

Potential Hazards of Chronic Inflammation to Human Health

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Abstract

Objective: To investigate the potential hazards of chronic inflammation to human health. **Methods:** A total of 186 patients with chronic inflammation admitted to our hospital from January 2022 to June 2025 were enrolled. Inflammatory marker levels and the incidence of organ-system injury were analyzed, and differences in organ damage were compared among patients with different inflammatory burdens and disease durations. **Results:** The high-level hs-CRP subgroup showed significantly higher detection rates of cardiovascular abnormalities, impaired glucose metabolism, fatty liver, cognitive decline, and combined multisystem involvement than the low-level hs-CRP subgroup ($P < 0.05$). With increasing disease duration, the proportion of patients with single-system involvement gradually decreased, whereas the proportion with involvement of three or more systems increased stepwise ($P < 0.05$). **Conclusion:** Chronic inflammation may cause extensive damage to the cardiovascular, metabolic, hepatic, and nervous systems. Higher inflammatory activity and longer persistence are associated with broader and more severe organ involvement.

Keywords

Chronic inflammation; human health; potential hazards; organ injury

Chronic inflammation is a pathophysiological state in which the immune system remains persistently activated at a low intensity in the absence of a clearly identifiable infectious trigger [1]. Unlike acute inflammation, this condition may persist for a prolonged period without obvious subjective symptoms, yet it can gradually damage multiple organ systems. In recent years, increasing evidence has suggested a close association between chronic inflammation and organ injury [2]. In the present retrospective study, 186 patients with chronic inflammation were included to explore the relationship between inflammatory intensity, disease duration, and multisystem organ involvement.

1. Materials and Methods

1.1 General Information

A total of 186 patients with chronic inflammation who were admitted to our hospital between January 2022 and June 2025 were selected as the study population. All patients and their family members provided informed consent. Among the 186 patients, 104 were male, and 82 were female. The age ranged from 35 to 75 years, with a mean age of 55.34 ± 10.82 years. The body mass index ranged from 20 to 32 kg/m^2 , with a mean of $26.12 \pm 3.76 \text{ kg/m}^2$. The disease duration ranged from 0.5 to 11 years, with a mean of 4.28 ± 0.43 years. This study was approved by the hospital ethics committee.

Inclusion criteria were as follows [3]: (1) patients who met the diagnostic criteria for chronic inflammation according to the Guidelines for Clinical Diagnosis and Stratified Management of Chronic Low-Grade Inflammatory States based on comprehensive clinical assessment; (2) patients with serum high-sensitivity C-reactive protein (hs-CRP)

levels >1 mg/L in two measurements performed at an interval of no less than 3 months; and (3) patients with complete clinical data.

Exclusion criteria were as follows [4]: (1) mental disorders; (2) severe dysfunction of important organs; (3) acute infectious diseases; (4) recent treatment with glucocorticoids or other immunomodulatory agents; (5) reproductive tract infectious diseases; and (6) malignant tumors.

1.2 Methods

Laboratory testing: Fasting venous blood samples were collected from all participants in the morning after an 8-hour fast. hs-CRP was measured by immunoturbidimetry. Interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha) were detected using an enzyme-linked immunosorbent assay. Fasting blood glucose (FBG) was measured by the glucose oxidase method, and glycated hemoglobin (HbA1c) was determined by high-performance liquid chromatography. Blood lipid parameters, including total cholesterol (TC), triglycerides (TG), and low-density lipoprotein cholesterol (LDL-C), were measured using an automatic biochemical analyzer. Alanine aminotransferase (ALT) was measured by the rate method. All blood samples were processed within 2 hours after collection.

Auxiliary examinations: A carotid plaque was considered positive when carotid ultrasonography showed an intima-media thickness ≥ 1.5 mm or focal thickening $>50\%$ compared with the adjacent segment. Ultrasonographic diagnosis of fatty liver was based on diffuse enhancement of hepatic parenchymal echogenicity, accompanied by poor visualization of intrahepatic vessels. Resting 12-lead electrocardiography was used to assess ischemic changes, including ST-segment depression or T-wave inversion. Cognitive function was evaluated using the Mini-Mental State Examination (MMSE), with a total score of 30 points; a score <27 was defined as cognitive decline. Ultrasound and electrocardiographic reports were reviewed by two independent assessors.

1.3 Observation Indicators

Patients were grouped according to the median hs-CRP level and disease duration (0.5-2 years, 2-5 years, and >5 years). The following outcomes were recorded: cardiovascular abnormalities, defined as carotid atherosclerotic plaque detected by ultrasonography or ischemic changes on electrocardiography; abnormal glucose metabolism, defined as FBG ≥ 6.1 mmol/L, HbA1c $\geq 6.5\%$, or a confirmed diagnosis of diabetes mellitus; hepatic steatosis confirmed by ultrasonography; cognitive decline, defined as MMSE score <27 ; and multisystem involvement, defined as the coexistence of at least two of the above four abnormalities [5].

1.4 Statistical Analysis

Statistical analyses were performed using SPSS 26.0. Normally distributed measurement data were expressed as mean \pm standard deviation and compared between groups using the t-test. Categorical variables were expressed as numbers and percentages and compared using the chi-square test. A two-sided P value <0.05 was considered statistically significant.

2. Results

2.1 Detection Rates of System Abnormalities According to the hs-CRP Level

The median hs-CRP level of the whole cohort was 4.2 mg/L and was used as the cutoff value. The detection rates of cardiovascular abnormalities, abnormal glucose metabolism, fatty liver, cognitive decline, and combined multisystem involvement were all significantly higher in the high-level hs-CRP subgroup than in the low-level subgroup ($P < 0.05$). Detailed data are shown in Table 1.

Table 1. Comparison of detection rates of system abnormalities according to hs-CRP level [n (%)]

Group	Cardiovascular abnormalities	Abnormal glucose metabolism	Fatty liver	Cognitive decline	Multisystem involvement
hs-CRP ≤ 4.2 mg/L (n=93)	33 (35.48)	27 (29.03)	25 (26.88)	11 (11.83)	35 (37.63)
hs-CRP > 4.2 mg/L (n=93)	58 (62.37) *	50 (53.76) *	42 (45.16) *	23 (24.73) *	63 (67.74) *

Note: * $P < 0.05$ compared with the hs-CRP ≤ 4.2 mg/L group.

2.2 Comparison of Multisystem Involvement According to Disease Duration

As the duration of inflammation increased, the proportion of patients with single-system involvement gradually decreased, whereas the proportion of patients with involvement of three or more systems increased stepwise ($P<0.05$). The detailed distribution is shown in Table 2.

Table 2. Comparison of system involvement according to duration of inflammation [n (%)]

Group	No system involvement	Single-system involvement	Two-system involvement	Involvement of ≥ 3 systems
0.5-2 years (n=74)	17 (22.97)	39 (52.70)	12 (16.22)	6 (8.11)
2-5 years (n=68)	8 (11.76) *	18 (26.47) *	22 (32.35)	20 (29.41) *
>5 years (n=44)	3 (6.82) *	7 (15.91) *	13 (29.55)	21 (47.73) *

Note: * $P<0.05$ compared with the 0.5-2 years group.

3. Discussion

The present study showed that the detection rates of cardiovascular abnormalities, abnormal glucose metabolism, fatty liver, cognitive decline, and combined multisystem involvement were significantly higher in the high-level hs-CRP subgroup than in the low-level subgroup ($P<0.05$). These findings indicate that the adverse effects of chronic inflammation are systemic and progressive. As hs-CRP levels increase, abnormalities involving the cardiovascular system, metabolism, liver, and cognitive function become more common. This phenomenon may be related to the persistent release of inflammatory mediators, such as IL-6 and TNF-alpha, under chronic low-grade inflammatory conditions. These mediators can directly impair vascular endothelial function, promote the formation of atherosclerotic plaques, interfere with insulin signaling pathways, and contribute to impaired insulin sensitivity and glucose metabolism [6].

This study also demonstrated that with prolonged disease duration, the proportion of patients with single-system involvement gradually decreased, whereas the proportion with involvement of three or more systems increased in a stepwise manner ($P<0.05$). This suggests a clear relationship between the persistence of inflammation and the expansion of organ-system involvement. If the inflammatory microenvironment is not effectively interrupted, injury may progress from functional disturbance to structural damage and gradually spread to other organs, eventually leading to complex multisystem involvement [7, 8].

In summary, long-term chronic inflammation may lead to multisystem organ damage. The higher the inflammatory burden and the longer its duration, the wider and more severe the scope of organ injury. Timely recognition of chronic inflammatory status and active control of disease progression are therefore important for protecting patient health.

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