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Yasser Mohammed Hassanain Elsayed*

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Case Report

Yasser's Wenckebach Reversal Phenomenon in a Caudate Nucleus Stroke, COVID-19 Pneumonia, Calculous Cholecystitis, and Ventricular Pacing-A new Cardiovascular Discovery and Interpretation

Yasser Mohammed Hassanain Elsayed*

Critical Care Unit, Kafr-El-Bateeck Central Hospital, Damietta, Egyptian Ministry of Health, Egypt.

***Corresponding Author:** Yasser Mohammed Hassanain Elsayed, Critical Care Unit, Kafr-El-Bateeck Central Hospital, Damietta, Egyptian Ministry of Health, Egypt.

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Abstract

Rationale: Wenckebach phenomenon is a progressive prolongation of each successive PR interval and is mostly due to a reversible conduction block at the level of the AV node—Malfunctioning AV nodal cells. Caudate nucleus stroke presents with non-progressive symptoms, speech disturbances, chorea, and migraine. COVID-19 infection greatly varies from asymptomatic infection to severe pneumonia. Chronic cholecystitis is a chronic inflammation of the gallbladder resulting in mechanical or physiological dysfunction in its emptying. Ventricle pacing and sensing (VVI) involves ventricles instead of the atrium. If native ventricular activity is sensed then pacing is inhibited. Patient concerns: An elder housewife widow Egyptian female patient presented to the intensive care unit (ICU) with an acute right-side cerebrovascular accident, left-side deviation in the face, migraine, acute confusion, and COVID-19 pneumonia on top of diabetes, hypertension, chronic cholecystitis and ventricle pacing and sensing (VVI).

Diagnosis: Yasser's Wenckebach reversal phenomenon in a caudate nucleus stroke, COVID-19 pneumonia, diabetes, hypertension, calculous cholecystitis, and ventricular pacing.

Interventions: Chest CT scan, brain CT scan, electrocardiography, oxygenation, and Ventricle pacing. Outcomes: A clinical and electrocardiographic improvement occurred. Lessons Yasser's Wenckebach reversal phenomenon is a new cardiovascular discovery. A caudate nucleus stroke, COVID-19 pneumonia, calculous cholecystitis, and ventricular pacing are associated with the new phenomenon. The normalization of T-wave inversion in anterior leads is also parallel with the reversal of the progressive prolongation of each successive PR interval until a dropped beat from the left to the right. This will signify the Yasser's Wenckebach reversal phenomenon as a signal for the above normalization.

Keywords: yasser's wenckebach reversal phenomenon; wenckebach phenomenon; caudate nucleus; cerebrovascular accident; pacing; cholecystitis; covid-19

Abbreviations

COVID-19: Coronavirus disease 2019
AMI: Acute myocardial infarction
CBC: Complete blood count
CN: Caudate nucleus
CVA: Cerebrovascular accident
ECG: Electrocardiography
ICU: Intensive care unit
LAD: Left axis deviation
LBBB: Left Bundle Branch Block
STEMI: ST-segment elevation myocardial infarction
VR: Ventricular rate
VVI: Ventricle pacing and sensing

1. Introduction

Clinically, manifestations of Coronavirus disease 2019 (COVID-19) infection greatly vary from asymptomatic infection to severe pneumonia [1]. A COVID-19 infection commonly has neurologic manifestations. In Mao L, et al. (2020) study in Wuhan, China, there was 2.3% of 214 hospitalized patients with COVID-19 showed an ischemic cerebrovascular accident (CVA) or stroke [2]. A very high cumulative incidence of ischemic CVA was reported in severe COVID-19 pneumonia [3]. Most patients with ischemic CVA had conventional vascular risk factors, and traditional CVA mechanisms were common [4]. The high rate of arterial and venous thromboembolic events diagnosed within 24 hours of admission by image scanning among the tested COVID-19 patients [5]. The vast majority of ischemic CVA patients associated with COVID-19 have common vascular risk factors such as hypertension, hyperlipidemia, and diabetes [6]. Although the cause of ischemic CVA associated with COVID-19 is unclear, previous studies have hypothesized that inflammatory cytokine storms may be a trigger for hypercoagulable state or endothelial damage [7]. There are paired caudate nuclei (CN) that represent a C-shaped subcortical structure that lies deep inside the brain near the thalamus. It plays a critical role in various neurological functions [8]. The CN functions not only in planning the execution of movement, but also in learning, memory, reward, motivation, emotion, and romantic interaction [9,10]. Caudate nucleus stroke presents with non-progressive symptoms, speech disturbances, chorea-acanthocytosis, and migraine [11,12]. CN stroke can prevent future airway closure during swallowing and may predict continuing aspiration issues [12]. CN stroke can have widespread effects depending upon their extent and the potential involvement of other nearby tissues. Left CN infarcts are associated with symptoms such as cognitive dysfunction, motor deficits, and various speech deficits [12]. CN stroke has a good prognosis [8]. Chronic cholecystitis is a chronic inflammation of the gallbladder resulting in mechanical or physiological dysfunction in its emptying. Chronic cholecystitis mostly occurs in the setting of cholelithiasis. The risk factors for cholelithiasis include female sex, obesity, rapid weight loss, pregnancy, advanced age, and Hispanic or Pima Indians. Symptomatic patients with chronic cholecystitis usually present with dull right upper abdominal pain that radiates around the waist to the mid back or right scapular tip. Leukocytosis and abnormal liver function tests may not be present. Computerized tomography (CT) with intravenous contrast usually reveals cholelithiasis. The preferred treatment for chronic cholecystitis is elective laparoscopic cholecystectomy [13]. Ventricle pacing and sensing (VVI) is similar to the atrial pacing and sensing (AAI) mode but involves ventricles instead of the atrium. If native ventricular activity is sensed then pacing is inhibited. If no native activity is sensed for a pre-determined time then ventricular pacing is initiated. Ventricular pacing spike precedes the QRS-complex. Right ventricle pacing lead placement results in a QRS morphology similar to LBBB. Left epicardial pacing lead placement results in a QRS morphology similar to RBBB. ST segments and T waves should be discordant with the QRS complex i.e. the major terminal portion of the QRS complex is located on the opposite side of the baseline from the ST segment and T wave. VVI is used in patients with chronic atrial impairment e.g. atrial fibrillation or flutter [14]. Wenckebach phenomenon ((Mobitz I block) is a progressive prolongation of each successive PR interval. It is characterized by the highest PR-interval in a non-conducted P-wave, longest PR-interval immediately before dropped beat, shortest PR-interval immediately after dropped beat, relatively remains constant P-P interval, greatest PR interval duration is typically between the first and second beats of the cycle, and the progressively shortens RR interval with each beat of the cycle [15,16]. Wenckebach phenomenon is often a normal variant [17]. Wenckebach phenomenon is mostly due to reversible conduction block at the level of the AV node. Malfunctioning AV nodal cells tend to progressively fatigue until they fail to conduct an impulse. Beta-blockers, calcium channel blockers, digoxin, amiodarone athletes, inferior myocardial infarction, myocarditis, hyperkalemia, cardiomyopathy, Lyme disease, mitral valve repair, Tetralogy of Fallot repair are implicated causes [15,16]. It is usually a benign rhythm, causing minimal hemodynamic disturbance and with a low risk of progression to third-degree heart block. Asymptomatic patients do not require treatment. Symptomatic patients usually respond to atropine. Permanent pacing is rarely required [16].

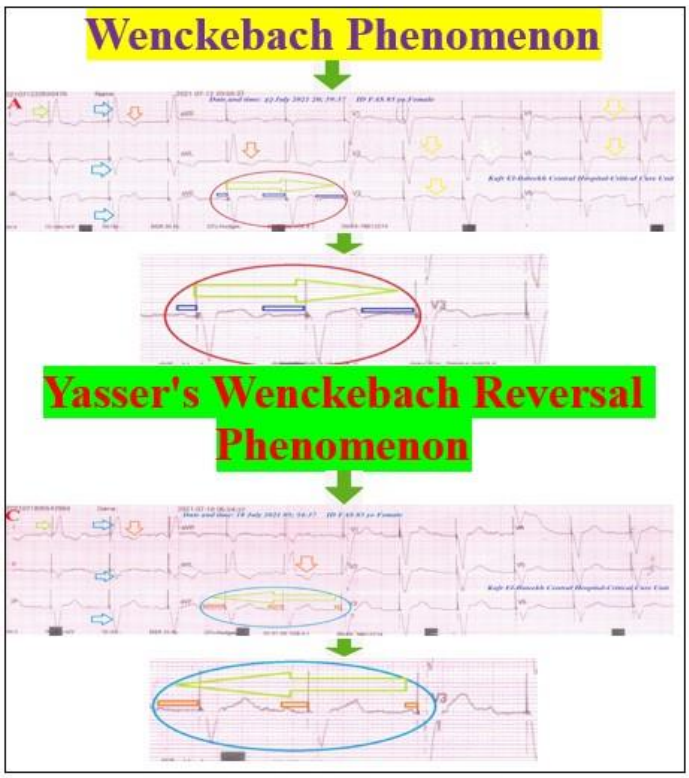

Figure 1-Showing graphical presentation of Yasser's Wenckebach Reversal Phenomenon

2. Case Report

An 85-year-old housewife widow Egyptian female patient presented to the intensive care unit (ICU) with acute weakness in the right side of the body, left side deviation in the face, migraine, and acute confusion. Generalized body aches, sore throat, cough, fatigue, loss of appetite, and loss of smell were associated symptoms. The patient started to complain of fever 7 days ago. She has a recent direct contact with a confirmed case of COVID-19 pneumonia 15 days ago. She has a history of hypertension on captopril tablets (25mg, OD) and diabetes on long-acting insulin (70/30, BID). Upon general physical examination; generally, the patient has confusion, right hemiplegia, left facial palsy, quick involuntary movement of the hands and feet, with a regular pulse rate of 70, blood pressure (BP) of 130/70 mmHg, respiratory rate of 17 bpm, GCS of 10/15, a temperature of 36.9 °C, and pulse oximeter of oxygen (O2) saturation of 97%. She seemed obese. Tests for latent tetany were positive. There are left-side hemiparesis, right-side facial palsy, and slurred speech. No more relevant clinical data were noted during the clinical examination. The patient was admitted to the ICU with an acute cerebrovascular accident with COVID-19 pneumonia in chronic calculous cholecystitis and ventricular pacing. Initially, she was treated with O2 inhalation (100%, by nasal cannula, 5L/min). The patient was maintained and treated with cefotaxime; (1000 mg IV TID), azithromycin tablets (500 mg, OD), and hydrocortisone sodium succinate (100 mg IV BID). SC enoxaparin 40 mg, BID), aspirin tablets (75 mg, OD), clopidogrel tablets (75 mg, OD), and atorvastatin tablets (40 mg, OD) were added. The patient was daily monitored for temperature, pulse,

blood pressure, GCS, ECG, and O2 saturation. The initial complete blood count (CBC); Hb was 12.6 g/dl, RBCs; 4.60*103/mm3, WBCs; 5.5*103/mm3 (Neutrophils; 81.6 %, Lymphocytes: 15.2%, and Mixed; 3.2%), and Platelets; 122*103/mm3. CRP was (98 g/dl). The troponin test was (1.5 ng/ml). SGPT was (32U/L), and SGOT was (24U/L), serum albumen was (2.93g/dl), total bilirubin was (1.8mg/dl), and direct bilirubin was (0.4mg/dl). Serum creatinine was (1.25mg/dl), blood urea was (59mg/dl), and serum uric acid was (6.4mg/dl). FBS was (113 mg/dl). INR was 1.59 and patient prothrombin time was 17.3 seconds. Serial ECG tracings were done. The initial ECG tracing was done on the initial ICU admission showing Left Bundle Branch Block (LBBB), ventricular pacing rhythm (ventricular spikes), pathological left axis deviation (LAD), high lateral (I and aVL), and anterior (V2-5) T-wave inversion. There is a progressive prolongation of each successive PR interval until the dropped beat (**Figure 1A**). The second ECG tracing done on the second day of the ICU admission showed LBBB, ventricular pacing rhythm (ventricular spikes), LAD, T-wave inversion in high lateral (I and aVL), and V2 leads. There is a progressive prolongation of each successive PR interval until the dropped beat (**Figure 1B**). The third ECG tracing was done on the seventh day of the ICU admission showing LBBB, ventricular pacing rhythm (ventricular spikes), LAD (light blue arrows), and high lateral (I and aVL) T-wave inversion. There is reversed progressive prolongation of each successive PR interval until the dropped beat (**Figure 1C**). The plain chest X-ray section was done on the day of the ICU presentation and within 3 days of CVA showing the position of the pacemaker, electrode, bilateral ground-glass opacities, and dysmorphic cardiac shadow (**Figure 2A**). The chest CT sections were done on the day of the ICU presentation and within 3 days of CVA showing bilateral halo sign in the left lung, right reversed halo sign, bilateral ground-glass opacities, and cardiomegaly (**Figure 2B**). The brain CT was done on the day of the ICU presentation and within 3 days of CVA showing evidence of left opacities indication of caudate nucleus infarction (**Figure 2C**). The abdominal ultrasound on the day of the ICU presentation showed chronic cholecystitis with multiple stones. Yasser's Wenckebach reversal phenomenon in a caudate nucleus stroke, COVID-19 pneumonia, diabetes, hypertension, calculous cholecystitis, and ventricular pacing were **the most probable diagnoses**. Within 7 days of the above management, the patient finally showed nearly clinical and ECG improvement. The patient was discharged within 7 days and continued on aspirin tablets (75 mg, OD), clopidogrel tablets (75 mg, OD), captopril tablets (25mg, OD), nitroglycerin retard capsules (2.5mg, BID), and long-acting insulin (70/30, BID). Future cardiac and neurological follow-up was advised.

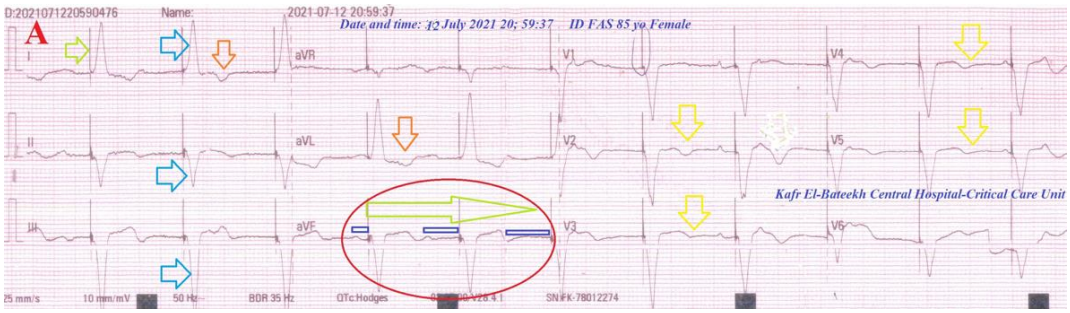

Figure 1-Serial ECG tracings; **A-** ECG tracing was done on the initial ICU admission and showed LBBB, ventricular pacing rhythm (VR; 69, ventricular spikes; lime arrow), LAD (light blue arrows), high lateral (I and aVL; orange arrows) and anterior (V2-5; golden arrows) T-wave inversion. There is a progressive prolongation of each successive PR interval until the dropped beat (red circle, dark blue rectangles, and lime arrow).

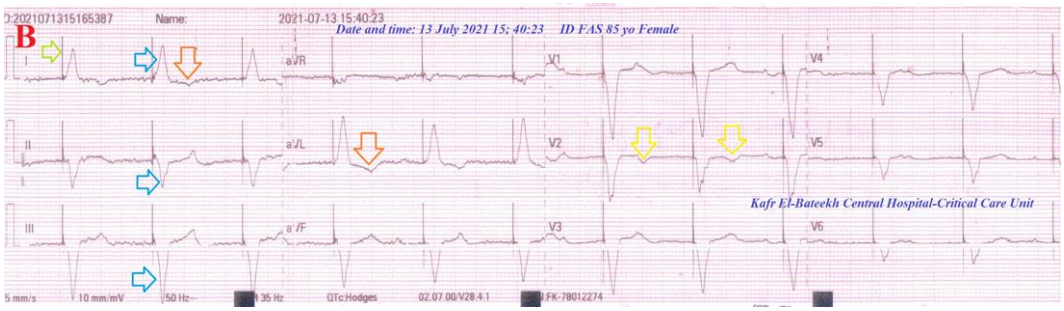

Figure 1-B- ECG tracing was done on the second day of the ICU admission and showed LBBB, ventricular pacing rhythm (VR; 72, ventricular spikes; lime arrow), LAD (light blue arrows), high lateral (I and aVL; orange arrows) and V2 (golden arrows) T-wave inversion. There is a progressive prolongation of each successive PR interval until the dropped beat.

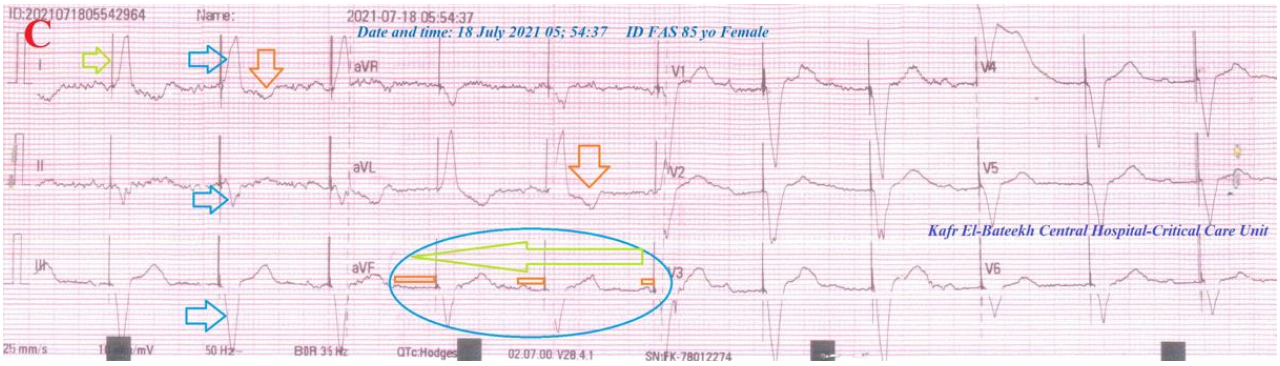

Figure 1-C- ECG tracing was done on the seventh day of the ICU admission and showed LBBB, ventricular pacing rhythm (VR; 89, ventricular spikes; lime arrow), LAD (light blue arrows), and high lateral (I and aVL; orange arrows) T-wave inversion. There is reversed progressive prolongation of each successive PR interval until the dropped beat (blue circle, orange rectangles, and lime arrow).

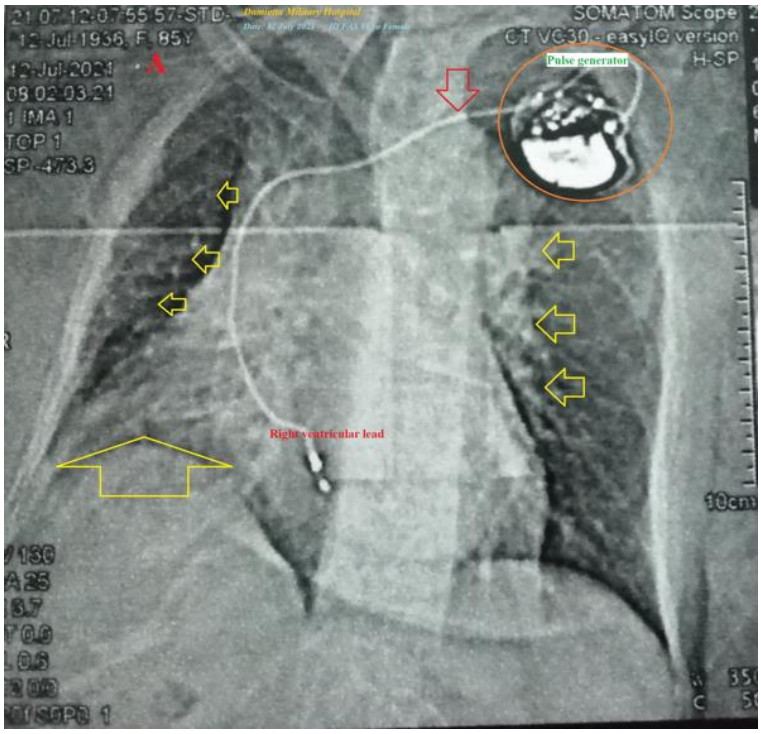

Figure 2: **A-Plain chest X-ray section of chest CT** was done on the day of the ICU presentation and within 3 days of CVA showed the position of the pacemaker (pulse generator; orange circle), electrode (RV lead, red arrow), bilateral ground-glass opacities (yellow arrows), and dysmorphic cardiac shadow.

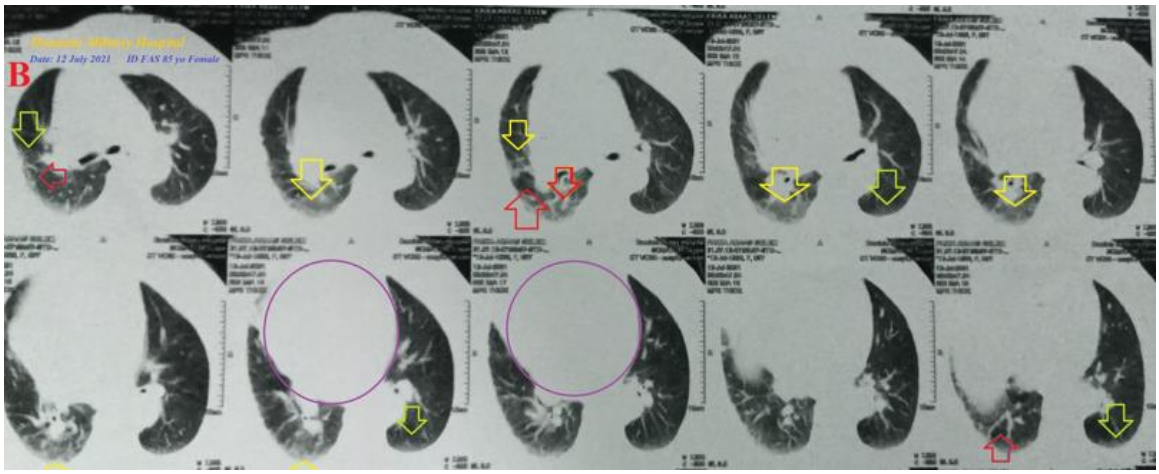

Figure 2-B-Chest CT sections was done on the day of the ICU presentation and within 3 days of CVA showed bilateral halo sign in the left lung (lime arrow), right reversed halo sign (red arrows), bilateral ground-glass opacities (yellow arrows), and cardiomegaly (purple circle).

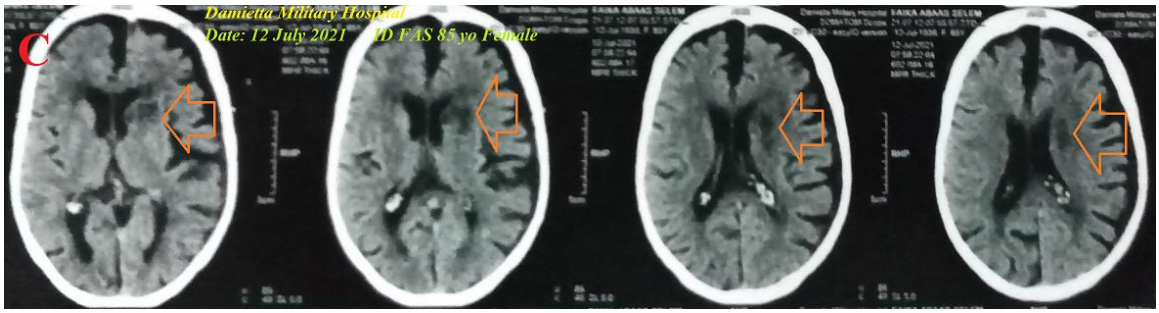

Figure 2-C- Brain CT was done on the day of the ICU presentation and within 3 days of CVA showed evidence of left opacities indication caudate nucleus infarction (golden arrows).

3. Discussion

• **Overview:**

• An elder housewife widow Egyptian female patient presented to the intensive care unit (ICU) with an acute right-side cerebrovascular accident, left-side deviation in the face, migraine, acute confusion, and COVID-19 pneumonia on top of diabetes, hypertension, chronic cholecystitis, Wenckebach phenomenon, and ventricle pacing and sensing (VVI).

• **The primary objective** for my case study was the presence of acute cerebrovascular accident and COVID-19 pneumonia on top of diabetes, hypertension, chronic cholecystitis, Wenckebach phenomenon, and ventricle pacing and sensing (VVI) in the admitted ICU patient.

• **The secondary objective** for my case study was the **question**; how did you manage the case?

• Interestingly, the presence of a positive history of contact with a confirmed COVID-19 case, bilateral ground-glass consolidation, and some laboratory COVID-19 suspicion on top of clinical COVID-19 presentation with fever, dry cough, generalized body aches, anorexia, and loss of smell will strengthen the higher suspicion of COVID-19 diagnosis.

• But the occurrence of acute cerebrovascular accident on top of COVID-19 pneumonia suggested that COVID-19 is an implicated cause.

• The presence of acute right-side cerebrovascular accident, left-side deviation in the face, migraine, acute confusion, and brain CT evidence of left opacities indication of caudate nucleus infarctions is a signal for the diagnosis of caudate nucleus infarction or left caudate nucleus stroke.

• The existence of initial high lateral and anterior (V2-5) T-wave inversion with elevated troponin I may indicate either myocardial injury Non-ST-segment elevation myocardial infarction/myocarditis (**Figure 1A**) or CN stroke (**Figure 2C**). Normalization of T-wave inversion in anterior leads strengthens the medication efficacy but there is still T-wave inversion in high lateral leads (**Figure 1C**) that potentiates either the pacing effect [**14**] or left ventricular strain.

• There is a progressive prolongation of each successive PR interval until the dropped beat from the right to the left (**Wenckebach phenomenon**) (**Figure 1A**). However, there is a reversal of the above progressive prolongation of each successive PR interval until a dropped beat from the left to the right (**Yasser's Wenckebach reversal phenomenon**).

• **Description and interpretation of Yasser's Wenckebach reversal phenomenon**:

• It is a reversal of the progressive prolongation of each successive PR interval until the dropped beat from the left to the right. It is a reversal form of the Wenckebach phenomenon.

• So, the reversal of characteristics of the Wenckebach phenomenon such as; highest PR-interval in a non-conducted P-wave, longest PR-interval immediately before dropped beat, shortest PR-interval immediately after dropped beat, relatively remains constant P-P interval, greatest PR interval duration is typically between the first and second beats of the cycle, and the progressively shortens RR interval with each beat of the cycle [**15,16**] may be applied for Yasser's Wenckebach reversal phenomenon.

• The **mechanisms** of Yasser's Wenckebach reversal phenomenon is unknown. Retrograde of reversible conduction block at the level of the AV node due to malfunctioning AV nodal cells tend to progressively fatigue until they fail to conduct an impulse is a suggested theory.

• The normalization of T-wave inversion in anterior leads is also a parallel with the reversal of the progressive prolongation of each successive PR-interval until a dropped beat from the left to the right. This will signify **Yasser's Wenckebach reversal phenomenon** (**Figure 1C**) as a signal for the above normalization.

• Spike ECG sign is the most probable electrocardiographic **differential diagnosis** for the current case study. However, there is no presence of small regular or irregular peaks between wide QRS complexes.

• I can't **compare** the current case with similar conditions. There are no similar or known cases with the same management for near comparison.

• The only limitation of the current study was the unavailability of long ECG strip tracing and echocardiography.

4. Conclusion and Recommendations

• Yasser's Wenckebach reversal phenomenon is a new cardiovascular discovery.

• A caudate nucleus stroke, COVID-19 pneumonia, calculous cholecystitis, and ventricular pacing are associated with the new phenomenon.

• The normalization of T-wave inversion in anterior leads is also parallel with the reversal of the progressive prolongation of each successive PR interval until a dropped beat from the left to the right.

• This will signify Yasser's Wenckebach reversal phenomenon as a signal for the above normalization.

• Future widening of relevant research will advised.

Conflicts of interest

• There are no conflicts of interest.

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