

# An observational study showing impact of COVID 19 pneumonia in causation of pancreatitis as long post COVID syndrome

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**Abstract-** Gastrointestinal symptoms are highly prevalent in COVID disease ranging from 17.6 % to 53%.The proposed mechanism for Gastrointestinal symptoms involves SARS-COV2 virus binding to the host epithelial cell's ACE 2 receptor,commonly found in GI tract epithelial cells.

After defeating corona virus to an extent there is now an interest in understanding post covid sequele .In this study we are tried understand if there was any impact of covid 19 infection on causation or prognosis of pancreatitis .

**Methodology :**

Clinical and laboratory data of all consecutive patients with a primary diagnosis of AP during past 6months with history of covid 19 infection was collected .Clinical and radiological grading was taken into consideration for finding severity.

**Result :** Out of 50 patients 35 patients had GI symptoms during there COVID infection phase while 15 patients had respiratory symptoms during COVID infection phase.Out of 50 patients 20 patients had severe covid 19 infection ,13 patients had moderate covid 19 infection and 18 patients had mild covid 19 infection.

Out of 50 patients 20 patient had severe pancreatitis ,13 had moderate pancreatitis and 18 patients had mild pancreatitis

Out of 20 patients with severe pancreatitis ,15 patients had GI symptoms during COVID infection phase and 5 patients had respiratory symptoms during COVID infection phase

**Conclusion:** Based on our study we conclude that more than severe covid 19infection mild covid 19infection with GI symptoms has a greater impact on the prognosis of patient with pancreatitis.

However there is still insufficient evidence showing that covid 19 can cause AP or negatively impact prognosis.Additional major studies are needed to clarify relationship between these two entities

**Keywords:** Pancreatitis, Pancreas, COVID-19, SARS-CoV-2

## Introduction

Gastrointestinal symptoms are highly prevalent in coronavirus disease ranging from 17.6 % to 53%.The proposed mechanism for GI symptoms involves SARS-COV2 virus binding to the host epithelial cell's ACE 2 receptor,commonly found in GI tract epithelial cells.

After defeating covid to an extent there is now an interest in understanding post covid sequele .In this study we are tried understand if there was any impact of covid 19 infection on causation or prognosis of pancreatitis .

The most common clinical manifestations of COVID-19 are respiratory, particularly fever and cough[2], but as cases have increased of widespread severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) across the globe, other symptoms and clinical scenarios have emerged. Gastrointestinal (GI) and hepatic involvement, among others, have been recognized and are mediated by the expression of angiotensin-converting enzyme 2 (ACE2) on the GI tract, the main receptor of SARS-CoV-2[3,4]. A recent systematic review and meta-analysis by Mao et al[5] showed that the estimated prevalence of digestive symptoms is 15%. Nausea, vomiting, diarrhea, and loss of appetite are the most frequent symptoms. Nineteen percent of patients present with liver injury, which may be more prevalent in fatal cases[5]. Furthermore, approximately 10% of COVID-19-positive patients may present with only GI symptoms. This clinical presentation may be associated with the delayed diagnosis of COVID-19 and a tendency of the disease to progress to more severe forms[5].

The expression of ACE2 in pancreatic cells (both exocrine glands and islets) renders the pancreas a potential target for SARS-CoV-2, but only recently has it received attention for its role in the COVID-19 clinical picture. Several case reports of pancreatic injury and acute pancreatitis (AP) caused by the novel coronavirus have been reported. About 1%-2% of non-severe and 17% of severe cases of COVID-19 exhibit pancreatic injury, which may have developed

before the patient’s admission[6]. However, there is still uncertainty about the physiopathological mechanisms involved and the precise etiology of pancreatic injury in the reported cases.

**Aim:**To find impact of covid 19 infection on causation and prognosis of pancreatitis

**Inclusion criteria :**

- 1)Patients with acute pancreatitis
- 2)Prior history of covid 19 infection

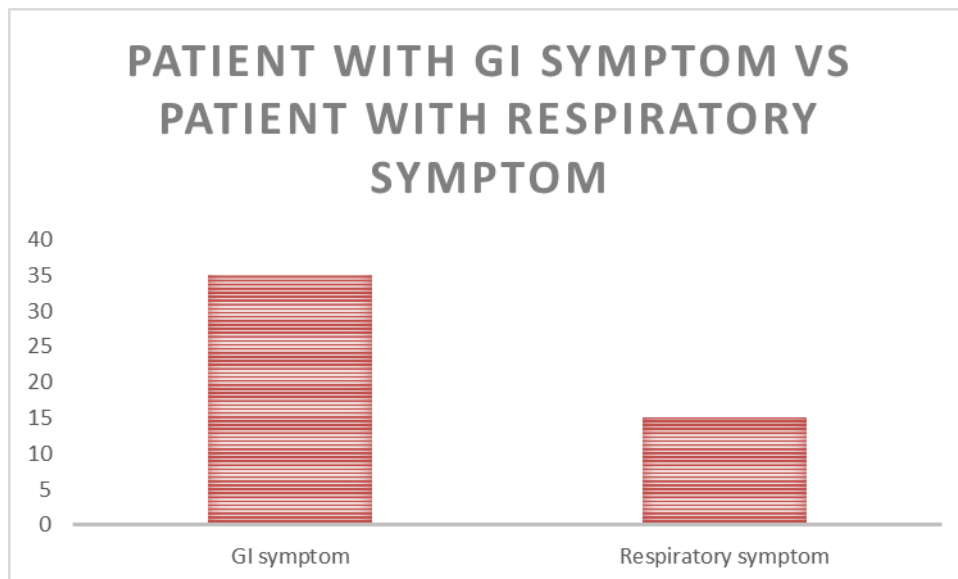
**Exclusion criteria:**

- Chronic pancreatitis patient
- Patient with on going covid 19 infection
- Immunocompromised patients

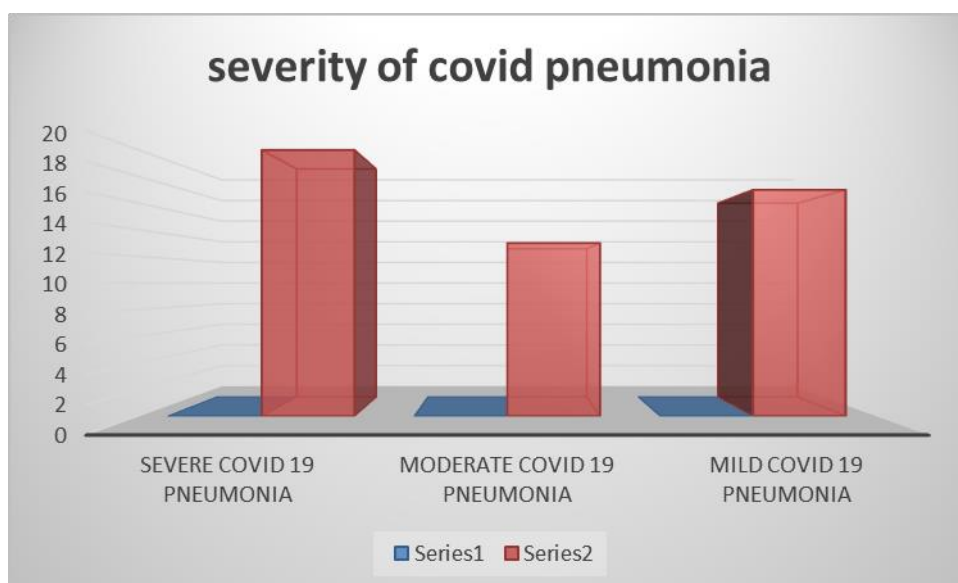
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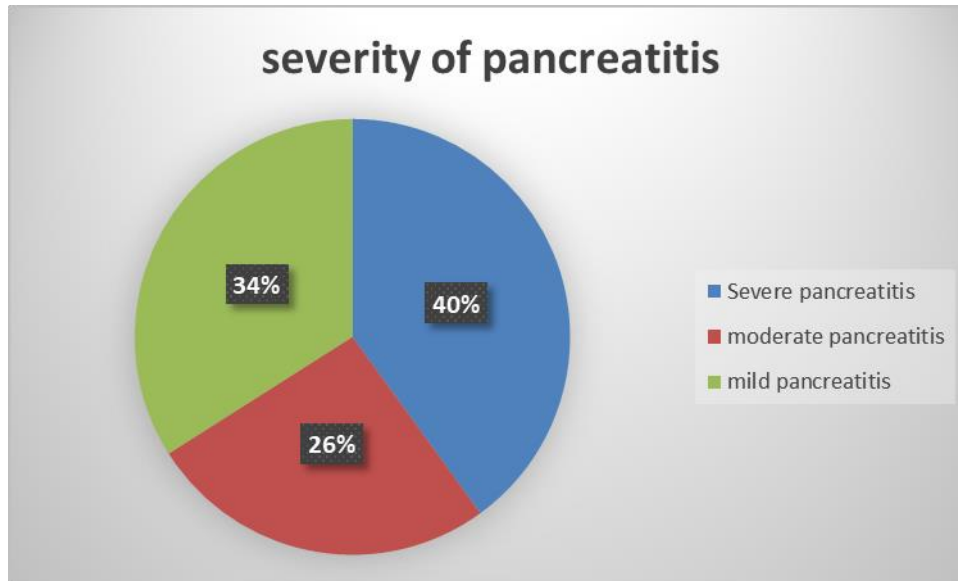
**Results:**



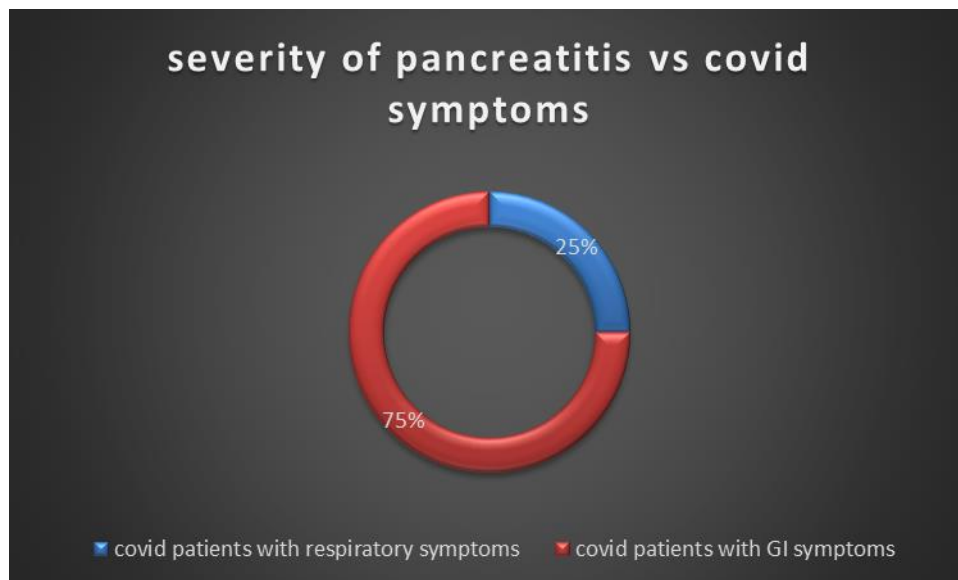
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Out of 50 patients 20 patient had severe pancreatitis ,13 had moderate pancreatitis and 18 patients had mild pancreatitis



Out of 20 patients with severe pancreatitis ,15 patients had GI symptoms during COVID infection phase and 5 patients had respiratory symptoms during COVID infection phase

### Discussion:

Pancreatic lesions, usually defined by serum amylase and/or lipase elevations, and cases of AP have been reported in COVID-19 patients. Autopsy studies in patients previously infected by SARS-CoV-2 identified areas of focal pancreatitis and pancreatic and/or peripancreatic necrosis and calcifications, but only two-thirds of these patients had exhibited symptoms suggestive of AP[8]. The diagnosis of AP, based on the modified Atlanta criteria, requires two of the following three features: Abdominal pain consistent with AP (acute onset of a persistent, severe, epigastric pain often radiating to the back), serum lipase activity (or amylase activity) at least three times greater than the upper limit of normal, and characteristic findings of AP on contrast-enhanced computed tomography (CECT) and less commonly magnetic resonance imaging or transabdominal ultrasonography[9]. This classification also divides AP into interstitial edematous pancreatitis and necrotizing pancreatitis and identifies local and systemic complications, which have a clear impact on disease progression, morbidity, and mortality[9]. All cases included in our study fulfilled the above criteria for the diagnosis of AP, but only a few included AP classification and local and systemic complications in the case report.

## Mechanisms of pancreatic injury by SARS-CoV-2

SARS-CoV-2 infection requires entry of the virus into the host cell. Metallopeptidase ACE2 has been identified as the cell receptor. Transmembrane serine protease 2 (TMPRSS2) facilitates viral entry at the plasma membrane surface. As such, co-expression of both ACE2 and TMPRSS2 is critical for successful SARS-CoV-2 infection

ACE2 is normally expressed in the pancreas. Liu et al[6] explored its expression and distribution, finding higher levels of ACE2 in the pancreas than in the lung and ACE2 expression in both exocrine glands and islets. Most studies have focused on ACE2 expression and there are few reports on TMPRSS2 expression in the pancreas. In one of these studies, Coate et al[11] found that ACE2 is mainly expressed in islet and exocrine tissue capillaries and some ductal cells, while TMPRSS2 is mainly expressed in ductal cells. However, ACE2 and TMPRSS2 are rarely co-expressed in pancreatic ducts. Pancreatic beta cells do not co-express ACE2 and TMPRSS2 and several authors have questioned the direct cytotoxic effects of SARS-CoV-2 on beta cells. It is still unknown whether SARS-CoV-2 directly and/or indirectly affects beta cell function[11]. However, COVID-19-associated glucose metabolism changes and diabetes appear to be multifactorial, resulting from systemic inflammation and metabolic changes in other organs, including the liver, muscle and adipose tissues, and are not exclusively the result of pancreatic damage. Further studies evaluating SARS-CoV-2 entry into beta cells and not only receptor expression are needed[11].

## Conclusion:

Based on our study we conclude that more than severe covid 19infection mild covid 19infection with GI symptoms has a greater impact on the prognosis of patient with pancreatitis.

However there is still insufficient evidence showing that covid 19 can cause AP or negatively impact prognosis. Additional major studies are needed to clarify relationship between these two entities

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