Pak. J. Chest Med. 2021;27(04)

Original Article

journal homepage: https:/www.pjcm.net/

Pakistan Journal of Chest Medicine

Official journal of Pakistan Chest Society

Pleural Effusion as a Predictor of Severity in Acute Pancreatitis

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Article History:

Received:	May 22, 2021
Revised:	July 20, 2021
Accepted:	Sep 12, 2021
Available Online:	Dec 02, 2021

Author Contributions:

MH IT conceived idea, MH IT drafted the study, IT S FU collected data, IT S FU statistical analysis and interpretation of data, IT MH critical review manuscript, all approved final version to be published.

Declaration of conflicting interests

The authors declare that there is no conflict to interest.

How to cite this article:

Hussain M, Tahir I, Shahbaz, Ullah F. Pleural Effusion as a Predictor of severity in Acute. Pak J Chest Med 2021; 27(4):262-266.

A B S T R A C T

Background: Acute pancreatitis frequently involves pulmonary radiographic abnormalities. Pulmonary infiltrates, pulmonary edema associated with adult respiratory distress syndrome (ARDS), and pleural effusion are examples of significant abnormalities. Elevation of the left hemidiaphragm and basilar atelectasis are two minor findings. Exudates in the effusions are frequently hemorrhagic. When pancreatitis subsides, pleural effusions may or may not become symptomatic and disappear. Pleural effusion has been suggested as a sign of severe pancreatitis. The present study was conducted with the aims to determine whether pleural effusion is a predictor of severity in acute pancreatitis and, if so, whether it is an independent predictor.

Methodology: This retrospective study was carried out at General /Thoracic Surgery Department of Hayatabad Medical Complex Peshawar. Total 70 cases of acute pancreatitis from Jan 2020 to Jan 2021, were included. Medical records were analyzed for evidence of pleural effusion by chest radiograph and severe acute pancreatitis by identification of pancreatic necrosis or organ system dysfunction. Data were analyzed to determine if identification of pleural effusion provided an early sign of severity.

Results: Mild acute pancreatitis cases were 60 (85.7%) while 10 (14.3%) severe acute pancreatitis cases were noted. In mild acute pancreatitis, gallstone was the most common etiology 21 (35%), followed by idiopathic 15 (25%), ERCP 13 (21.7%) and alcohol 11 (18.3%) respectively. Of 55 (78.6%) patients with mild pancreatitis who underwent chest radiography, 4 (7.2%) had pleural effusion, while 7 (70%) out of 10 patients had pleural effusion in severe acute pancreatitis (P <0.001).

Conclusion: Pleural effusion is strongly associated with severity in acute pancreatitis but provides independent information on severity in only a minority of cases.

Key words: Acute pancreatitis; Pleural effusion; Severe acute pancreatitis

Introduction

Acute pancreatitis frequently involves pulmonary radiographic abnormalities. In up to 55% of patients, abnormal chest radiography findings may be present.[1] Pulmonary infiltrates, pulmonary edema associated with adult respiratory distress syndrome (ARDS), and pleural effusion are examples of significant abnormalities. Elevation of the left hemidiaphragm and basilar atelectasis are two minor findings.[2,3] In 4-17% of patients with acute pancreatitis, pleural effusion develops.[4,5] Exudates in the effusions are frequently hemorrhagic. When pancreatitis subsides, pleural effusions may or may not become symptomatic and disappear. Pleural effusion has been suggested as a sign of severe pancreatitis.[6,7]

In this report, we have evaluated the association between pleural effusion on chest radiograph and severity of acute pancreatitis as defined by the Atlanta Symposium. In addition, we determined whether effusion is documented early enough to be an independent predictor of severity. The results of this research was the first to demonstrate a correlation between pleural effusion on chest radiograph and severity in accordance with the Atlanta criteria. The findings of our study confirm the link between pleural effusion and severity of acute pancreatitis. Pleural effusion on chest radiograph had a sensitivity of 84.2% and a specificity of 91.4% for severe disease, as manifested by pancreatic necrosis or organ failure. However, this report also shows that pleural effusion is rarely an early, independent predictor of severity.[8]

Methodology

This retrospective study was carried out at General Surgery/Thoracic Surgery Department of Hayatabad Medical Complex Peshawar between Jan 2020 and Jan 2021. The study covered all patients with acute pancreatitis. The detection of acute pancreatitis was done using two different techniques. First, all patients with the discharge diagnosis of acute pancreatitis were examined, and those whose serum levels of amylase or lipase were elevated to more than three times the upper limit of normal and whose clinical symptoms were compatible with acute pancreatitis were included in the study.

Second, a computer search was done to find all patients who had at least once during their hospital stay had an amylase or lipase increased to more than three times the upper limit of normal. Each patient's medical file was examined, and if the clinical course was compatible with acute pancreatitis, the patient was included. Pancreatitis following endoscopic retrograde cholangiopan creatography (ERCP) was defined as abdominal pain necessitating hospital admission for intravenous pain medication along with an increase in amylase or lipase to more than three times the upper limit of normal. Cases were excluded from the study if they met one of the following criteria: pleural effusion on chest radiograph preceding the development of acute pancreatitis, a comorbid medical condition that could lead to effusion (e.g., congestive heart failure), or acute pancreatitis in a moribund patient as a component of the terminal illness.

Cases that satisfied inclusion criteria were analyzed according to demographic data of age, sex, and etiology of acute pancreatitis. Etiology was identified from the medical record using the diagnosis of the clinicians caring for the patient. Records were reviewed to determine whether cases of pancreatitis represented the patient's first, second, or more than second episode of acute pancreatitis.

Cases were classified as mild or severe pancreatitis. Pancreatitis was considered severe in the presence of one of the following: necrosis as demonstrated by dynamic contrast-enhanced CT scan, surgical pathology, or autopsy or organ system dysfunction as reflected by hypotension (systolic blood pressure <90 mm Hg or dependence on intravenous pressors), hypoxemia (Pao <60 mm Hg breathing room air), renal insufficiency (elevation of serum creatinine above 2 mg/dl in the absence of preexisting renal insufficiency), or significant gastrointestinal bleeding. Reports of chest radiographs performed at any time during the hospitalization were reviewed for the presence of pleural effusions. In cases of acute pancreatitis transferred to our hospital, reports of chest radiographs performed at the referring hospital were reviewed for the presence of pleural effusion. Statistical analysis was done using SPSS 23.0 for windows 10. P value of ≤0.05 was considered statistically significant.

Table 1. Etiology and severity of study cases

Etiology	Mild AP (n=60)	Severe AP (n=10)
Gallstone	21 (35.0%)	3 (30.0%)
Idiopathic	15 (25.0%)	2 (20.0%)
ERCP	13 (21.7%)	2 (20.0%)
Alcohol	11 (18.3%)	3 (30.0%)

Table 2. Pleural effusion relationship with Acute Pancreatitis

Pleural effusion on chest radiography	Frequency	Percentage	P value
Mild Acute pancreatitis	4	7.2%	0.001
Severe Acute pancreatitis	7	70.0%	

Results

Total 70 cases of acute pancreatitis were included in the study. Age ranged between 20-70 years with a mean age was 45 years. There were 40 (57.2%) males and 30 (42.8%) females. Chest radiographs were performed in 55 (78.6%) cases. Mild acute pancreatitis cases were 60 (85.7%) while 10 (14.3%) severe acute pancreatitis cases were noted (Figure 1).

In mild acute pancreatitis, Gall stone was the most common etiology 21 (35%), followed by idiopathic 15 (25%), ERCP 13 (21.7%) and alcohol 11 (18.3%) respectively (Table 1).

Of 55 (78.6%) patients with mild pancreatitis who underwent chest radiography, 4 (7.2%) had pleural effusion, while 7 (70%) out of 10 patients had pleural effusion in severe acute pancreatitis (P <0.001) (Table 2).

Pleural effusion by anatomic location was noted in 8 (11.4%), effusions were bilateral in 4 (50%), left-sided in 3 (37.5%) and right-sided in 1 (12.5%). The distribution of effusions was similar in mild and severe pancreatitis: bilateral effusions comprised 60% of all effusions in severe disease and 52% of all effusions in mild disease (Figure 2).

In severe acute pancreatitis 4 mortalities were noted amongst which 3 (75%) had pleural effusion on chest radiograph, while the remaining 1 (25%) patient underwent no radiography after admission.

Discussion

Previous reports have noted a correlation between pleural effusion and severity in acute pancreatitis, as evidenced by pseudocysts, necrosis, and mortality.[9] A recent study linked pleural effusion detected by ultrasonography with severe pancreatitis in accordance with criteria outlined by the Atlanta Symposium.[10] However, none of these studies evaluated pleural effusion detected by chest radiography as a predictor of severity using the Atlanta criteria. Only 1 of 10 patients with severe pancreatitis who underwent admission chest radiography had a pleural effusion on admission. Of the 10 patients with severe pancreatitis who developed pleural effusion after admission, 6 had a negative chest radiograph within 2 days of the documentation of effusion. This finding demonstrates that the identification of pleural effusion after admission reflects the actual clinical course rather than delayed detection of an existing effusion. Pleural effusion demonstrated severity earlier than standard criteria in only 20% of cases.[11] Because the most severe bouts of acute pancreatitis generally occur during the patient's first or second episode. As a test for the presence of severe disease, pleural effusion had a sensitivity of 84.2% and a specificity of 91.4% (positive predictive value, 61.5% negative predictive value, 97.2%).[12] However, there was no significant correlation between the etiology of pancreatitis and the incidence of pleural effusion. We wanted to establish that the difference in rates of pleural effusion between mild and severe pancreatitis was not related to a difference in the number of previous episodes of acute pancreatitis. To evaluate the number of previous episodes

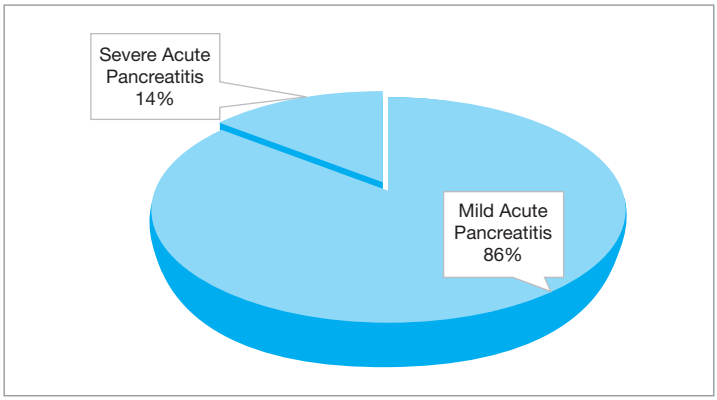

Figure 2: Pleural effusion relationship of severity to anatomic location

of acute pancreatitis as a possible confounder, we compared the rate of pleural effusion in mild and severe pancreatitis in the subset of patients suffering from their first or second bout of pancreatitis.

Balthazar EJ et al noted an association between pleural effusion on chest radiograph obtained within 6 days of admission and severe disease, as evidenced by Ranson's criteria and pseudocyst formation.[12] Li M et al showed a correlation between pleural effusion on admission chest radiograph and severe disease, as evidenced by pancreatic necrosis and death.[13] Gezer NS et al described an association between pleural effusion on computed tomography (CT) scan obtained within 72 h of admission and the extent of pancreatic necrosis, inflammatory changes, and mortality.[14] Kumar P et al noted a link between pleural effusion on ultrasound and severe disease as defined by the Atlanta Symposium (organ system dysfunction or local complications, i.e. necrosis, abscess, or pseudocyst).[15]

We aimed to establish that the difference in pancreatitis was not due solely to a difference in the number of prior episodes. To clarify this issue, we evaluated the subset of patients with fewer than two prior episodes of acute pancreatitis. We found that within this subset, 14 % of patients with mild pancreatitis and 86% of patients with severe pancreatitis had pleural effusion. The difference in rates of pleural effusion persisted after controlling for number of prior episodes. From these data, we conclude that the incidence of pleural effusion correlates with the severity of pancreatitis, not the number of prior episodes.

We wanted to determine whether pleural effusion was associated with the etiology of acute pancreatitis. Our study found no relationship between the etiology of acute pancreatitis and the development of pleural effusion. This finding corroborates those of Raghu MG et al but contradicts the findings of Liu D who found a higher incidence of pleural effusions in alcoholic than gallstone pancreatitis.[16,17] From our findings we conclude that effusion is related to severity but not etiology. We investigated whether there was a link between the severity of pancreatitis and the anatomic location of pleural effusions. This report found no relationship between severity of acute pancreatitis and anatomic distribution of pleural effusions. In both mild and severe disease, the majority of effusions was bilateral. Thus it appears that the site of pleural effusion offers no additional prognostic information. Our results corroborate those of Raghuwanshi S et al, who reported that 77% of pleural effusions were bilateral.[18] To summarize, pleural effusion is associated with severity in acute pancreatitis but predicts severity independently in only 20% of cases. Admission chest radiographs are rarely helpful. However, if a patient is noted to have pleural effusion without other manifestations of severity, the clinician should remain vigilant for the development of severe disease.[19]

Conclusion

Pleural effusion is a common chest finding in patients with acute pancreatitis. Pleural effusion volume quantified on chest CT examination positively associated with the duration of hospitalization and CRP levels. Pleural effusion volume can be a reliable radiologic bio-marker in the prediction of severity and clinical outcomes of acute pancreatitis.

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