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Analysis of clinical characteristics and risk factors for digestive system manifestations in rheumatic diseases

Анализа клиничких карактеристика и фактора ризика за манифестације дигестивног система код реуматских болести

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SUMMARY

Introduction/Objective The objective of the paper was to explore the risk factors and prediction models of digestive system complications in patients with rheumatic diseases.

Methods This case-control retrospective study enrolled 326 patients with rheumatic diseases (163 with digestive system involvement and 163 without), collecting demographic data, laboratory parameters (liver enzymes, lipid profiles, etc.) and disease types (rheumatoid arthritis, connective tissue diseases, gout, etc.). Potential variables were screened through univariate analysis, with independent risk factors subsequently identified using multivariate logistic regression analysis.

Results Univariate analysis revealed significantly higher proportions of males (35% *vs.* 20.2%, p = 0.003), alcohol consumption history (23.9% *vs.* 14.7%, p = 0.035), triglyceride (TG) (1.59 *vs.* 1.13 mmol/L, p < 0.001) and aspartate aminotransferase (AST) (21 *vs.* 19 U/L, p = 0.005) in the digestive system involvement group compared with controls. Multivariate logistic regression confirmed male gender (odds ratio [OR] = 2.276, 95% confidence interval [CI]:1.184–4.376), elevated AST (OR = 1.039/U/L, 95% CI:1.005–1.074) and hypertriglyceridemia (OR = 5.456, 95% CI:3.217–9.252) as independent risk factors (all p < 0.05).

Conclusion Male gender, elevated AST and hypertriglyceridemia constituted core predictive factors for digestive complications in rheumatic diseases, with a 445.6% increased risk observed at TG levels >1.6 mmol/L, necessitating targeted intensive monitoring and clinical intervention.

Keywords: rheumatic diseases; digestive system; risk factors

САЖЕТАК

Увод/Циљ Циљ рада био је истражити факторе ризика и моделе предвиђања компликација дигестивног система код пацијената са реуматским болестима.

Методе Ова ретроспективна студија случај-контрола обухватила је 326 пацијената са реуматским болестима (163 са захваћеношћу дигестивног система и 163 без), прикупљајући демографске податке, лабораторијске параметре (ензими јетре, липидни профили итд.) и врсте болести (реуматоидни артритис, болести везивног ткива, гихт итд.). Потенцијалне варијабле су обрађене путем униваријантне анализе, а независни фактори ризика су накнадно идентификовани коришћењем мултиваријантне логистичке регресионе анализе.

Резултати Униваријантна анализа је открила значајно већи удео мушкараца (35% наспрам 20,2%, *p* = 0,003), историје конзумирања алкохола (24,5% наспрам 14,7%, *p* = 0,035), триглицерида (*TG)* (1,59 наспрам 1,13 mmol/L, *p* < 0,001) и аспартат аминотрансферазе (*AST*) (21 наспрам 19 *U/L*, *p* = 0,005) у групи са захваћеним дигестивним системом у поређењу са контролном групом. Мултиваријантна логистичка регресија је потврдила мушкарце (*OR* = 2,276, 95% *CI*: 1,184–4,376), повишен *AST* (*OR* = 1,039/*U/L*, 95% *CI*:1,005–1,074) и хипертриглицеридемију (*OR* = 5,456, 95% *CI*: 3,217–9,252) као независне факторе ризика (сви *p* < 0,05).

Закључак: Мушкарци, повишен *AST* и хипертриглицеридемија представљали су основне предиктивне факторе за дигестивне компликације код реуматских болести, са повећаним ризиком од 445,6% при нивоима *TG* > 1,6 *mmol/L*, што је захтевало циљано интензивно праћење и клиничку интервенцију.

Кључне речи: реуматске болести; дигестивни систем; фактори ризика

INTRODUCTION

Rheumatic and autoimmune diseases, characterised by chronic inflammation of joints, muscles and connective tissues, exhibit significant global prevalence, affecting approximately 1–3% of the population [1]. This disease spectrum encompasses dozens of subtypes, including rheumatoid arthritis, systemic lupus erythematosus, sicca syndrome and systemic sclerosis, with their complex pathological mechanisms leading to multi-system damage that has become a major contributor to patient disability, impaired quality of life and reduced life expectancy [2, 3, 4].

Among these systemic manifestations, digestive system complications warrant particular attention. Clinical studies demonstrate that over 50% of patients with rheumatic disease experience varying degrees of digestive involvement, which not only exacerbates the primary condition but often leads to diagnostic and therapeutic delays due to nonspecific symptoms. Gastrointestinal diseases of systemic lupus erythematosus include several clinical manifestations with different frequencies (0.5–10.7% of cases), and liver involvement includes lupus-associated hepatitis (9.3%) and autoimmune hepatitis (2.3%) [5]. Patients with systemic sclerosis have gastroesophageal manifestations (93.3%), as well as intestinal manifestations (67.8%) and anorectal manifestations (18.9%) [6]. Autoimmune diseases can cause a variety of gastrointestinal manifestations, especially oesophageal motility disorder and small intestinal pseudo-obstruction [7, 8]. This high prevalence and complexity make rheumatic diseases with digestive complications a significant challenge in clinical practice.

The pathological mechanisms linking rheumatic diseases and digestive system damage are multidimensional. On the one hand, autoimmune abnormalities can directly trigger systemic vasculitis and mucosal inflammation. For example, Behçet's disease frequently involves the ileocecal region (occurring in approximately 88% of cases), manifesting as right lower quadrant pain, haematochezia and other symptoms that closely resemble Crohn's disease, posing significant diagnostic challenges. On the other hand, secondary damage from therapeutic agents cannot be overlooked. Nonsteroidal anti-inflammatory drugs (NSAIDs) and glucocorticoids can compromise the gastrointestinal mucosal barrier, with gastric mucosal erosion observed in approximately 30% of patients undergoing long-term NSAID therapy, substantially increasing the risk of peptic ulcers and perforation. Moreover, immunosuppressants (e.g. methotrexate) and biologic agents may directly induce hepatotoxicity and pancreatitis [8, 9, 10]. This dual pathological mechanism establishes digestive complications as a distinctive concern in rheumatic disease management, necessitating close collaboration between rheumatology and gastroenterology specialists.

Current clinical practice faces significant challenges in diagnosing rheumatic diseases with concomitant digestive system involvement. As gastrointestinal symptoms frequently serve as the initial manifestations of rheumatic diseases yet lack specificity, misdiagnosis is common. Data indicate that approximately 23% of patients with systemic lupus erythematosus presenting with abdominal pain and diarrhoea are misdiagnosed with inflammatory bowel disease or acute abdomen, whereas the oesophageal reflux caused by salivary gland dysfunction in sicca syndrome is often mistaken for primary gastroesophageal reflux disease [11]. Such misdiagnoses not only

delay treatment of the underlying disease but may also exacerbate the condition through inappropriate management. Limitations in research further constrain clinical progress. For example, critical mechanisms such as immune cross-reactivity in the gut–joint axis and the association between gut dysbiosis and rheumatic activity remain underexplored, and stratified management strategies specifically targeting digestive comorbidities are notably lacking. These issues collectively contribute to the existing knowledge gaps in current clinical practice [12].

This study systematically investigates the clinical characteristics and risk factors of digestive system manifestations in rheumatic diseases. Utilising a rigorously designed retrospective case-control study (163 patients with digestive manifestations *vs.* 163 without), the focus is on analysing demographic characteristics (e.g. gender differences), metabolic parameters (e.g. triglycerides [TG]) and hepatic function indices (e.g. aspartate aminotransferase [AST], alanine aminotransferase [ALT]) to identify clinical markers for high-risk populations. Concurrently, the mechanistic links between elevated liver enzymes and both intestinal barrier dysfunction and systemic inflammatory responses are explored. Ultimately, this research seeks to establish a clinical management pathway for rheumatic-digestive comorbidities, providing evidence-based support for developing individualised screening protocols (e.g. regular liver function tests and endoscopic surveillance) and optimising therapeutic strategies. By integrating epidemiological, molecular biological and clinical evidence, this study endeavours to address existing knowledge gaps and offer both theoretical foundations and practical guidance for reducing the incidence and mortality of digestive complications in rheumatic diseases.

METHODS

Study population

This retrospective case-control study consecutively enrolled 326 patients with rheumatic diseases admitted to the rheumatology department of a tertiary care hospital between January 2020 and December 2023, with grouping based on the presence or absence of digestive system clinical manifestations.

The study employed a sample size calculation formula for unmatched case-control studies, with the following parameter settings:

$$n=\frac{[Z_{\alpha/2}\sqrt{2\bar{P}(1-\bar{P})}+Z_{\beta}\sqrt{P_1(1-P_1)+P_0(1-P_0)}]^2}{(P_1-P_0)^2},$$

where $Z_{\alpha/2}$ and Z_{β} represent the critical values of the standard normal distribution corresponding to the significance level and statistical power ($Z_{\alpha/2}$ = 1.96 at α = 0.05; Z_{β} = 1.282 at β = 0.10); P_0 denotes the estimated exposure rate of the target factor (e.g. NSAID use history) in controls, set at 20% based on literature and preliminary data; P_1 indicates the exposure rate in cases, calculated as $P1 = 1-P_0 + OR \times P_0 OR \times P_0 = 33.3\%$ using the anticipated odds ratio (OR = 2.0); and $\bar{P}$ represents the mean exposure rate across groups ($\bar{P}$ = 0.267). Substituting these values into the formula yielded a minimum sample size of 170 per group. Accounting for a potential 10% missing data in retrospective studies, the adjusted sample size was 189 per group (total ≥ 378). Although the actual enrolment of 326 patients (combined cases and controls) fell below this target, power analysis confirmed > 85% statistical power under α = 0.05, OR = 2.0 and P_0 = 0.20, meeting methodological requirements. Sensitivity analysis demonstrated that sample size requirements would decrease to < 150 per group if P_0 rose to 25% or the OR increased to 2.5. Given that consecutive enrolment minimised selection bias, the final sample of 326 patients remained adequate for analysing associations between target exposures and digestive manifestations.

The inclusion criteria for the case group (with digestive manifestations) were as follows: (1) age ≥ 18 years; (2) fulfilment of international diagnostic criteria for rheumatic diseases (e.g. American College of Rheumatology/European Alliance of Associations for Rheumatology classification criteria for rheumatoid arthritis, Systemic Lupus International Collaborating Clinics criteria for systemic lupus erythematosus) [13]; (3) presence of at least one digestive system manifestation, including but not limited to gastritis, splenomegaly, hyperbilirubinemia, hyperlipidaemia, hepatic steatosis, abnormal liver function, pancreatitis, gastrointestinal bleeding or endoscopically confirmed mucosal lesions. The control group (without digestive manifestations) shared identical criteria except for the absence of aforementioned digestive involvement. Exclusion criteria applied to both groups included (1) concurrent solid or haematologic malignancies; (2) major surgery or trauma within 3 months; (3) pregnancy or lactation; and (4) missing key laboratory data (e.g. TG, AST).

Data collection

Study variables were systematically collected through the hospital's electronic medical record system. Demographic characteristics included age, gender, body mass index (BMI), smoking history (defined as continuous smoking > 6 months) and alcohol consumption history (ethanol intake > 30 g/day for ≥ 1 year). Disease characteristics encompassed rheumatic disease types

(classified into 21 subtypes, as shown in Table 1) and disease duration (calculated from initial diagnosis). Laboratory measurements were uniformly performed using Beckman AU5800 automated analysers. Liver function tests (AST, ALT, gamma-glutamyl transferase [GGT], alkaline phosphatase [ALP]) employed rate assays, lipid profiles (TG, total cholesterol) utilised enzymatic colorimetry, immunological markers (rheumatoid factor, immunoglobulins IgG/IgA/IgM, complements C3/C4) were measured via immunonephelometry and tumour markers (alpha-fetoprotein, carbohydrate antigen 19-9, carbohydrate antigen 72-4) were analysed via chemiluminescence immunoassays.

Data acquisition was independently performed by two uniformly trained researchers using standardised case report forms, with all imaging results interpreted under double-blind conditions by two radiologists holding associate chief physician qualifications or higher ($\kappa = 0.84$).

Statistical analysis

Statistical analyses were performed using SPSS 26.0 software. Normality was assessed using the Kolmogorov–Smirnov test. Normally distributed continuous variables were expressed as mean ± standard deviation ($\bar{x} \pm s$) and compared using independent samples t-tests, whereas non-normally distributed data were presented as median (interquartile range) (M [Q1, Q3]), with Mann–Whitney U tests used for inter-group comparisons. Categorical variables were reported as frequencies (percentages) and analysed using chi-square (χ^2) or Fisher's exact tests. Variables with $p < 0.05$ in univariate analysis were entered into multivariate logistic regression models (forward stepwise method) to calculate ORs with 95% confidence intervals (95% CI), with statistical significance set at $p < 0.05$.

Ethics: This study was conducted in accordance with the 1975 Declaration of Helsinki and approved by the Ethics Committee of The Third Affiliated Hospital of Jinzhou Medical University (Approval number: JYDSY-KXYJ-IEC-2025-051). As patient identities were anonymised and this was an observational study, the ethics committee granted a waiver of informed consent following review.

RESULTS

Distribution characteristics of rheumatic disease spectrum

Analysis of 326 patients with rheumatic diseases systematically compared the disease spectrum distribution and clinical parameters between the digestive system involvement group (n =

163) and the non-digestive involvement group (n = 163). Regarding disease type distribution (Table 1), the non-digestive group was predominantly rheumatoid arthritis (99 cases, 60.7%), followed by connective tissue diseases (17 cases, 10.4%) and ankylosing spondylitis (11 cases, 6.7%). In contrast, the digestive involvement group showed not only rheumatoid arthritis (80 cases, 49.1%) but also a significantly higher proportion of gouty arthritis (18 cases, 11%), with eight disease subtypes, including ANCA-associated vasculitis and myositis, exclusively observed in this group, suggesting unique associations between specific rheumatic conditions and digestive complications.

Univariate analysis: risk factors for digestive complications

Univariate analysis (Table 2) revealed multiple significant inter-group differences. The digestive involvement group had a higher male proportion (57 *vs.* 33 cases, $\chi^2 = 8.841$, $p = 0.003$) and more frequent alcohol consumption (39 *vs.* 24 cases, $\chi^2 = 4.427$, $p = 0.035$). Laboratory profiles demonstrated prominent metabolic and hepatic dysfunction in this group. Median TG levels were significantly elevated (1.59 *vs.* 1.13 mmol/L, $z = -7.837$, $p < 0.001$); liver enzymes were uniformly increased, including AST (21 *vs.* 19 U/L, $z = -2.839$, $p = 0.005$), ALT (20 *vs.* 16 U/L, $z = -3.039$, $p = 0.002$), GGT (29 *vs.* 22 U/L, $z = -4.360$, $p < 0.001$) and ALP (80 *vs.* 72 U/L, $z = -2.598$, $p = 0.009$). Notably, rheumatoid factor levels were paradoxically lower (20.0 *vs.* 22.5 IU/mL, $z = -2.485$, $p = 0.013$). No statistical differences were observed in 23 parameters, including age, BMI and complement levels.

Multivariate logistic regression: identification of independent risk factors

Multivariate logistic regression analysis (Table 3) incorporating statistically significant factors from univariate analysis identified three independent risk factors. First, male gender conferred a 127.6% increased risk of digestive complications (OR = 2.276, 95% CI: 1.184−4.376, $p = 0.014$); second, each 1 U/L increment in AST level was associated with a 3.9% risk elevation (OR = 1.039, 95% CI: 1.005–1.074, $p = 0.026$); and most notably, TG demonstrated the highest OR of 5.456 (95% CI: 3.217–9.252, $p < 0.001$), indicating a 445.6% risk escalation among individuals with elevated TG. Although alcohol consumption history and ALT levels showed significance in univariate analysis, they lost independent predictive value after adjusting for confounders ($p>0.05$). Other parameters, including immunoglobulins, complements and bilirubin, also failed to achieve statistical significance in the multivariate model.

DISCUSSION

This large-scale case-control study systematically identified independent risk factors and underlying mechanisms for digestive complications in rheumatic diseases. Male gender, elevated serum AST and hypertriglyceridemia were established as core predictors, with OR values of 2.276, 1.039 and 5.456, respectively, providing crucial evidence for early clinical identification of high-risk patients. Notably, the heightened risk in male patients was closely associated with sex hormone-mediated immune dysregulation. Androgens promote Th1/Th17 cell differentiation, enhancing the release of proinflammatory factors such as tumour necrosis factor-alpha and interleukin (IL)-17 that disrupt the intestinal mucosal barrier, whereas oestrogens exert anti-inflammatory protection by upregulating Treg cell function [14, 15, 16]. This immunological divergence explains male patients' increased susceptibility to drug-induced liver injury (e.g. methotrexate toxicity) and ischemic colitis, consistent with our finding of significantly higher male representation (35.0% *vs.* 20.2%) in the complication group [17].

The independent predictive value of serum AST ($p = 0.026$), surpassing that of ALT ($p = 0.655$), carries significant pathological implications. As AST is predominantly localised in mitochondria, its elevation indicates organelle-level damage, reflecting the gut–liver axis vicious cycle activated by endotoxin translocation following intestinal barrier disruption [18]. Increased intestinal permeability allows portal vein endotoxins to activate hepatic Kupffer cells, releasing pro-fibrotic factors such as IL-6 and transforming growth factor-beta that exacerbate intestinal mucosal damage. This mechanism explains the comprehensive elevation of liver enzymes (AST: 21 U/L; GGT: 29 U/L) in the digestive involvement group and confirms the 3.9% increased risk per 1 U/L AST rise in multivariate models. In contrast, ALT primarily reflects cytoplasmic injury and shows inadequate sensitivity for early enterogenic hepatopathy, suggesting that AST should serve as the core biomarker for monitoring intestinal complications in rheumatic diseases [19].

Triglyceride demonstrated the strongest predictive efficacy (OR = 5.456) through three pathological mechanisms. (1) Free fatty acids activate the TLR4/NF-κB pathway, inducing intestinal macrophages to secrete IL-1β and IL-18 that directly disrupt intestinal epithelial tight junctions; (2) chylomicron deposition causes mesenteric microvascular occlusion, leading to mucosal ischemic necrosis – particularly prominent in patients with systemic sclerosis; and (3) hyperlipidaemic environments significantly increase local NSAID concentrations in the intestinal lumen, exacerbating chemical injury via COX-1 inhibition [20, 21]. The median TG level in our complication group reached 1.59 mmol/L (significantly higher than the 1.13 mmol/L in

controls, $p < 0.001$), with gouty arthritis accounting for 11.0% of cases, further evidencing the synergistic activation of NLRP3 inflammasomes by hyperuricemia and hypertriglyceridemia, forming a 'metabolic-inflammatory storm'. This unique association underscores the necessity for enhanced lipid monitoring and intervention in patients with gouty arthritis.

The disease spectrum analysis revealed critical clinical patterns. Eight disease subtypes, including ANCA-associated vasculitis and myositis, were exclusively observed in the digestive involvement group, with patients with myositis exhibiting 100% complication rates attributable to anti-Jo-1 antibody-mediated smooth muscle inflammation. These patients typically presented with oesophageal dysmotility (hypotensive esophagus) and delayed gastric emptying, warranting cautious use of conventional prokinetics (e.g. domperidone) due to QT prolongation risks. These findings indicate the need for disease-specific individualised management protocols for digestive complications. However, it should be noted that while alcohol consumption showed significance in univariate analysis ($p = 0.035$), it failed to maintain significance in multivariate models ($p = 0.922$), potentially reflecting confounding effects of alcohol-induced CYP450 enzyme alterations on drug metabolism, necessitating further pharmacokinetic analyses [22].

Based on these findings, we propose a three-tier prevention system. First, high-risk patients (males, or those with TG ≥ 1.6 mmol/L or AST ≥ 21 U/L) should undergo quarterly monitoring of liver enzyme profiles and faecal occult blood tests, with consideration for preventive interventions. Specifically, fibrates (fenofibrate 200 mg/day) should be the first-line treatment for hypertriglyceridemia, as they activate PPARα to downregulate the NF-κB pathway, thereby reducing intestinal inflammation. For patients with persistently elevated AST, a combination of ursodeoxycholic acid (10 mg/kg/day) and bifidobacterium preparations is recommended to restore the intestinal mucosal barrier. Male patients with gout require strict control of serum uric acid levels (< 360 μmol/L) to prevent intestinal urate crystal deposition. Notably, glucocorticoids significantly increase TG levels (by approximately 28%) – for patients with hyperlipidaemia, IL-6 inhibitors such as tocilizumab should be prioritised over glucocorticoids.

This study has several limitations. First, the analysis did not account for medication exposure history (e.g. duration and dosage of NSAID use). Second, it lacked assessments of gut microbiota diversity and barrier function markers such as serum zonulin. Third, limited subgroup sample sizes (e.g. myositis) restricted stratified statistical power.

CONCLUSION

This retrospective case-control analysis of 326 patients with rheumatic diseases identified male gender (OR = 2.276), elevated serum AST (OR = 1.039/U/L) and TG (OR = 5.456) as independent risk factors for digestive complications in rheumatic diseases. Here, TG (≥ 1.59 mmol/L) demonstrated the most prominent predictive value, conferring a 445.6% increased risk, mechanistically linked to free fatty acid-mediated activation of the intestinal TLR4/NF-κB inflammatory pathway. In clinical practice, we recommend quarterly monitoring of liver enzyme profiles and faecal occult blood tests for high-risk populations (males, persistent AST > 21 U/L, or TG > 1.6 mmol/L). These findings provide critical evidence for establishing an early warning system for rheumatic-digestive comorbidities, warranting future multicentre cohort studies to validate the efficacy of interventions in reducing complication incidence.

Author contributions

Conception and design of the work: Zhu D; Data collection: Hao YQ; Supervision: Zhu D; Analysis and interpretation of the data: Hao YQ; Statistical analysis: Zhu D, Hao YQ; Drafting the manuscript: Zhu D; Critical revision of the manuscript: all authors; Approval of the final manuscript: all authors.

Conflict of interest: None declared.

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Table 1. Patients by type of rheumatic disease with and without gastrointestinal disorders

Type	Rheumatic diseases with digestive involvement (n = 163)	Rheumatic diseases without digestive system involvement (n = 163)
Behçet's disease	0	2
Enteropathic arthritis	0	1
Polymyalgia rheumatica	1	2
Sjögren's syndrome	8	10
Osteoarthritis	3	4
Spondylarthritis	0	1
Connective tissue diseases	12	17
Rheumatoid arthritis	80	99
Ankylosing spondylitis	11	11
Generalized osteoarthritis	1	2
Systemic lupus erythematosus	10	9
Systemic sclerosis	5	3
Psoriatic arthritis	2	2
ANCA-associated vasculitis	2	0
Adult-onset Still's disease	1	0
Takayasu arteritis	2	0
Reactive arthritis	1	0
Myositis	4	0
Gouty arthritis	18	0
Amyopathic dermatomyositis	1	0
Palmoplantar pustulosis	1	0

Table 2. Univariate factor analysis of rheumatic diseases with and without gastrointestinal involvement

Index	Rheumatic diseases with digestive involvement (n = 163)	Rheumatic diseases without digestive system involvement (n = 163)	z/t/χ^2	p
Age (year)	59.95±14.05	61.77±13.03	1.214	0.226
Sex			8.841	0.003
Male	57	33		
Female	106	130		
BMI	23.19 ±3 .19	23.73 ± 3.19	1.521	0.129
Rheumatoid factor	20 (20, 55.18)	22.5 (20, 117)	-2.485	0.013
CPR	0.98 (0.34, 2.23)	0.63 (0.26, 2.07)	-1.016	0.310
ESR	35 (20, 65)	33 (15, 78)	-1.625	0.104
IGg	13.20 (10.5, 16.3)	13.70 (11.4, 17.2)	-0.383	0.702
IGa	3.51 (2.47, 4.3)	3.34 (2.34, 4.57)	-0.636	0.525
IGm	1.17 (0.75, 1.74)	1.12 (0.74, 1.82)	-0.006	0.995
C3	1.03 (0.82, 1.22)	0.99 (0.81, 1.18)	-1.041	0.298
C4	0.27 (0.22, 0.35)	0.26 (0.19, 0.33)	-1.280	0.201
AST	21 (17, 28)	19 (15, 23)	-2.839	0.005
ALT	20 (13, 32)	16 (11, 24)	-3.039	0.002
GGT	29 (19, 48)	22 (15, 34)	-4.360	< 0.001
ALP	80 (63, 95)	72 (58, 89)	-2.598	0.009
Bilirubin	10. 20 (6.8, 14.5)	9.70 (7.3, 12.7)	-1.354	0.176
TG	1.59 (1.13, 2.29)	1.13 (0.85, 1.34)	-7.837	< 0.001
CHO	4.77 (4.03, 5.49)	4.56 (3.96, 5.22)	-1.840	0.066
LLLD	2.74 (2.17, 3.39)	2.57 (2.20, 3.24)	-0.625	0.532
AFP	3.12 (1.83, 6.06)	2.98 (1.81, 2.98)	-0.234	0.815
PG	6 (4.2, 8.86)	5.90 (3.85, 8.83)	-0.614	0.539
CA199	6.46 (3.73, 15.4)	7.06 (3.89, 15.08)	-0.187	0.851
CA724	3.62 (1.56, 7.3)	4.30 (1.46, 8.3)	-0.781	0.435
Disease course	5.08±6.93	5.87±8.06	0.950	0.343
Smoking history			0.366	0.545
No	139	135		
Yes	24	28		
Alcohol consumption history				
No	124	139	4.427	0.035
Yes	39	24		

BMI – Body Mass Index; CPR – C-reactive Protein; ESR – Erythrocyte Sedimentation rate; IGg – immunoglobulin G; IGa – immunoglobulin A; IGm – immunoglobulin M; C3 – complement C3; C4 – complement C4; AST – aspartate aminotransferase; ALT – alanine aminotransferase; GGT – gamma-glutamyl transferase; ALP – alkaline phosphatase; TG – triglyceride; CHO – total cholesterol; LLLD – low-density lipoprotein cholesterol; AFP – alpha-fetoprotein; PG – prostaglandin; CA199 – carbohydrate antigen 19-9; CA724 – carbohydrate Antigen 72-4

Table 3. Multivariate logistic analysis of rheumatic diseases combined with digestive system

Influencing factor	B	SE	Wald χ^2 value	p	OR	95% confidence interval	
						Lower limit	Upper limit
Sex*	0.823	0.334	6.082	0.014	2.276	1.184	4.376
Alcohol consumption history*	-0.035	0.363	0.010	0.922	0.965	0.474	1.964
Rheumatoid factor	0.000	0.000	0.350	0.554	1.000	0.999	1.001
AST	0.038	0.017	4.983	0.026	1.039	1.005	1.074
ALT	-0.004	0.010	0.199	0.655	0.996	0.976	1.015
GGT	0.006	0.007	0.755	0.385	1.006	0.992	1.021
ALP	0.004	0.006	0.521	0.470	1.004	0.993	1.016
TG	1.697	0.270	39.625	0.000	5.456	3.217	9.252
Constant	-3.836	0.716	28.667	0.000	0.022		

AST – aspartate aminotransferase; ALT – alanine aminotransferase; GGT – gamma-glutamyl transferase; ALP – alkaline phosphatase; TG – triglyceride;

*variable assignments: sex (Male = 0, Female = 1); alcohol consumption history (No = 0, Yes = 1); other continuous variables (e.g., AST, TG) used raw measured values

