

**Case Report***Copyright © All rights are reserved by Yasser Mohammed Hassanain Elsayed*

# “A Case Report in Surgery, Infectious Diseases, Oncology, Chest, And Critical Care Medicine”

## “COVID Pancreatitis with Pneumonia; Strong Suspicion with Possible Drug Exacerbation and Associations”

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**Received Date:** January 06, 2022**Published Date:** February 02, 2022**Abstract**

**Rationale:** Acute pancreatitis is an unforeseeable and probably fatal disease that has remarkable morbidity and mortality. Acute pancreatitis is not particularly caused by SARS-CoV-2. Failure to diagnose drug-inducing pancreatitis can be serious. Nowadays, the correlation between COVID-19 and acute pancreatitis is still non-evidenced. Unfortunately, the animal studies of COVID-19 causing acute pancreatitis are defective. Patient

**Concerns:** An elderly, housewife, married, Egyptian female patient was presented with acute pancreatitis with mild COVID-19 pneumonia and chronic hypertension.

**Diagnosis:** Acute pancreatitis with mild COVID pneumonia in previously hypertensive patient with possible drug inducing and pancreatic carcinomas association.

**Interventions:** Contrast abdominal CT, abdominal MRI, non-contrast chest CT, electrocardiography, oxygenation, and echocardiography.

**Outcomes:** Good response and better outcomes despite the presence of numerous remarkable risk factors were the results.

**Lessons:** The association of acute pancreatitis with COVID-19 pneumonia and pancreatic carcinoma is a highly interesting combination. Captopril, hydrochlorothiazide, and furosemide may be inducing for acute pancreatitis but not for pancreatic carcinoma. An elder age, female sex, COVID-19 pneumonia, acute pancreatitis, elevated CA 19-9 and CEA markers, hypocalcemia, captopril, hydrochlorothiazide, and furosemide, and shock are constellation serious risk factors.

**Keywords:** COVID-19 pneumonia; Pancreatitis; Drug inducing pancreatitis; Pancreatic carcinomas; Drug exacerbation and associations

**Abbreviations**

ALP: Alkaline phosphatase

CA 19-9: Cancer antigen 19-9

CEA: Carcinoembryonic antigen

COVID-19: Coronavirus disease 2019

ECG: Electrocardiogram

GGT: Gamma-glutamyl transferase

ICU: Intensive care unit

O<sub>2</sub>: Oxygen

SGOT: Serum glutamic-oxaloacetic transaminase

SGPT: Serum glutamic-pyruvic transaminase

VR: Ventricular rate

## Introduction

The coronavirus disease 2019 (COVID-19) pandemic has currently affected 298,737,426 people and caused 5,484,896 deaths worldwide by January 6, 2020 [1]. Acute pancreatitis is a frequent disease, being the gastrointestinal (GI) disease most commonly indicating emergency hospitalization. Acute pancreatitis is an unforeseeable and probably fatal disease [2]. Pancreatitis is a serious disease with remarkable morbidity and mortality [3]. Acute pancreatitis is not particularly caused by SARS-CoV-2. Numerous causes are implicated but the idiopathic etiology is still present in 15–25% of cases [2]. Nowadays, the correlation between COVID-19 and acute pancreatitis is still non-evidenced. Unfortunately, the animal studies of COVID-19 causing acute pancreatitis are defective. It is so difficult to interpret the obtainable data that acute pancreatitis is a result of COVID-19 disease. The relationship between the current SARS-CoV-2 infection and the occurrence of acute pancreatitis is heterogeneous [4]. Some patients develop COVID-19 manifestations with abdominal pain at the onset of the infection, but the remaining has acute pancreatitis within a few days post-diagnosis of COVID-19 infection [4]. The drug-induced pancreatitis is mostly mild to moderate despite the severe fatal cases that may be present [3]. Mild cases of acute pancreatitis carry a mortality of less than 1%, but severe pancreatitis may reach as high as 30%. Medications are implicated in 0.1%-2% of acute pancreatitis incidents [3]. However, failure to diagnose drug-inducing pancreatitis can be critical [5]. Prevention of drug-induced pancreatitis requires knowledge updating of drugs for its strong correlation for the development of pancreatitis [5]. Controversy presents regarding the accurate mechanisms of drug-induced pancreatitis [5]. Angiotensin-converting enzyme inhibitors (ACEI), statins, oral contraceptives (OCP) or hormone replacement therapy (HRT), diuretics, highly active antiretroviral therapy (HAART), and hypoglycemic agents are common causes of pancreatitis [3]. Ranson's criteria are mainly used in inpatient monitoring. It mainly is used to estimate the role of operative treatment, weighted toward multi-systemic failure, SIRS, and vascular leak [6]. Ranson's criteria are often used to predict the severity and mortality of acute pancreatitis [6]. Five parameters are assessed on admission, and the other six are assessed at 48 hours post-admission. One point is given for each positive parameter for a maximum score of 11. The modified criteria have a max score of 10. Five parameters are assessed on admission and the other 5 at the 48-hour mark [6-8]. The criteria with 11 parameters are used to assess the severity of alcoholic pancreatitis. The 5 parameters on admission are age older than 55 years, WBC count greater than 16,000 cells/mm<sup>3</sup>, blood glucose (RBS) greater than 200 mg/dL (11 mmol/L), serum AST greater than 250 IU/L, and serum LDH greater than 350 IU/L. At 48 hours, the remaining 6 parameters are serum calcium less than 8.0 mg/dL (less than 2.0 mmol/L), hematocrit fall greater than 10%, PaO<sub>2</sub> less than 60 mmHg, BUN increased by 5 mg/dL or more (1.8 mmol/L or more) despite intravenous (IV) fluid hydration, base

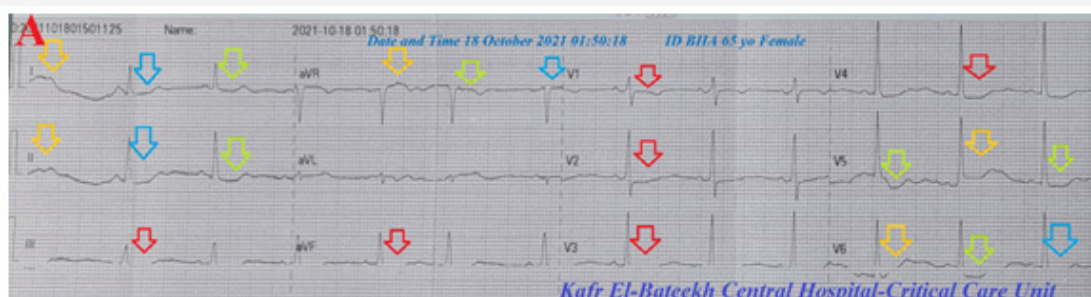
deficit greater than 4 mEq/L, and reservation of fluids greater than 6 L [9]. The modified Ranson criteria are commonly used to evaluate gallstone pancreatitis. The five parameters on admission are age older than 70 years, WBC greater than 18,000 cells/mm<sup>3</sup>, blood glucose greater than 220 mg/dL (greater than 12.2 mmol/L), serum AST greater than 250 IU/L, and serum LDH greater than 400 IU/L. At 48 hours, the remaining 5 parameters are serum Ca less than 8.0 mg/dL (less than 2.0 mmol/L), hematocrit (Hct) fall greater than 10%, BUN increased by 2 or more mg/dL (0.7 or more mmol/L) despite IV fluid hydration, base deficit greater than 5 mEq/L, and reservation of fluids greater than 4 L. Score Interpretation as; 0-2 points has mortality 0% to 3%, 3-4 points have mortality 15%, 5-6 points has mortality 40%, and 7-11 has mortality nearly 100% [9]. Management of drug-induced acute pancreatitis needs stoppage of the causative drug with using of supportive measures [3]. The prognosis is fundamentally based on the evolution to multi-systemic failure, secondary infection of pancreatic, and peripancreatic necrosis [2].

## Case presentation

A 65-year-old, housewife, married, Egyptian female patient was presented to the emergency department (ED) with fever, tachypnea, abdominal pain, and severe vomiting. She gave a recent history of palpitations, fatigue, dry cough, generalized body aches, anorexia, and loss of smell 2 days ago. The abdominal pain was epigastric knifelike pain in the central abdomen radiates towards the mid-central back and the pain worse on deep breathing. There is a history of chronic hypertension who continued on captopril/hydrochlorothiazide tablets (25/15mg, OD) and furosemide tablets (40mg, OD). There is a recent contact with a confirmed case of COVID-19 pneumonia 15 days ago. The patient denied a history of other relevant diseases, drugs, or other special habits. Informed consent was taken. Upon general physical examination, generally, the patient was tachypneic, restless, distressed, jaundiced, with a regular pulse rate of VR; 78 bpm, blood pressure (BP) of 90/60 mmHg, respiratory rate of 22 bpm, the temperature of 38 °C, and pulse oximeter of oxygen (O<sub>2</sub>) saturation of 91%. Tests for latent tetany were elicited. Local epigastric tenderness was elicited. Currently, the patient is admitted to the intensive care unit. She was initially managed with COVID-19 pneumonia with hypovolemic shock and suspected pancreatitis. Initially, the patient was treated with O<sub>2</sub> inhalation by O<sub>2</sub> cylinder (100%, by nasal cannula, 5L/min as needed). The patient was maintained treated with Ringer solution (500ml; TDS), normal saline 0.9% (500ml; TDS) cefotaxime; (1000 mg IV every 8hours), azithromycin tablets (500 mg, OD), oseltamivir capsules (75 mg, BID only for 5 days), and paracetamol (500 mg IV every 8 hours as needed). SC enoxaparin 80 mg, BID), aspirin tablet (75 mg, OD), clopidogrel tablets (75 mg, OD), hydrocortisone sodium succinate (100 mg IV every 12 hours), and metoclopramide slowly IV (10mg, as needed) were given. Maintenance therapy with IVI calcium gluconate ampoules

(10% with the rate; 0.5 mg/kg/hour over IV over 6 hours) was infused. The patient was daily monitored for temperature, pulse, blood pressure, and O<sub>2</sub> saturation. Ranson's criteria with CV line are used in the assessment of the patient's severity. The initial ECG tracing was done on the day of the presentation to the ICU showing NSR of VR; 77. There is ST-segment depression in both anterior (V1-4) and inferior leads (III and aVF). There is a Wavy triple sign (Yasser's sign) in I, aVR, and V6 leads and a Wavy double sign (Yasser's sign) in V5 lead (Figure 1A). The second ECG tracing was repeated within 30 seconds of the above tracing showing NSR of VR; 79. There is ST-segment depression in both anterior (V1-6) and inferior leads (III, III, and aVF). There is a Wavy triple sign (Yasser's sign) in I lead and a Wavy double sign (Yasser's sign) in aVR lead (Figure 1B). Chest CT without contrast was done on the day of the presentation to the ICU showing mild bilateral multiple ground-glass opacities (Figure 2A). Abdominal CT with contrast was done on the day of the presentation to the ICU showing filling defect opacities in the body of the pancreas with pancreatic calcifications. (Figure 2B). Laboratory workup was done during the third day of the presentation. The initial complete blood count (CBC); Hb was 11.9 g/dl, RBCs; 0.3\*10<sup>3</sup>/mm<sup>3</sup>, WBCs; 11\*10<sup>3</sup>/mm<sup>3</sup> (Neutrophils; 91.4 %, Lymphocytes: 5%, Monocytes; 3.5%, Eosinophils; 0.1% and Basophils 0%), Platelets; 184\*10<sup>3</sup>/mm<sup>3</sup>, and Hct; (40%). S. lipase was high (1536 U/L). S. amylase was high (608.9 U/L). Total

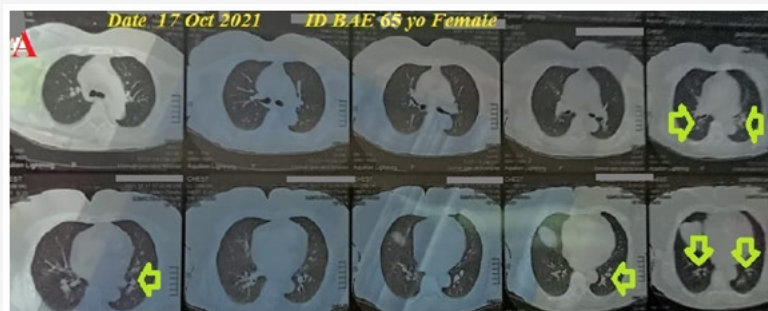
bilirubin was high (3.6 mg/dl). CA 19.9 was high (135U/ml). CEA was high (3.40 ng/ml). S. ferritin was high (398. ng/ml). D-dimer was high (0.774 ng/ml). CRP was high (129 g/dl). LDH was high (611U/L). SGPT was high (278 U/L), SGOT was high (315 U/L). Serum albumen was low (2.3 gm/dl). Serum creatinine was normal (1.2 mg/dl) and blood urea was normal (34 mg/dl). S triglycerides were high (182 mg/dl). ALP was high (365 U/L). GGT was high (274 U/L). ESR was high (first hour; 86mms and first hour; more than 100mms). HbA1C was normal (5.57 %). RBS was high (287 mg/dl). Plasma sodium was low (131mmol/L). Serum potassium was low (3.4 mmol/L). Ionized calcium was low (0.8 mmol/L) and total calcium was low (9.9 mg/dl). The troponin test was negative (0.1 U/L). MRI was done within 3 days of the above abdominal CT showing filling defect opacities in the body of the pancreas with pancreatic calcifications (Figure 2C). Echocardiography was done during the seventh day of the presentation with EF; 69 % showed no detected abnormalities. Acute pancreatitis with mild COVID pneumonia in previously hypertensive patient with possible drug inducing and pancreatic carcinomas association was the most probable diagnosis. The patient was dramatically and symptomatically improved within 7 days of admission. The patient was referred after clinical stabilization for further surgical and oncological intervention.



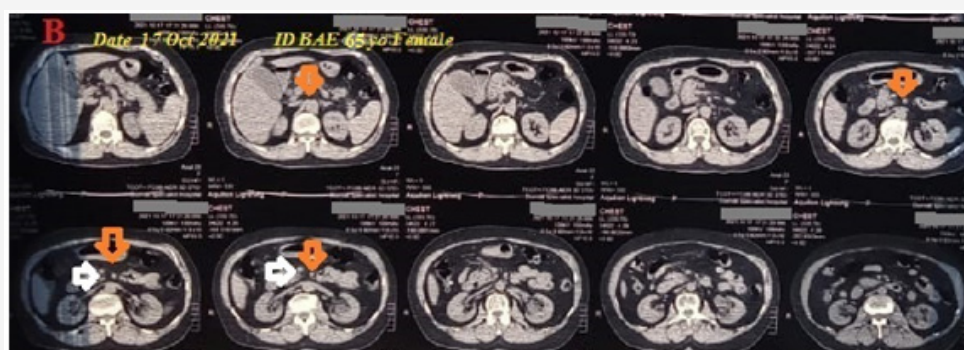
**Figure 1A:** ECG tracing was done on the day of the presentation to the ICU showing NSR of VR; 77. There is ST-segment depression in both anterior (V1-4; red arrows) and inferior leads (III and aVF; red arrows). There is a Wavy triple sign (Yasser's sign) in I, aVR, and V6 leads (orange, lime, and light blue arrows) and a Wavy double sign (Yasser's sign) in V5 lead (orange and lime arrows).



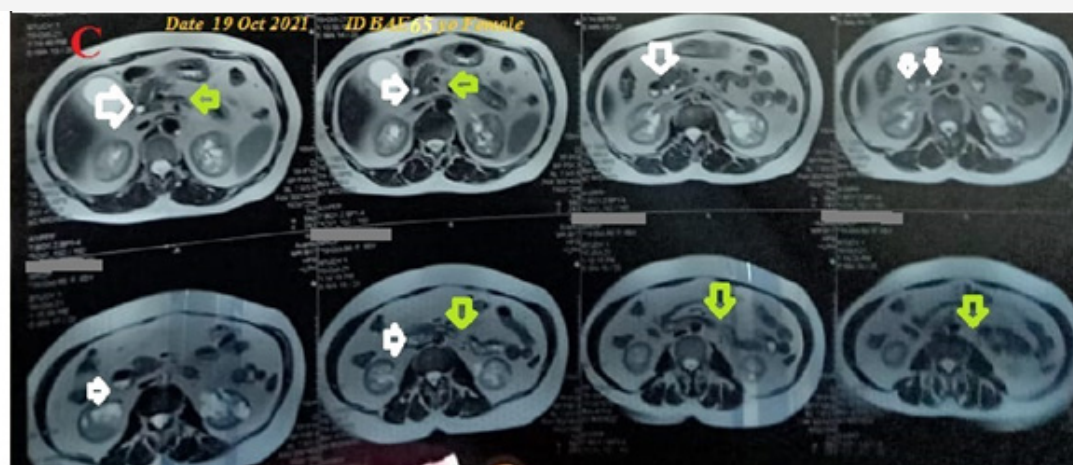
**Figure 1B:** ECG tracing was repeated within 30 seconds of the above tracing showing NSR of VR; 79. There is ST-segment depression in both anterior (V1-6; red arrows) and inferior leads (III, III, and aVF; red arrows). There is a Wavy triple sign (Yasser's sign) in I lead (lime, orange, and light blue arrows) and a Wavy double sign (Yasser's sign) in aVR lead (orange and lime arrows).



**Figure 2A:** Chest CT without contrast was done on the day of the presentation to the ICU showing mild bilateral multiple ground-glass opacities (lime arrows).



**Figure 2B:** Abdominal CT with contrast was done on the day of the presentation to the ICU showing filling defect opacities in the body of pancreas (orange arrows) with pancreatic calcifications (white arrows).



**Figure 2C:** MRI was done within 3 days of the above abdominal CT showing filling defect opacities in the body of pancreas (lime arrows) with pancreatic calcifications (white arrows).

## Discussion

A 65-year-old, housewife, married, Egyptian female patient was presented with acute pancreatitis with mild COVID-19 pneumonia and chronic hypertension. The primary objective for my case study was the presence of an elderly, housewife; married, Egyptian female patient who was admitted to the ICU with acute pancreatitis, hypovolemic shock, possible pancreatic carcinoma, mild COVID-19 pneumonia, and chronic hypertension. The secondary objective for

my case study was the question of; how did you manage the case at the ICU? There is an initial undoubtedly presentation of acute pancreatitis that confirmed with clinical symptoms, laboratory, and radiological workup. The severity of the case presentation with acute pancreatitis was assessed with Ranson's criteria (Score; 6) and its modification (Score; 5) with a CV line. The elevated Cancer antigen 19-9 (CA 19-9) tumor marker with very high ESR are guide for possible association of the pancreatic carcinoma [10].

Radiological filling defects in the body of the pancreas may be helpful for its primary detection. The elevated carcinoembryonic antigen (CEA) is reported in the colon and rectum, but it can be elevated with gastric, ovarian, and other cancers such as pancreatic carcinoma [11].

The elevated alkaline phosphatase (ALP) refers to the existence of the cholestatic liver disease. The marked elevations of gamma-glutamyl transferase (GGT) are seen in intra- or posthepatic biliary obstruction although its elevation is seen in all forms of liver disease. The elevated of both ALP, GGT, and total bilirubin indicates the presence of obstructive jaundice. Evidence of hypocalcemia in ECG with acute pancreatitis and COVID-19 pneumonia is reasonable. The changes in the ECG leads which are affected with both Wavy triple (Yasser's sign) and wavy double signs (Yasser's sign) of hypocalcemia in both ECG tracing is a signal for the Movable phenomenon of hypocalcemia (Yasser's phenomenon) [12]. Interestingly, the presence of the positive history of contact with a confirmed COVID-19 case, bilateral ground-glass consolidation, and laboratory COVID-19 suspicion on top of clinical COVID-19 presentation with fever, tachypnea, dry cough, generalized body aches, anorexia, and loss of smell will strengthen the higher suspicion of COVID-19 diagnosis. Captopril, hydrochlorothiazide, and furosemide are known inducing agents for acute pancreatitis [3]. An elder age female sex, COVID-19 pneumonia, acute pancreatitis, elevated CA 19-9 and CEA markers, hypocalcemia, captopril, hydrochlorothiazide, and furosemide, and shock are risk factors. Perforated peptic ulcer disease was the most probable differential diagnosis for the current case study. 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 MRCP and ERCP.

### Conclusion and Recommendations

The association of acute pancreatitis with COVID-19 pneumonia and pancreatic carcinoma is a highly interesting combination. Captopril, hydrochlorothiazide, and furosemide may be inducing for acute pancreatitis but not for pancreatic carcinoma. An elder age, female sex, COVID-19 pneumonia, acute pancreatitis, elevated CA 19-9 and CEA markers, hypocalcemia, captopril,

hydrochlorothiazide, and furosemide, and shock are constellation serious risk factors.

### Conflicts of interest

There are no conflicts of interest.

### Acknowledgment

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