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ORIGINAL ARTICLE

Current clinical and pathogenetic characteristics of patients with chronic pancreatitis depending on biological age and smoking

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ABSTRACT

Aim: To investigate the relationships between age, smoking status, inflammatory markers, and endotoxicosis in patients with chronic pancreatitis, focusing on C-reactive protein (CRP) and middle molecular peptides, specifically MMP254 and MMP280.

Materials and Methods: The study involved the examination of 108 patients diagnosed with chronic pancreatitis. These patients were categorized by age according to the World Health Organization (WHO) guidelines. Additionally, patients were stratified based on smoking status. Key biochemical markers were assessed, including fecal α-elastase, medium molecular weight peptides, and C-reactive protein levels. This approach allows for a comprehensive evaluation of how age and smoking may influence the course of chronic pancreatitis, while also considering the diagnostic value of these specific biomarkers in monitoring pancreatic function and inflammatory responses in these patients.

Results: A statistically significant impact of age on fecal α-elastase, C-reactive protein, and medium molecular peptides levels has been identified. Additionally, smoking has been shown to exacerbate pathological changes in these markers.

Conclusions: these findings underscore the necessity for individualized treatment approaches that consider age and smoking history, particularly in older patients. Future research should further explore the underlying mechanisms linking these variables to chronic pancreatitis, with an emphasis on the long-term effects of smoking cessation and interventions targeting inflammatory markers and endotoxicosis. This understanding is crucial for enhancing management strategies and improving the quality of life for patients suffering from chronic pancreatitis.

KEY WORDS: Chronic Pancreatitis, Fecal α-elastase, C-reactive Protein, Middle Molecular Peptides, Endotoxicosis, Smoking Status, Age

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INTRODUCTION

Chronic pancreatitis (CP) is a progressive inflammatory disorder characterized by irreversible damage to the pancreas, leading to exocrine and endocrine insufficiency [1-3]. The pathophysiology of CP involves repeated episodes of inflammation, resulting in fibrosis and functional impairment of the pancreas [2, 4-6]. Fecal α-elastase, middle molecular peptides (MMPs), and C-reactive protein (CRP) are biomarkers commonly used to assess pancreatic function, endotoxicosis, and inflammation in patients with chronic pancreatitis [7-9]. Investigating the variations in these biomarkers across different patient demographics, such as age and smoking status, provides valuable insights into the progression and systemic effects of chronic pancreatitis [10-12].

Several studies have highlighted the importance of age and lifestyle factors, such as smoking, in the development and progression of chronic pancreatitis [10, 13, 14]. Aging is associated with a decline in pancreatic exocrine function, and smoking has been shown to exacerbate inflammation and tissue damage in CP patients [4, 15-17]. The fecal α-elastase enzyme serves as an indicator of pancreatic exocrine function, while middle molecular peptides (MMPs) are markers of endotoxicosis and tissue damage [18]. Elevated CRP levels, a marker of systemic inflammation, are often observed in patients with chronic inflammatory diseases, including CP [16, 19, 20]. Therefore, evaluating the age- and smoking-related changes in these biomarkers is essential for understanding the disease progression and tailoring clinical management strategies.

AIM

This study aims to analyze the levels of fecal α-elastase, middle molecular peptides (MMP254 and MMP280), and CRP in patients with chronic pancreatitis across different age groups and smoking statuses.

MATERIALS AND METHODS

This study included 108 patients diagnosed with chronic pancreatitis (CP) and 30 healthy individuals as the control group. The diagnosis of CP was confirmed based on clinical symptoms, imaging studies (ultrasound, CT, or MRI), and laboratory tests following international guidelines. The study population was stratified into three age groups: up to 45 years, 46-65 years, and over 65 years. Additionally,

participants were categorized by smoking status into smokers and non-smokers. Informed consent was obtained from all participants, and the study was approved by the institutional ethics committee. The inclusion criteria for this study were as follows: patients aged 18 years and older with a confirmed diagnosis of chronic pancreatitis based on clinical symptoms, laboratory tests, and imaging studies (ultrasound, CT, or MRI). All participants provided written informed consent for participation and for the collection of biological samples (feces and blood) for analysis. Additionally, participants were required to have no acute exacerbations of chronic pancreatitis for at least 4 weeks prior to the study. The exclusion criteria included a history of acute pancreatitis within the last 6 months, significant gastrointestinal disorders (e.g., pancreatic cancer, inflammatory bowel disease) that could affect biomarker levels, severe comorbidities (e.g., cardiovascular, renal, or hepatic diseases) that may confound the study results, use of medications known to affect pancreatic function or inflammatory markers within 3 months prior to the study (e.g., corticosteroids, non-steroidal anti-inflammatory drugs), a history of alcohol abuse or substance dependence within the last year, and pregnancy or lactation.

Fecal samples were collected from all participants to measure α-elastase levels, which serve as a marker of exocrine pancreatic function. The enzyme-linked immunosorbent assay (ELISA) method was used to quantify fecal α-elastase concentrations, following the manufacturer's protocol. The results were expressed in micrograms per gram (μg/g) of feces.

To assess endotoxicosis, serum samples were analyzed for middle molecular peptides (MMP254 and MMP280), which are indicators of toxic metabolite accumulation. Blood samples were drawn from all participants after an overnight fast. MMP levels were determined using UV-spectrophotometry at wavelengths of 254 nm and 280 nm.

CRP levels, a marker of systemic inflammation, were measured using a high-sensitivity CRP assay (hs-CRP) in serum samples collected from each participant. The analysis was performed using an automated immunoassay analyzer. CRP concentrations were expressed in milligrams per liter (mg/L).

For the statistical analysis, continuous variables were expressed as mean ± standard deviation (SD). Parametric tests, including one-way ANOVA for comparisons among multiple groups, were used where data followed a normal distribution. Post-hoc analysis was performed using the Tukey test. For data not following a normal distribution, non-parametric tests, the Kruskal-Wallis test for multiple group comparisons, were applied. A p-value of less than 0.05 was considered statistically significant.

RESULTS

Among the etiological factors associated with the development of chronic pancreatitis, 22.3% of patients had biliary tract disease, 9.92% reported a history of acute pancreatitis, 36.5% engaged in alcohol consumption, 15.4% experienced metabolic disorders, and 5.8% were diagnosed with autoimmune diseases.

An analysis was conducted to compare fecal α-elastase concentrations across different age groups in patients with chronic pancreatitis (Table 1).

The analysis demonstrated statistically significant differences in fecal α-elastase levels across the comparison groups in patients with chronic pancreatitis ($p < 0.05$). In the control group, the concentration was the highest. Among participants aged up to 45 years, the value decreased, reflecting a 33.4% reduction compared to the control group. In the 46-65 years age group, the concentration further declined, representing a 49.2% decrease relative to the control. In the oldest group, aged over 66 years, the levels were the lowest, showing a significant 65.3% reduction compared to the control. These findings indicate a clear age-related decline.

An analysis was conducted to compare fecal α-elastase concentrations between smokers and non-smokers in patients with chronic pancreatitis (Table 2).

The analysis revealed statistically significant differences in fecal α-elastase levels between the comparison groups in patients with chronic pancreatitis ($p < 0.05$). The control group exhibited the highest concentration. In non-smokers, a reduction of 40.4% was observed compared to the control group. Smokers demonstrated slightly higher fecal α-elastase levels than non-smokers, though still showing a 35.6% decrease relative to the control. These results indicate a significant decrease in fecal α-elastase levels in both non-smokers and smokers, with the decline being somewhat less pronounced in the smoking group. The comparison between smokers and non-smokers highlights that smoking may have a moderate impact on fecal α-elastase levels, though both groups exhibit a notable reduction compared to the control group.

An analysis was conducted to compare the levels of middle molecular peptides, MMP254 and MMP280, across different age groups in patients with chronic pancreatitis (Table 3).

Table 1. Comparison of Fecal α-Elastase Concentrations by Age

Indicator	Group			
	Control (n=30)	under 45 years (n=34)	46-65 years (n=39)	over 65 years (n=35)
Fecal α-elastase, μg/g	278.57 ± 6.74	185.57 ± 5.71	141.57 ± 5.54	96.76 ± 4.81

** – statistically significant difference between comparison groups ($p < 0.05$)*

Table 2. Comparison of Fecal α-Elastase Concentrations in Smokers and Non-Smokers

Indicator	Group		
	Control (n=30)	Non-Smokers (n=67)	Smokers (n=41)
Fecal α-elastase, μg/g	278.57 ± 6.74	165.97 ± 7.61	179.52 ± 8.02

** – statistically significant difference between comparison groups (p < 0.05)*

Table 3. Comparison of MMP254 and MMP280 Measurements by Age

Indicator	Group			
	Control (n=30)	under 45 years (n=34)	46-65 years (n=39)	over 65 years (n=35)
MMP254, con. unit	254.45 ± 3,57	385.56 ± 5.87	428.65 ± 4.98	459.88 ± 5.07
MMP280, con. unit	137.49 ± 4,07	209.65 ± 3.45	228.96 ± 3.56	± 4.16

** – statistically significant difference between comparison groups (p < 0.05)*

Table 4. Comparison of CMP254 and CMP280 Measurements Based on Smoking Status

Indicator	Group		
	Control (n=30)	Non-Smokers (n=67)	Smokers (n=41)
MMP254, con. unit	254.45 ± 3,57	412.52 ± 8.56	450.72 ± 9.18
MMP280, con. unit	137.49 ± 4,07	209.45 ± 7.16	239.45 ± 8.56

** – statistically significant difference between comparison groups (p < 0.05)*

The analysis revealed statistically significant differences in MMP254 and MMP280 levels across the comparison groups in patients with chronic pancreatitis ($p < 0.05$). For MMP254, the control group exhibited the lowest values. In participants under 45 years old, MMP254 levels increased by 51.5% compared to the control group. In the 46-65 years age group, levels were 68.4% higher than the control, and the oldest group (over 65 years) showed the highest increase, with MMP254 levels 80.7% above the control.

A similar pattern was observed for MMP280 levels. The control group had the lowest values, while those under 45 years old demonstrated a 52.5% rise in MMP280 compared to the control. In the 46-65 years group, levels were 66.5% higher, and in the over 65 years group, MMP280 levels were 79.5% higher than in the control group. These results highlight a clear age-related increase in both MMP254 and MMP280 levels.

A study was performed to assess the levels of middle molecular peptides, MMP254 and MMP280, in relation to smoking status among patients with chronic pancreatitis (Table 4).

The analysis revealed statistically significant differences in MMP254 and MMP280 levels between the groups in patients with chronic pancreatitis ($p < 0.05$). MMP254 levels were the lowest in the control group. Among non-smokers, MMP254 levels increased by 62.1% compared to the control group, while smokers showed an even greater increase of 77.1%.

A similar pattern was observed for MMP280 levels. The control group had the lowest values, with non-smokers showing a 52.4% increase compared to the control. Smokers exhibited an even larger rise, with MMP280 levels 74.2% higher than in the control group. These results indicate significant increases in MMP254 and MMP280 levels in both non-smokers and smokers, with the highest levels in smokers.

A study was carried out to evaluate C-reactive protein (CRP) levels across various age groups in patients with chronic pancreatitis (Table 5).

The analysis demonstrated statistically significant differences in CRP levels across the comparison groups in patients with chronic pancreatitis ($p < 0.05$). In the control group, CRP levels were the lowest. Among participants aged up to 45 years, CRP levels increased by 170.1% compared to the control group. In the 46-65 years age group, CRP levels were 193.5% higher than the control. The oldest group, aged over 65 years, showed the highest CRP levels, with an increase of 222.7% relative to the control group. These findings indicate a significant age-related rise in CRP levels across the groups.

A study was performed to evaluate C-reactive protein (CRP) levels in relation to smoking status among patients with chronic pancreatitis (Table 6).

The analysis revealed statistically significant differences in CRP levels among the comparison groups in patients with chronic pancreatitis ($p < 0.05$). The control group exhibited

Table 5. Comparison of CRP Measurements by Age

Indicator	Group			
	Control (n=30)	under 45 years (n=34)	46-65 years (n=39)	over 65 years (n=35)
CRP, mg/L	1.54 ± 0.19	4.16 ± 0.28	4.52 ± 0.25	4.97 ± 0.22

** – statistically significant difference between comparison groups ($p < 0.05$)*

Table 6. Comparison of CRP Measurements Based on Smoking Status

Indicator	Group		
	Control (n=30)	Non-Smokers (n=67)	Smokers (n=41)
CRP, mg/L	1.54 ± 0.19	4.37 ± 0.87	4.81 ± 0.97

** – statistically significant difference between comparison groups ($p < 0.05$)*

the lowest levels. Non-smokers showed an increase in CRP levels of 183.4% compared to the control group. Smokers demonstrated even higher levels, with a 211.7% increase relative to the control. These findings suggest a significant rise in CRP levels in both non-smokers and smokers, with smokers displaying the highest levels overall.

DISCUSSION

The findings of this study highlight the significant differences in C-reactive protein (CRP) levels and middle molecular peptides (MMP254 and MMP280) among patients with chronic pancreatitis based on age and smoking status. The observed age-related increase in CRP levels suggests that older patients may experience more severe inflammatory processes associated with chronic pancreatitis. This is consistent with existing literature indicating that inflammation tends to escalate with age [21-23]. Several studies have reported a similar trend, where older adults exhibit elevated levels of pro-inflammatory cytokines, correlating with increased disease severity [24, 25]. These findings underscore the need for careful monitoring and tailored management strategies for elderly patients to mitigate the risks of exacerbated inflammation and related complications.

Furthermore, our results demonstrate that smoking significantly impacts inflammatory markers in patients with chronic pancreatitis. Smokers exhibited markedly higher CRP levels compared to non-smokers, indicating that smoking may exacerbate the inflammatory response associated with the disease. Previous research has found that smokers with chronic pancreatitis experience worse clinical outcomes and higher hospitalization rates [26, 27]. The detrimental effects of smoking on pancreatic health are well-documented, emphasizing the importance of smoking cessation as a crucial component of management for individuals with chronic pancreatitis [28].

The analysis of fecal α-elastase levels revealed a clear age-related decline in pancreatic exocrine function, which could contribute to the overall inflammatory status observed in patients. Similar correlations have been reported in the literature, indicating that reduced α-elastase levels are indicative of pancreatic insufficiency in older adults, often leading to malnutrition and further deterioration of health [29-31]. These relationships highlight the importance of early detection and intervention for pancreatic insufficiency in elderly patients, potentially improving their quality of life and reducing hospitalizations [32-34].

This study has several limitations that should be considered when interpreting the results. Firstly, the patient sample was limited to a specific geographic region, which may reduce the generalizability of the findings to other populations. Additionally, the study utilized a cross-sectional design, making it difficult to establish causal relationships between inflammatory markers, age, and smoking status. Potential variations in data collection methods may also affect the accuracy of the results; for instance, C-reactive protein and middle molecular peptide levels were measured based on single samples, which may not capture fluctuations over time. Moreover, the study did not account for potential confounding factors such as comorbidities, dietary habits, and levels of physical activity, which could also influence inflammatory marker levels. Finally, smoking data were self-reported, potentially introducing bias as participants may underestimate or overestimate their smoking habits. These limitations highlight the need for further research with larger and more diverse samples, as well as the inclusion of additional variables to provide a clearer understanding of the impact of age and smoking on chronic pancreatitis.

CONCLUSIONS

In conclusion, this study highlights significant associations between age, smoking status, and levels of inflammatory markers in patients with chronic pancreatitis. The results indicate a clear age-related increase in C-reactive protein (CRP) levels, suggesting that older individuals may experience

heightened inflammatory responses that could contribute to disease progression. Additionally, the study demonstrates that smoking significantly elevates CRP levels, indicating a potential exacerbation of inflammatory processes among smokers with chronic pancreatitis. The analysis of middle molecular peptides, MMP254 and MMP280, further supports the notion that both age and smoking status are critical factors influencing pancreatic function and inflammatory responses. An important aspect to note is that middle molecular peptides may serve as indicators of endotoxicosis, which can arise from a compromised intestinal barrier function. This increases the risk of systemic inflammation, which may, in turn, worsen the condition of patients with chronic pancreatitis. These findings underscore the necessity for individualized treatment approaches that consider these variables, particularly in older patients and those with a history of smoking. Future research should aim to explore the underlying mechanisms driving these associations, as well as the long-term effects of smoking cessation and age-related interventions on inflammatory markers, endotoxicosis, and middle molecular peptides. Deepening our understanding of these relationships may assist healthcare providers in enhancing treatment strategies and improving the quality of life for patients suffering from chronic pancreatitis.

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CONFLICT OF INTEREST

The Authors declare no conflict of interest

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