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RESEARCH ARTICLE

MYOCARDIAL INFARCTION COMPLICATING ACUTE PANCREATITIS ON COVID19 PNEUMONIA

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Abstract

Acute pancreatitis is a frequent yet not well-known complication of sars-cov2 pneumonia, its occurrence increases the mortality rate so it is necessary to eliminate it in front of any digestive clinical symptoms. The complication of acute pancreatitis by myocardial infarction has already been described in literature but the pathophysiology is still unclear, several theories suggest that there may be a link between the presence of excessive inflammation, coronary spasm and or the presence of an embolic component, which can also be present in the context of sars-cov2 infection. Our work aims to increase knowledge of this pathology by describing the case of a North African female patient that was hospitalized during the second wave of the pandemic in our country, where delta was the most prevalent variant of covid19. She presented for a severe clinical form of acute respiratory distress syndrome secondary to viral pneumonia that was complicated by acute pancreatitis and myocardial infarction.

Introduction:-

Case Presentation:-

A 51 year old female patient with no particular medical history, most importantly not known to be diabetic nor hypertensive, no history of dyslipidemia and no family history of pancreatitis or heart disease. She was admitted to the intensive care unit for acute respiratory distress syndrome associated with upper abdominal pain and vomiting beginning 10 days before her hospitalization. On admission, the clinical examination found a conscious patient, stable with blood pressure at 100/70 mmHg, heart rate at 83 beats per minute, blood oxygen levels at 91% on ambient air, respiratory rate at 20 cycles/min, no signs of struggle breathing, no cyanosis, no lower limb edema, capillary blood glucose at 3.30g/L, urinary dipstick glycosuria without ketonuria. An electrocardiogram (EKG) was done immediately and it showed no abnormalities. The initial thoracic CT-scan objectified ground glass opacities in favor of viral pneumonia with lesions estimated at 30% of the lung volume, the diagnosis of the infection was confirmed by a positive PCR test. An abdominal ultrasound was normal showing no cholelithiasis, gallbladder and

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bile ducts free and of normal size. The blood tests showed signs of inflammation with an elevated white blood cells count at 12400/mm3, CRP levels at 91.2 mg/L, lipasemia at 793 IU/L, D-Dimer at 1482 ng/mL, procalcitonin at 0.07ng/mL, HbA1c 11.2%. Hepatic, renal, lipid and thyroid blood tests were normal. The fibrinogen level, prothrombin level and activated partial thromboplastin time have not been determined in our patient. We performed a thoraco-abdomino-pelvic scan, which objectified a worsening of the lung damage with lesions extending to more than 75% of the total lung volume without signs of pulmonary embolism (figure 1, 2). There was also a bilateral pleural effusion of low abundance (figure 3), the bile ducts were fine and free of lithiasis and there was severe pancreatitis according to the mCTSI classification (figure 4).

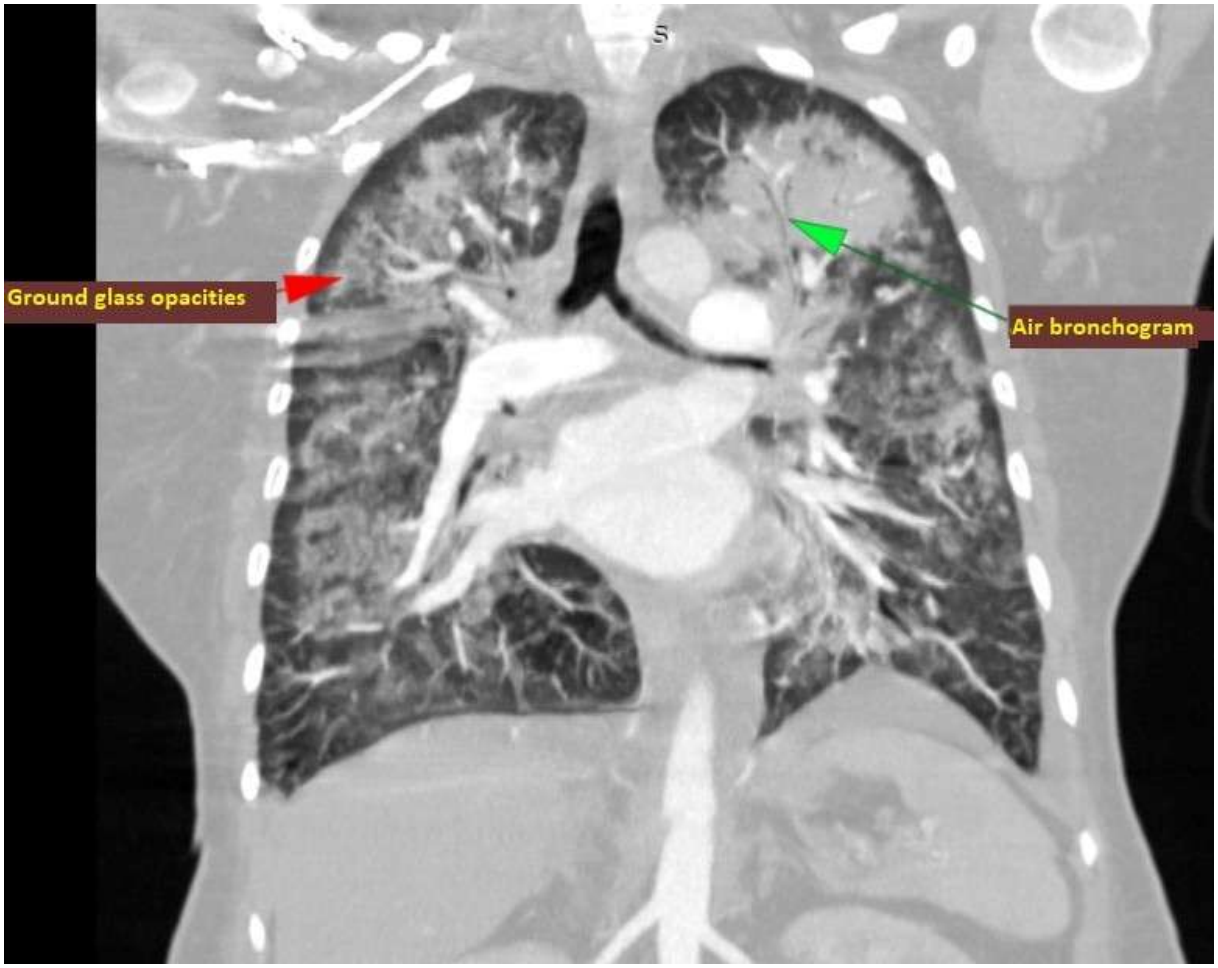

FIGURE 1: thoracic ct scan showing lung damage with ground glass opacities.

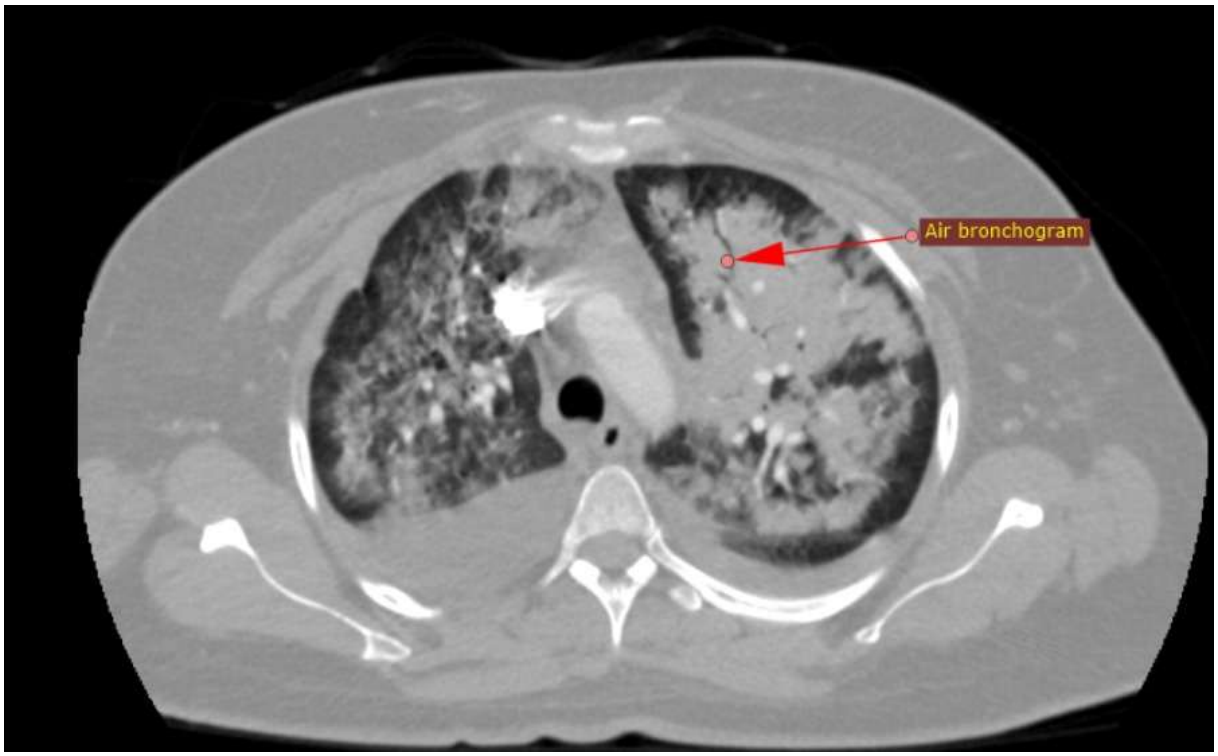

FIGURE 2: axial view of the lungs in the thoracic ct scan.

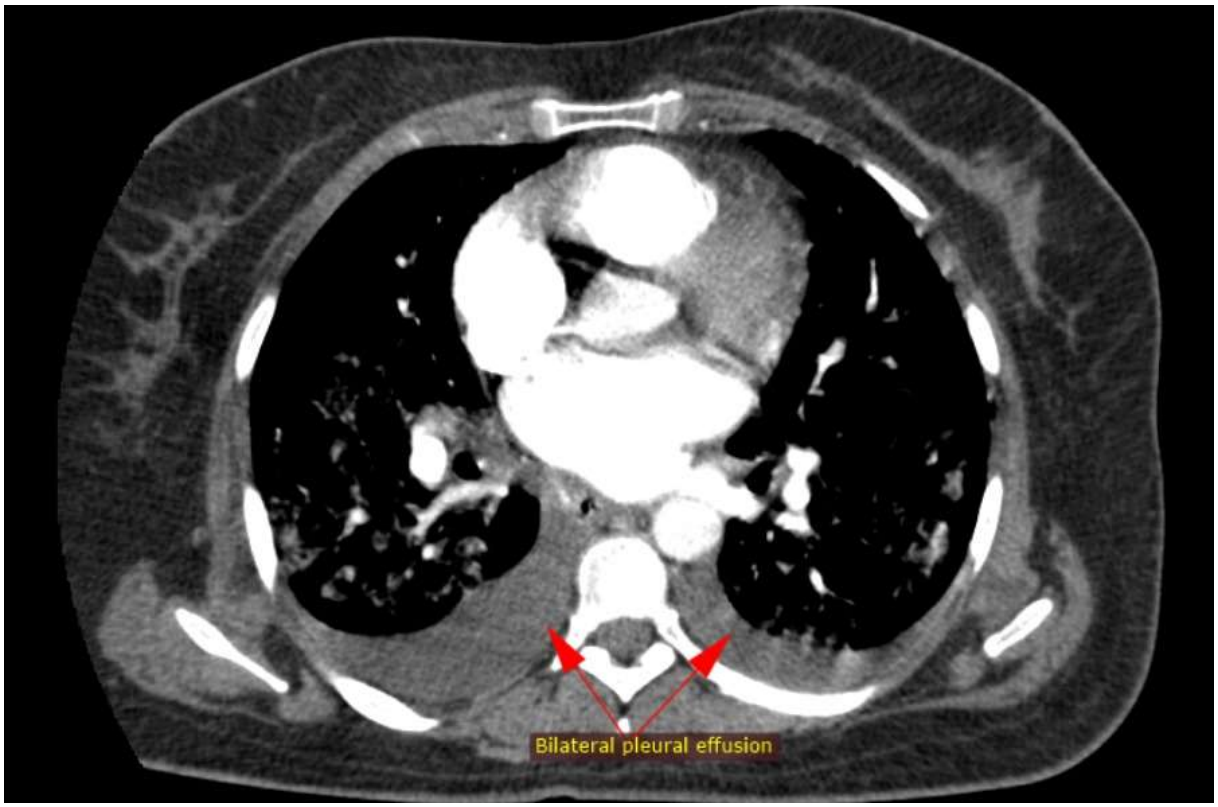

FIGURE 3: bilateral pleural effusion in the thoracic ct scan.

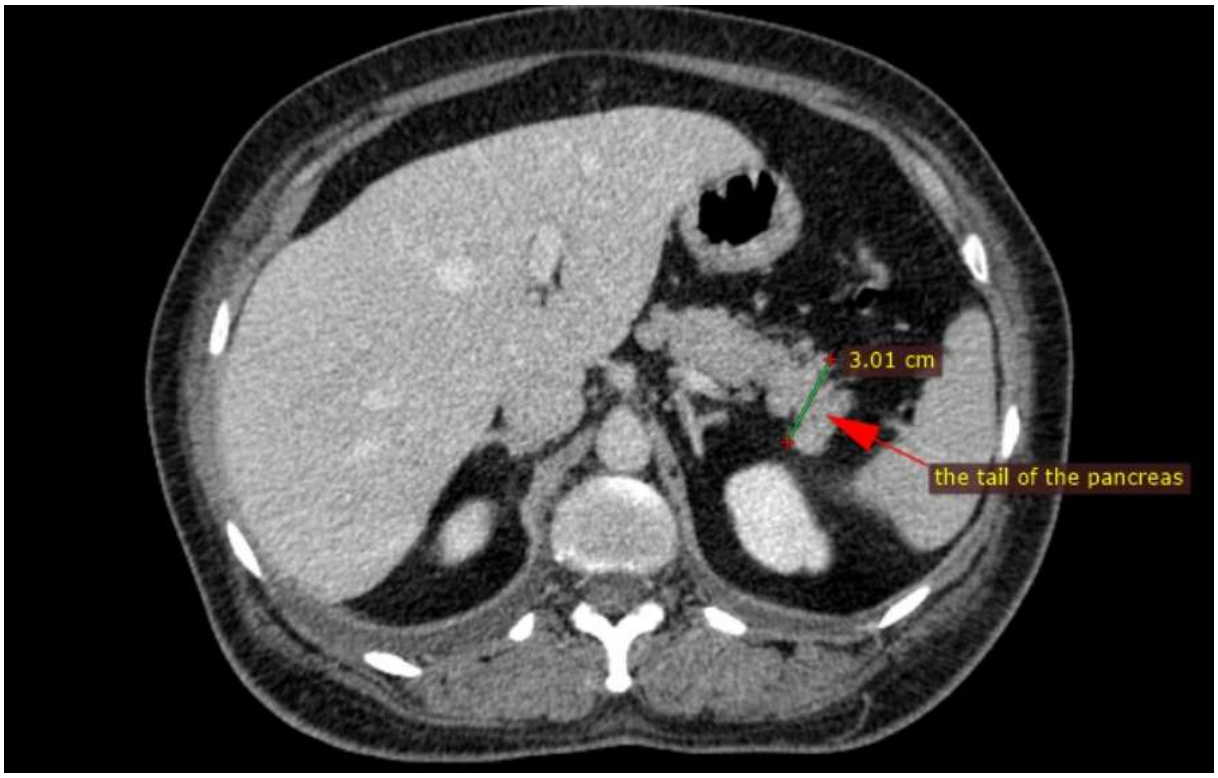

FIGURE 4: acute pancreatitis with enlarged tail of the pancreas in the abdominal ct scan.

Faced with the persistence of the upper abdominal pain, we preformed another EKG that showed an ST segment elevation with subsequent Q wave formation in precordial leads and right lateral leads with blood tests showing a high rate of troponin at 10.011 ng/mL. We performed a transthoracicechocardiography that showed signs of ischemic cardiomyopathy with Regional wall motion akinesiaof the apex and adjacent segments and a left ventricular ejection fraction at 35-40%. The patient developed global heart failure complicated by the acute pulmonary edema and loss of consciousness, then by the occurrence of cardiorespiratory arrest leading to the death of the patient after 14 days of hospitalization in the intensive care unit.

Discussion:-

Viral infections are known to be one of the many causes of acute pancreatitis yet we find it hard to diagnose and manage. We currently find more cases of acute pancreatitis in patients with covid19 pneumonia, but the causal link is still unclear. Expression of the angiotensin converting enzyme 2 receptor (ACE-2) in the pancreas is one of the factors involved in the pathophysiology of this type of pancreatitis [1]. Some studies suggest that the usage of pancreatic cells as a host for viral replication could be the cause of this pathology [2]. There may be a correlation between the occurrence of acute pancreatitis and the severity of lung involvement, some studies suggest that the rate of pancreatitis increased as the rate and severity of pneumonias did [3]. Whatever the origin of the pancreatitis, the inflammatory response of acinar cells can generalize and lead to systemic immune response syndrome (SIRS) [4]. High levels of pro-inflammatory cytokines aggravate pancreatic cell damage with an influx of activated leukocytes from the pancreas to the pulmonary, renal, vascular system and to the hepatic parenchyma. The cytokines interleukin (IL)-1β, tumor necrosis factor (TNF)- α, IL-6, IL-8, the platelet activating factor, and anti-inflammatory cytokines IL-10, TNF-soluble receptor and antagonist of the IL-1 receptor are also involved in the inflammatory response linked to acute pancreatitis and the occurrence of multiple organ failure [4].

The procalcitonin levels had a positive predictive value of 86% in acute pancreatitis and can be used as part of the detection of this pathology [5]. Several studies suggest that the binding of ACE-2 with sars-cov2 could be responsible for the concomitant development of acute pancreatitis and diabetes mellitus, apart from the already known risk factors [6] [7] [8] [9]. Our patient was not known to be diabetic but the diabetes was revealed during her hospitalization. She had no risk factors for acute pancreatitis and no familial history of this condition. The gallbladder and bile ducts were free and of normal size on ultrasound and abdominal CT angiography. Our patient

had biological signs of inflammation but procalcitonin levels were normal, as well as her renal, hepatic and lipid blood levels. Acute pancreatitis is often associated with elevated serum creatinine levels, significant inflammation and a high procalcitonin levels which are all linked to a longer hospitalization and higher mortality rates [3] [10]. The cardiovascular system can be affected at any stage of acute pancreatitis, with the occurrence of arrhythmias, disturbance of contractility and tone of peripheral vessels [4]. The exact pathophysiology of myocardial involvement is unclear but its correlation to SIRS is suggested. Electrical changes found in the electrocardiogram of these patients may include arrhythmias, conduction disorders, ST segment elevation as well as the Q waves of necrosis. These electrical changes can mimic the appearance of a myocardial infarction [4]. Rarely, acute pancreatitis can be directly complicated by myocardial infarction secondary to hypovolemia, hypercoagulability and coronary spasms [4] [11]. A case of myocardial ischemia secondary to acute pancreatitis has been described in literature, where a patient had acute pancreatitis and his EKG showed myocardial ischemia, a transthoracic ultrasound confirmed heart failure while an emergency coronary angiography revealed no signs of coronary artery obstruction [11]. A second case reports the occurrence of myocardial ischemia on acute necrotic pancreatitis with the development of heart failure which worsened the prognosis [12]. Our patient had an EKG and a transthoracic ultrasound showing myocardial ischemia, she also developed secondary heart failure that aggravated her clinical condition. Coronary angiography was not performed given the patient's unstable state and the absence of the right equipment platform locally.

Conclusions:-

Although we cannot directly relate the myocardial infarction in our patient to the acute viral pancreatitis, we sought it was important to discuss this case report with the aim of bringing more attention to the diagnosis of viral acute pancreatitis, a common yet underrated complication of covid19 pneumonia. The association of acute pancreatitis, myocardial infarction and covid19 infection is endowed with several diagnostic and therapeutic challenges necessitating a multidisciplinary approach, but the prognosis remains poor.

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