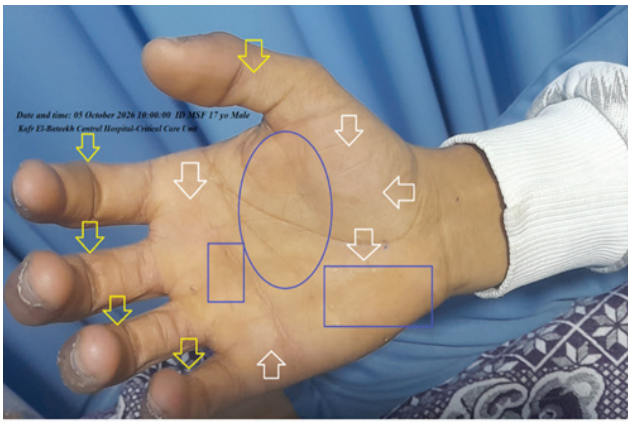


Figure 1. A photo of the patient's right hand of the patient on the initial ICU presentation showed a claw-hand mimic contracture (yellow arrows) with brownish (dark blue circle and rectangles) and whitish discoloration spots (white arrows).

His vital signs were a regular pulse rate (VR of 80), blood pressure (BP) of 110/80 mmHg, respiratory rate of 22 bpm, a temperature of 36.0°C, pulse oximetry oxygen saturation (SpO2) of 95%, and GCS of 15/15. No more relevant clinical data were noted during the clinical examination. The patient was admitted to the ICU after electrical injury with chest pain and variant ECG changes. Initially, the patient was treated with O2 inhalation by O2 inhalation central oxygen system (100%, by simple mask, 5L/min). Two initial calcium gluconate ampoules (10 ml 10% over IV over 20 minutes) were given as an emergency dose with maintenance therapy with calcium gluconate ampoules (10% with the rate; 0.5 mg/kg/hour over IV over 6 hours) was the infused was added. Calcium gluconate IV was stopped later after laboratory assessment of ionized calcium level. The patient was hourly monitored for vital signs and O2 saturation. The initial ECG was obtained on initial presentation in the Emergency Department (ER), showing NSR at a (VR of 82), tremor artifacts, A wavy triple sign in II, V4, and V6 leads, ST-segment depression with T-wave inversion in lead III, and small peaked T waves in V2 and V3 leads. There are sporadic inversed QRS complexes in aVR, aVL, and aVF leads. There is also sporadic QTc prolongation in a single QTc interval in lead V2 (Figure 2A). The second ECG tracing was obtained in the ER within 47 seconds of the above ECG tracing, showing NSR, A wavy triple sign in II, aVF, and V5 leads, and small peaked T waves in V2 and V3 leads. There is sporadic T-wave inversion in a sporadic QRS complexes in V1-3 leads. There is also a spike sign in I, II, and III leads (Figure 2B). The third ECG tracing was obtained in the ICU within 18 hours of the above ECG tracing and after O2 inhalation of the above ECG tracing, showing NSR and A wavy triple sign in V4-5 leads (Figure 2C).

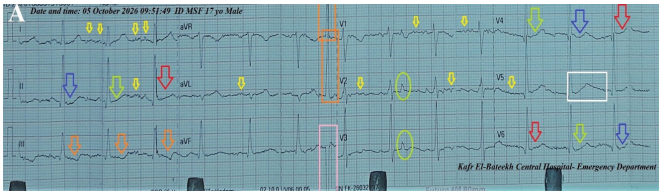


Figure 2. Serial ECG tracings; A. Tracing was obtained on initial presentation in the ED, showing NSR at a (VR of 82), tremor artifacts (small yellow arrows), A wavy triple sign in II, V4, and V6 leads (dark blue, lime, and red arrows), ST-segment depression with T-wave inversion in lead III (golden arrows), and small peaked T waves in V2 and V3 leads (lime circles). There are sporadic inversed QRS complexes in aVR, aVL, and aVF (golden and rosy rectangles). There is also sporadic QTc prolongation in a single QTc interval in lead V2 (white rectangle).

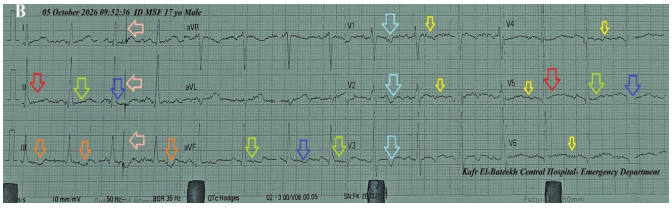


Figure 2B. Tracing was obtained in the ED within 47 seconds of the above ECG tracing, showing NSR at (VR of 90), tremor artifacts (small yellow arrows), A wavy triple sign in II, aVF, and V5 leads (dark blue, lime, and red arrows). There are sporadic T-wave inversions in V1-2leads (light blue arrows). There is also a spike sign in I, II, and III leads (rosy arrows).

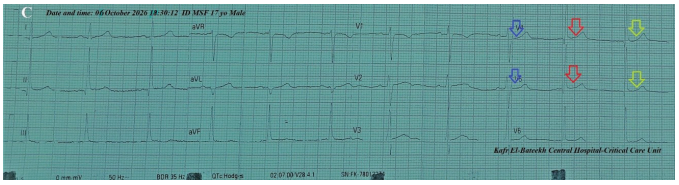


Figure 2C. Tracing was obtained in the ICU within 18 hours and after O2 inhalation of the above ECG tracing, showing NSR at (VR of 65) and A wavy triple sign in V4-5 leads (dark blue, lime, and red arrows).

The initial complete blood count (CBC); Hb was 12.5 g/dl, RBCs; 3.6*103/mm3, WBCs; (missed)*103/mm3, Platelets; 142*103/mm3. ABG showed partially compensated respiratory alkalosis. SGPT was (27 U/L) and SGOT was (30 U/ L). Serum creatinine was (1.0 mg/dl) and blood urea was (37mg/dl). RBS was (98 mg/dl). Total calcium was (2.47 mmol/l). Ionized calcium was (1.20 mmol/l). Plasma sodium was (140.6 mmol/l). Serum potassium was 3.73 mmol/l). Serum chloride was 108.0 mmol/l). PH was 7.44. The troponin test was negative. A plain PA CXR was obtained on initial presentation in the ED, showing a zigzag folding rule that is more at the right heart border and less at the left border. (Figure 3A). Echocardiography was done on the third day of the presentation, showing trivial tricuspid regurgitation and diastolic dysfunction with an EF of 71% (Figure 3B). Electrical injury-inducing the innovations "Fleeting claw hand mimic-tetany sign", "Yasser's cardiac contracture", "Fading mosaic palm sign", and "ECG haphazard Yasser's syndrome" were the most probable diagnoses. Within 24 hours of the above management, the patient finally showed nearly complete clinical and ECG improvement. The patient was discharged with further recommended cardiac follow-up.

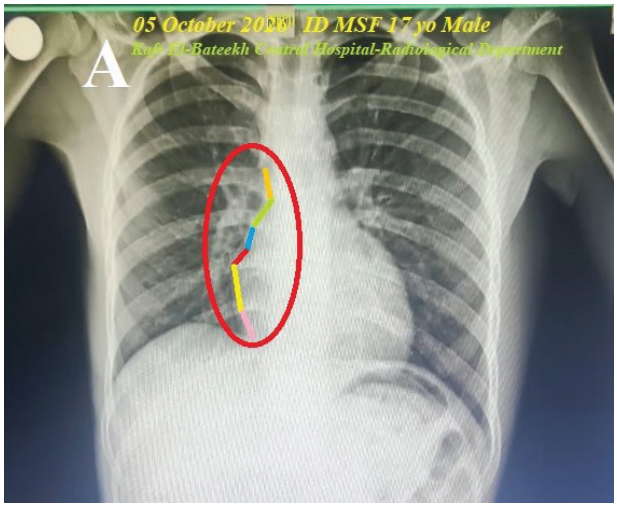


Figure 3A. A plain PA CXR was obtained on initial presentation in the ED, showing a mimic zigzag folding rule that is more at the right heart border (discolored line and red circle) and less on the left border.

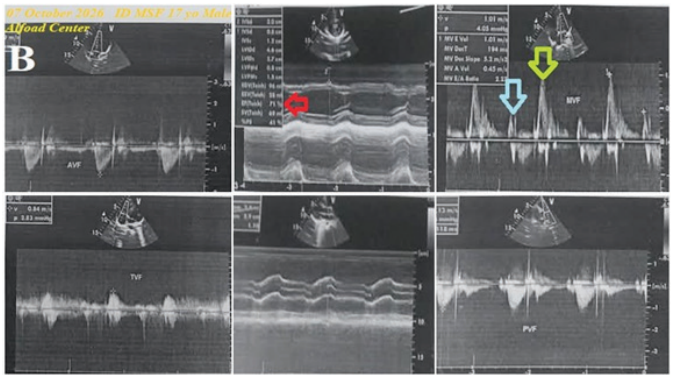


Figure 3B. Echocardiography was done on the third day of the presentation, showing trivial tricuspid regurgitation and diastolic dysfunction (lime and light blue arrows) with an EF of 71% (red arrow).

Discussion

Overview: A single adolescent Egyptian male student was admitted to the intensive care unit (ICU) after an electrical injury with chest pain and variant ECG changes. The case was diagnosed as electrical injury-inducing "Fleeting claw hand mimic-tetany sign, "Yasser's cardiac contracture", "Fading mosaic palm sign', and "ECG haphazard Yasser's syndrome". The primary objective for my case study was the presence of a single adolescent Egyptian male student was admitted to the intensive care unit (ICU) after an electrical injury with chest pain and variant ECG changes in ICU. The secondary objective for my case study was the question: how did you manage the case? There was a recent history of direct contact with a bare electrical cable. The presence of severe generalized pallor with tachypnea, anxiety, fine tremors, and palpitations may be a usual reflective autonomic manifestation of electrical injury. The presence of a claw-like hand (Figure 1) with normal ionized calcium, may support the exclusion of tetany. This claw-like hand disappears after O2 inhalation, which will strengthen the role of O2. The claw-like hand that disappears after O2 inhalation is a new description. It may be named "Fleeting claw hand mimic-tetany sign" (Figure 4). There is no known pathogenesis for this sign. The current of low voltage inducing transient or fleeting contracture may be a suggested theory. Otherwise, the presence of small local bullae in the palm of the affected right hand during electrical injury that rapidly disappear, with no local burn and no inlet or outlet for the electrical wound. This may confirm that the hand of patient was a dry and not a wet. Also, the current was low voltage. A zigzag folding rule that is more at the right heart border and less at the left border in a plain CXR view (Figures 3A and 4).) may be evidence of cardiac contracture. There is no known pathogenesis for this sign. The current of low voltage, fluctuating, and passing to the cardiothoracic may be a suggested interpretation theory. This is a new description. It may be named "Yasser's cardiac contracture" (Figure 4). The palm of the right hand showed different areas of brown alternating with white discoloration (Figure 1). This is a new description. It may be named "Yasser's fading palm sign" (Figure 4). There is no known pathogenesis for this sign. The suggested interpretation is that the brown discoloration may be a pre-burn stage of electrical injury, which may extend in size and in depth according to the current voltage and duration. White discoloration may indicate diminished capillary perfusion. The presence of spike sign (Figure 2B) which is type artifact may be evidence of irritability of the patient Indeed, the author observed that there are contradictory changes in the serial ECG tracings (Figure 2A-C). These are the following findings:

1. Sporadic inverted QRS complexes aVR, aVL, and aVF leads.

2. A wavy triple sign in II, V4, and V6 leads despite a normal ionized calcium level; ST-segment depression.
3. T-wave inversion in lead III only.
4. Small peaked T waves in leads V2 and V3.
5. Sporadic inverted QRS complexes in aVR, aVL, and aVF leads (Yasser's QRS complex reversal).
6. Sporadic QTc prolongation in a single QTc interval in lead V2, and sporadic T-wave inversions V1-3 leads.

This is a new description. It may be named "ECG haphazard Yasser's syndrome" (Figure 4). No pathogenesis is known for this syndrome. Randomization of the electrical current in the heart may be one possible explanation. The dramatic reversal of ECG findings after O2 inhalation may strengthen its role in the management of electrical injuries. I can't compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison. There are no limitations of the current case report.

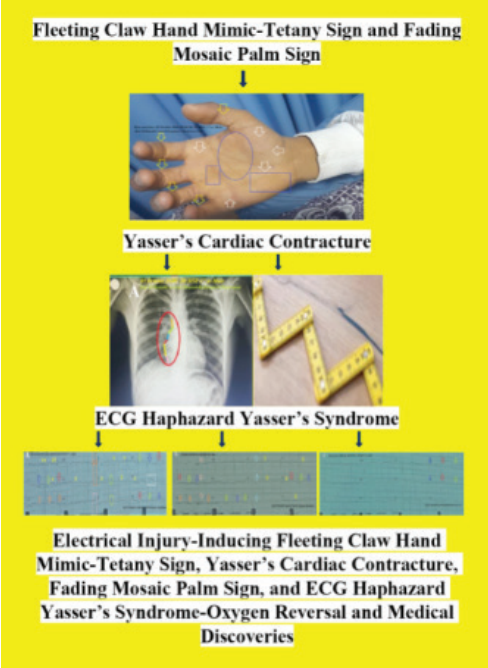


Figure 4. The author's graphical presentation of Fleeting claw hand mimic-tetany, fading mosaic palm sign, Yasser's cardiac contracture, and ECG haphazard Yasser's syndrome.

Conclusion and Recommendations

- Fleeting claw hand mimic-tetany, Fading mosaic palm sign, Yasser's cardiac contracture, and ECG haphazard Yasser's syndrome are innovations in clinical medicine.
- Oxygen is a vital therapy to counteract the devastating deep-tissue destruction caused by electrical injuries.
- Differentiation between tetany and contracture is inevitable.

Conflicts of interest

There are no conflicts of interest.

Acknowledgment

I wish to thank my wife for saving time and improving the conditions for helping me.

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