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Clinical Characteristics and Prognostic Analysis of Hepatitis Complicated with Acute Pancreatitis

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Abstract: *Objective:* To explore the clinical characteristics and prognostic risk factors of patients with hepatitis complicated by acute pancreatitis, aiming to provide insights for early clinical intervention. *Methods:* Clinical data from patients diagnosed with hepatitis complicated by acute pancreatitis and acute pancreatitis alone, admitted to our hospital from January 2017 to December 2023, were collected. General information, symptoms, laboratory results, imaging findings, and prognostic outcomes were analyzed and compared between the two groups. Statistical analyses included *t*-tests, Mann-Whitney U tests, χ^2 tests, univariate regression, and multivariate binary logistic regression to identify independent prognostic risk factors. *Results:* A total of 109 patients were included: 53 with hepatitis complicated by acute pancreatitis and 56 with acute pancreatitis alone. The hepatitis-complicated group had significantly longer hospital stays and lower levels of blood amylase, lipase, PTA%, and PLT, while TBil was higher compared to the acute pancreatitis group ($P < 0.05$). The positive rates of ultrasound, CT, and MRI in detecting complications showed no significant differences between the two groups. Among hepatitis-complicated cases, viral hepatitis was the most common cause (52.8%), and liver failure was the most common clinical type (49.1%). Univariate analysis identified factors such as liver failure, NEUT%, and REC as risk factors for poor prognosis. Multivariate logistic regression showed that liver failure, NEUT%, and REC were independent prognostic risk factors ($P < 0.05$). *Conclusion:* Hepatitis can complicate acute pancreatitis, with viral hepatitis and liver failure being the most common. Symptoms are non-specific, often including fatigue and digestive discomfort. Early diagnostic tests, especially abdominal imaging, are essential for accurate diagnosis. Prognosis is influenced by the degree of liver damage, with liver failure, NEUT%, and REC being key independent risk factors.

Keywords: Hepatitis; Acute pancreatitis; Clinical characteristics; Prognosis; Risk factors

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1. Introduction

Liver inflammation refers to inflammatory changes in the liver caused by viral infections, drugs, metabolic

abnormalities, and other damages. It can be classified into viral hepatitis, autoimmune liver disease, drug-induced liver disease, alcoholic liver disease, non-alcoholic fatty liver disease, and various clinical disease stages that result from their occurrence and progression, such as cirrhosis and liver failure [1]. Hepatitis is prevalent worldwide, and China is a major country for hepatitis cases. In 1944, Linsey reported that pancreatitis is a complication of hepatitis. Acute pancreatitis (AP) is an inflammatory response caused by the activation of pancreatic enzymes in acinar cells due to various stimuli, leading to the release of inflammatory factors and a local-to-systemic inflammatory reaction. The liver is often the most vulnerable and the first organ to be damaged in acute pancreatitis. Between 15% and 60% of patients with acute pancreatitis will develop liver injury. The global incidence of acute pancreatitis is 30–40 cases per 100,000 people per year, with a mortality rate of 1%–5% [2]. Among the fatal cases, 83% are attributed to liver failure [3]. From 1961 to 2016, the annual incidence rate of acute pancreatitis in countries such as the U.S. and Europe has increased by 2.77% to 3.67% [4]. Hepatitis and pancreatitis are both major causes of hospitalization for liver and gastrointestinal-related diseases, with considerable morbidity and mortality, contributing significantly to the medical and economic burden on society. Studies have shown that hepatitis viral infections are an independent predictor of poor outcomes in hospitalized AP patients, including higher mortality rates, longer hospital stays, and life-threatening complications [5]. Other studies have found that NAFLD can worsen the severity of AP, with the severity of AP increasing as NAFLD worsens [6]. When hepatitis is complicated by acute pancreatitis, AP can cause chronic liver disease to progress to ACLF [7]. During diagnosis and treatment, the clinical manifestations of hepatitis itself often make it difficult to detect the symptoms and signs of acute pancreatitis, which can affect the prognosis of patients with hepatitis complicated by acute pancreatitis. Most previous studies on hepatitis complicated by pancreatitis are case reports [8-10], and relevant research is limited. This study analyzes and compares the etiology, clinical types, symptoms, signs, imaging findings, treatment, and prognosis of patients with hepatitis complicated by acute pancreatitis and acute pancreatitis alone, exploring its pathogenesis and summarizing the factors affecting prognosis, providing a basis for clinical diagnosis, treatment, and monitoring of the disease.

2. Materials and methods

2.1. Study subjects

Clinical data were collected from patients diagnosed with acute pancreatitis at Chengdu Public Health Clinical Medical Center from January 2017 to December 2023. Patients were included according to the diagnostic criteria for various types of hepatitis combined with acute pancreatitis and those diagnosed with acute pancreatitis alone. Cirrhosis and liver failure were diagnosed according to the 2023 “Chinese Clinical Diagnosis and Treatment Consensus on Liver Cirrhosis” and the 2018 revised “Guidelines for the Diagnosis and Treatment of Liver Failure” [11,12]. Drug-induced hepatitis was diagnosed based on the 2023 “Chinese Guidelines for the Diagnosis and Treatment of Drug-Induced Liver Injury” [13]. Non-alcoholic fatty liver disease (NAFLD) was diagnosed based on the 2018 “Guidelines for the Prevention and Treatment of Non-Alcoholic Fatty Liver Disease (2018 Update)” [14].

2.2. Diagnosis and inclusion criteria

(1) Acute pancreatitis clinical diagnostic criteria [15]: Acute pancreatitis is diagnosed when two of the

following three criteria are met: (a) persistent and typical upper abdominal pain radiating to the back; (b) serum amylase and/or lipase levels exceeding three times the normal upper limit; (c) characteristic imaging findings of AP.

(2) Inclusion criteria for hepatitis combined with acute pancreatitis patients: (a) Discharged diagnosis of any type of hepatitis; (b) Discharged diagnosis of acute pancreatitis; (c) No clear common causes of pancreatitis at admission; (d) Complete clinical and laboratory data.

(3) Inclusion criteria for acute pancreatitis patients: (a) Discharged diagnosis of acute pancreatitis; (b) No associated hepatitis, cirrhosis, liver failure, etc.; (c) Presence of clinical manifestations or test results related to pancreatitis at admission; (d) Complete clinical and laboratory data.

2.3. Data collection

(1) General information: Age, gender, type and severity of hepatitis (presence of cirrhosis or liver failure), vital signs, and length of hospital stay.

(2) Laboratory test indicators: Blood amylase, blood lipase, triglycerides (TG), total bilirubin (TBil), white blood cell (WBC) count, neutrophil percentage (NEUT%), C-reactive protein (CRP) levels, PTA%, ALT, AST, hemoglobin (HB), red blood cell (RBC) count, platelet (PLT) count, and viral load and genotype for viral hepatitis.

(3) Imaging results: Ultrasound, CT, MRI findings.

(4) Treatment: Medications and prognosis.

2.4. Statistical analysis

The normality of continuous variables was first tested using the Kolmogorov-Smirnov test. For normally distributed continuous variables, data were expressed as mean ± standard deviation (SD) and compared using the independent two-sample *t*-test. For non-normally distributed variables, data were expressed as median (interquartile range) M (P25–P75) and compared using the Wilcoxon rank-sum test. Categorical variables were expressed as counts and percentages [*n* (%)], and compared using the chi-squared (χ^2) test. Variables with statistically significant differences in univariate analysis were included in a multivariate binary logistic regression model. All statistical analyses were performed using SPSS 26.0, and a P-value < 0.05 was considered statistically significant.

3. Results

3.1. Etiology and clinical type distribution characteristics

Among the 53 patients with hepatitis complicated by acute pancreatitis, 29 (54.7%) had viral hepatitis (see **Table 1** for the distribution and outcomes of various types of hepatitis). Among the 56 patients with acute pancreatitis, the causes were gallstones in 19 cases (33.9%), alcohol consumption in 4 cases (7.1%), hyperlipidemia in 15 cases (26.8%), and other or unknown causes in 18 cases. **Table 2** shows the distribution and outcomes of various clinical types.

Table 1. Etiology and prognosis of hepatitis complicated with pancreatitis

Etiology		Number of cases	Prognosis	
			Improved	Discharged or death
Viral hepatitis	HAV	2	1	1
	HBV	23	16	7
	HCV	3	3	0
	HBV + HCV	1	1	0
Fatty liver		13	10	3
Drug-induced hepatitis		4	4	0
Unknown cause		7	6	1

Table 2. Clinical types and outcomes of hepatitis complicated with pancreatitis

Etiology		Number of cases	Prognosis	
			Improved	Discharged or death
Acute hepatitis		7	7	0
Chronic hepatitis	Moderate hepatitis	4	4	0
	Severe hepatitis	13	12	1
Cirrhosis		3	3	0
Liver failure	Acute	2	1	1
	Subacute	4	2	2
	Chronic acute	13	6	7
	Chronic	8	7	1

3.2. Comparison of clinical characteristics between the two groups

3.2.1. General information

A total of 109 patients were included in this study. Among them, 53 had hepatitis complicated by acute pancreatitis, with an average age of 47.62 ± 14.058 years. There were 37 males (69.8%) and 16 females (30.2%), with a male-to-female ratio of 2.31:1. The remaining 56 patients had acute pancreatitis, with an average age of 52.57 ± 15.682 years. There were 35 males (60.3%) and 21 females (36.2%). There was no significant difference in age and gender between the two groups. The mean hospitalization time in the hepatitis and pancreatitis group was significantly longer than that in the pancreatitis-only group ($P < 0.05$).

3.2.2. Clinical manifestations

Among the 53 patients with hepatitis complicated by acute pancreatitis, 48 experienced abdominal pain, 16 had abdominal bloating or discomfort, 14 had nausea and/or vomiting, and 5 had a fever (1 with a peak of 38.9°C, 1 with 38.7°C, the others had a low-grade fever). Three patients had diarrhea, 22 had fatigue, poor appetite, and aversion to oily foods, and 2 had acid reflux or heartburn. Two patients had no abdominal pain, diarrhea, nausea, vomiting, or fever, and were diagnosed only by routine imaging and blood tests for lipase and amylase

levels. Among the physical signs, 7 patients had muscle rigidity, 35 had abdominal tenderness, and 9 had rebound tenderness. 18 patients had no obvious abdominal signs, and 2 had neither symptoms nor signs of acute pancreatitis.

Among the 56 patients with acute pancreatitis, only 2 had no abdominal pain. 22 patients experienced abdominal bloating or discomfort, 24 had nausea and/or vomiting, 6 had fever (1 with a peak of 39.0°C, others had low-grade fever), 1 had chills, 3 had diarrhea, 3 had belching, 3 had poor appetite or anorexia, and 1 had acid reflux or heartburn. Physical signs included muscle rigidity in 8 patients, abdominal tenderness in 54 patients, and rebound tenderness in 3 patients. Only 2 patients had no significant abdominal signs, and 1 patient had neither symptoms nor signs of acute pancreatitis.

3.2.3. Laboratory examination characteristics and comparison

Laboratory test results were compared between the hepatitis complicated with pancreatitis group and the pancreatitis-only group. The hepatitis and pancreatitis group had significantly lower blood amylase, lipase, PTA%, and PLT levels compared to the pancreatitis-only group (P values all < 0.05). The TBil level in the hepatitis and pancreatitis group was significantly higher than in the pancreatitis-only group (P values all < 0.05). There were no significant differences between the two groups in WBC (× 10^9/L), NEUT (%), ALT, AST, hs-CRP, and triglyceride levels (**Table 3**).

Among the patients with viral hepatitis complicated by pancreatitis, 23 had hepatitis B and completed HBV DNA testing. Among them, 6 had HBV DNA levels ≥ 1.0E+06 IU/mL, 5 had levels ≥ 1.0E+03 IU/mL and ≤ 1.0E+06 IU/mL, and 12 had levels < 1.0E+03 IU/mL. Four patients had hepatitis C and completed HCV RNA testing and genotyping. Among them, HCV RNA levels were concentrated in the range of ≥ 1.0E+03 IU/mL and ≤ 1.0E+06 IU/mL, with genotypes of 1b in 1 patient, 3b in 2 patients, and 6a in 1 patient.

Table 3. Comparison of laboratory indicators between two groups (mean ± SD)

Indicator	Hepatitis complicated with pancreatitis (n = 53)	Acute pancreatitis (n = 56)	Statistic	P-value
Age	47.62 ± 14.058	52.57 ± 15.682	1.731	> 0.05
Hospitalization time (days)	15.36 ± 7.73	10.38 ± 5.75	-3.233	< 0.05
Blood amylase (U/L)	321.5 (105.0–776.5)	582.5 (281.25–1,583.50)	2.404	0.016
Blood lipase (U/L)	243.0 (105.5–665.0)	720.5 (246.25–1,499.98)	3.275	0.001
TBil (μmol/L)	94.8 (59.85–203.00)	21.5 (12.2– 33.3)	-6.167	< 0.001
PTA (%)	55.625 (36.025–86.225)	93.75 (77.775–109.400)	5.833	< 0.001
PLT (×10^9/L)	152.5 (104.5–203.0)	191.5 (153.75–249.25)	2.658	0.008
ALT (U/L)	90.5 (37.8–266.5)	61 (37.25–234.00)	-1.248	0.212
AST (U/L)	111.5 (40.5–293.0)	71 (30.5–188.5)	-1.767	0.077
WBC (×10^9/L)	9.51 ± 4.83	10.28 ± 4.19	0.867	> 0.05

3.2.4. Imaging features and comparison

In the hepatitis complicated with pancreatitis group, 22 patients underwent ultrasound examination. Four cases showed pancreatic enlargement and reduced echogenicity in the pancreatic parenchyma, suggesting pancreatitis, with a positive rate of 18.2% (4/22). Two cases showed pancreatic fullness, which was considered

in conjunction with the clinical condition but was not definitively diagnosed as pancreatitis. The remaining 16 cases had no abnormal pancreatic echogenicity or poor imaging due to gas interference.

In the pancreatitis-only group, 30 patients underwent ultrasound examination. Six cases reported pancreatic enlargement, thickening of the surrounding fascia, and other signs suggestive of pancreatitis, with a positive rate of 20% (6/30).

In the hepatitis complicated with pancreatitis group, 30 patients underwent upper abdominal CT scans. Twenty-three cases showed pancreatic enlargement or fullness, with peripancreatic edema and exudation, suggesting pancreatitis. One case reported extensive necrotizing pancreatitis and one case showed pancreatic tail enlargement with reduced echogenicity but without a clear diagnosis of pancreatitis. Five cases showed no obvious pancreatic morphological abnormalities, with a positive rate of 80% (24/30). In the pancreatitis-only group, 39 patients underwent upper abdominal CT scans, and 31 cases showed pancreatic enlargement and swelling with peripancreatic exudative changes. Two cases were considered necrotizing pancreatitis, with a positive rate of 84.6% (33/39).

In the hepatitis complicated with pancreatitis group, 8 patients underwent MRI. Seven cases showed peripancreatic exudative changes consistent with pancreatitis, with a positive rate of 87.5% (7/8). In the pancreatitis-only group, 11 patients underwent MRI, and pancreatitis was clearly reported in 10 cases, with a positive rate of 90.91% (10/11). Both groups had 3 patients who refused further imaging examinations after elevated lipase or amylase levels were detected. There were no significant differences in the positive rates of ultrasound, CT, or MRI between the two groups.

3.3. Univariate analysis of prognosis in hepatitis complicated with acute pancreatitis patients

Univariate analysis showed that TBil, PTA%, liver failure, NEUT%, HB, REC, PCT, and diuretic use were associated with prognosis in patients with hepatitis complicated by acute pancreatitis.

3.4. Multivariate analysis of prognosis in hepatitis complicated with acute pancreatitis patients

Liver failure was correlated with TBil and PTA% levels, HB, and REC levels. Factors with smaller *P*-values were included in the multivariate analysis. Multivariate binary logistic regression analysis showed that liver failure, NEUT%, and REC were independent risk factors affecting the prognosis of patients with hepatitis complicated by acute pancreatitis (P values all < 0.05).

4. Discussion

From the general data, there were no significant differences in age between the hepatitis complicated with pancreatitis group and the pancreatitis-only group. The majority of patients were male, with a male gender tendency observed in the hepatitis complicated with pancreatitis group, though the exact cause remains unclear [16]. Regarding etiology, various types of hepatitis, including viral hepatitis, drug-induced hepatitis, and non-alcoholic fatty liver disease, can be complicated by pancreatitis, with more than half of the patients having viral hepatitis. Previous studies [17-19] have shown that acute pancreatitis is associated with hepatitis A virus (HAV), hepatitis B virus (HBV), and hepatitis C virus (HCV) infections. Since 1999, multiple studies have reported cases of acute

pancreatitis associated with the hepatitis E virus (HEV) [20-23], and there have been reports of autoimmune pancreatitis associated with autoimmune hepatitis [24,25], though most of these are case reports. This study did not find a clear link to these rarer cases. Among the 56 pancreatitis patients, the most common cause was cholelithiasis, followed by hyperlipidemia, consistent with studies by Zhu *et al.* and Yin *et al.* [26,27]. Recent research by Wu *et al.* [28] shows that hyperlipidemia has become the most common cause of pancreatitis in the Yangzhou region of China, with its incidence rising globally. In this study, 32.1% of the pancreatitis cases had an unknown cause, and some of these patients did not undergo lipid testing, so hyperlipidemia may well be the most common cause. Acute pancreatitis can occur at any stage of hepatitis, with the highest incidence of liver failure and severe hepatitis. It is therefore understandable that the hospitalization time for patients with hepatitis complicated by pancreatitis was significantly longer than for the pancreatitis-only group.

In this study, most patients with hepatitis complicated by acute pancreatitis experienced upper abdominal dull or distending pain, with only 6 cases presenting with more severe pain radiating to the lower back. These patients often had fatigue, poor appetite, and aversion to greasy food, along with fewer cases of nausea and vomiting. In contrast, acute pancreatitis patients often had more severe abdominal pain, radiating to the back or waist, and some had colicky pain, with more noticeable nausea and vomiting. During physical examination, a higher proportion of pancreatitis patients showed significant signs, with only 2 cases lacking notable tenderness. Clinical symptoms and signs of hepatitis complicated by acute pancreatitis were atypical, with abdominal pain not clearly defined and often accompanied by digestive symptoms like fatigue, poor appetite, and aversion to greasy foods. This made it prone to misdiagnosis or missed diagnosis. In terms of imaging, although the positive diagnostic rates for ultrasound, CT, and MRI in the pancreatitis group were slightly higher than in the hepatitis complicated by the pancreatitis group, no significant difference was found. Both groups had slightly higher MRI-positive rates compared to abdominal CT, but the differences were not significant. Both abdominal CT and MRI had significantly higher positive rates than abdominal ultrasound. For diagnosing hepatitis complicated by acute pancreatitis, when clinical symptoms are atypical and indicators like lipase or amylase are insufficient for a clear diagnosis, abdominal CT or MRI should be performed promptly. If the patient cannot cooperate or refuses further examination, ultrasound should be the first choice.

From the laboratory tests, the levels of blood amylase, lipase, PTA%, and PLT in the hepatitis complicated with pancreatitis group were significantly lower than in the pancreatitis-only group (P values all < 0.05), while the TBil level in the hepatitis complicated with pancreatitis group was significantly higher than in the pancreatitis-only group (P values all < 0.05). In hepatitis complicated by pancreatitis, the higher TBil level was likely influenced by the underlying liver damage, with worse coagulation function and platelet levels. Hepatitis-related liver damage might also affect pancreatic secretion and metabolic function, which could influence blood amylase and lipase levels. Alternatively, the difference in the timing of clinical diagnosis between the two groups, influenced by atypical symptoms and signs, could explain the differences in lab results. The WBC ($\times 10^9$/L) and NEUT (%) levels in the hepatitis complicated with pancreatitis group were lower than in the pancreatitis-only group, while ALT and AST levels were higher, but without significant statistical differences. Hepatitis patients might have lower baseline WBC values, so the actual increase after developing pancreatitis may be less noticeable, with the lack of significant differences possibly due to a small sample size.

Among patients with viral hepatitis complicated by pancreatitis, 22 cases of hepatitis B virus (HBV) were tested for HBV DNA. Five cases had levels ≥ 1.0E+06 IU/mL, five cases had levels ≥ 1.0E+03 IU/mL and ≤ 1.0E+06 IU/mL, and 12 cases had levels < 1.0E+03 IU/mL. Four cases of hepatitis C virus (HCV) were tested

for HCV RNA and genotyping. The HCV RNA levels were concentrated between ≥ 1.0E+03 IU/mL and ≤ 1.0E+06 IU/mL, with genotypes 1b, 3b, and 6a identified, but there was no notable concentration of any specific genotype. A study [29] confirmed that HBV viral load is positively correlated with the incidence of pancreatitis, but this study did not find a clear trend in the HBV viral load distribution, likely because most of the patients had clinical types of liver failure or cirrhosis, and some had previously undergone antiviral treatment.

To date, no research has fully elucidated the exact pathogenesis of hepatitis-associated pancreatitis. As early as 1995, Cavallari *et al.* reported a case of a hepatitis B virus (HBV) patient with fatal acute necrotizing pancreatitis, showing strong signs of viral replication [30]. The autopsy revealed the presence of HBsAg and HBV DNA in the cytoplasm of pancreatic acinar cells, suggesting that pancreatitis may be directly caused by HBV infection. The potential role of impaired immunity might allow hepatitis viruses to affect the pancreas, reaching it via the bloodstream or bile. Current research suggests possible mechanisms, including direct cellular damage caused by extrahepatic viral replication or immune processes induced by overwhelming host immune responses during liver inflammation, both of which are potential sources of hepatitis-related pancreatitis. Hepatitis viruses are distributed in immune cells across various organs, including the liver, with lymphoid tissues being a potential target for HEV replication. Jung and colleagues recorded signs of cytotoxic and inflammatory cell infiltration in the pancreas of pigs infected with porcine HEV-3 and found that pancreatic cell damage was associated with necrotic apoptosis [16]. Necrotic apoptosis, also known as programmed cell necrosis, can be triggered by various stimuli, with the most studied being TNF signaling [31]. When TNF-α binds to TNF receptor 1 (TNFR1), the intracellular death domain of TNFR1 binds to the TNFR1-associated death domain protein. This recruits Fas-associated protein with a death domain and caspase 8 to form a death-inducing signaling complex, leading to apoptosis [32]. In this process, inhibiting caspase activity alternately activates RIP3, which then phosphorylates and oligomerizes MLKL downstream, causing necrotic apoptosis [33]. Further research is needed to uncover the mechanism by which HEV inhibits caspase activity. IFN α may participate in the development of pancreatitis through various mechanisms. Hypertriglyceridemia, a well-known cause of acute pancreatitis, is a result of interferon treatment. On another pathway, interferon α can stimulate immune responses leading to pancreas-specific autoimmune diseases. Furthermore, RBV-induced anemia and chronic hemolysis may lead to gallstones, one of the most common causes of acute pancreatitis [34]. Steroid and diuretic treatments for hepatitis can increase pancreatic enzyme secretion, thickening pancreatic fluid, causing pancreatic duct blockage, and triggering acute pancreatitis. Previous studies [35,36] suggest that pancreatic blood circulation disorders may be the main cause of acute pancreatitis, and the degree of microcirculation disorder in the pancreas is strictly correlated with the severity of acute pancreatitis. Improving pancreatic blood flow circulation can protect the pancreas [37]. In cases of liver damage, especially in severe hepatitis, cirrhosis, and liver failure, pancreatic injury may be caused by coagulation dysfunction, infection, or ischemic-hypoxic states.

Univariate analysis showed that lower TBil, PTA%, liver failure, NEUT%, lower HB, REC, PCT, and diuretic use were all risk factors for poor prognosis in hepatitis complicated by acute pancreatitis. Multivariate binary logistic regression analysis revealed that liver failure, NEUT%, and REC were independent risk factors affecting prognosis in patients with hepatitis complicated by acute pancreatitis (P values all < 0.05).

5. Conclusion

In conclusion, this study demonstrates that hepatitis complicated by acute pancreatitis presents unique clinical

features, including atypical symptoms and altered laboratory values such as lower amylase, lipase, PTA%, and PLT, alongside higher TBil levels. Imaging with CT or MRI proves valuable for diagnosis, especially in cases with nonspecific symptoms, though ultrasound remains a useful initial tool. Key independent prognostic factors—liver failure, NEUT%, and REC—indicate poorer outcomes, suggesting that patients with underlying liver disease require close monitoring. These findings highlight the complex interplay between hepatic and pancreatic conditions and the need for tailored diagnostic and therapeutic approaches to improve outcomes in affected patients.

Disclosure statement

The authors declare no conflict of interest.

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