Preplanned Studies

Accessible Blood Based Biomarkers Reflecting Inflammation-Lipid Dysregulation in Silicosis Progression — Jiangsu Province, China, 2021–2024

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Summary

What is already known about this topic?

Silicosis, an occupational lung disease caused by exposure to silica dust, is characterized by persistent inflammation and lipid dysregulation. Clinicians typically rely on radiographic imaging and pulmonary function tests instead of accessible blood-based biomarkers to quantify inflammation-lipid imbalance and predict functional decline in disease management.

What is added by this report?

We evaluate two novel hematological indices — Neutrophil-to-HDL Ratio (NHR) and Platelet-to-HDL Ratio (PHR) — in 160 patients with silicosis and 123 silica-exposed controls from Jiangsu Province, China. Both the NHR and PHR of patients in the silicosis group are significantly higher than those in the control group, with the highest levels observed in advanced stages (Stage II–III). Both ratios show significant negative correlations with lung function decline (FVC%, FEV_1%, FEV_1/FVC), and these correlations strengthen in patients with advanced silicosis.

What are the implications for public health practice?

NHR and PHR are low-cost, accessible biomarkers of inflammation-lipid dysregulation during the progression of silicosis. Incorporating these ratios into routine occupational health screening for silica-exposed workers could serve as a complementary method to radiographic examination or spirometry tests, thereby improving silicosis monitoring and management.

ABSTRACT

Introduction: The activation of inflammatory cells and lipid metabolism disorder initiated by silica are the key pathological mechanisms of silicosis. This study aims to analyze easily accessible blood biomarkers that reflect this inflammation-lipid imbalance and improve disease monitoring and management.

Methods: A cross-sectional investigation was conducted in 160 male patients with silicosis, and 123 silica-exposed control workers (male ceramic workers without silicosis). Neutrophil-to-HDL ratio (NHR) and Platelet-to-HDL ratio (PHR) were calculated from routine blood counts and high-density lipoprotein (HDL). Pulmonary function parameters (FVC%, FEV_1%, FEV_1/FVC) were evaluated by pulmonary function test. We studied group differences and correlations using statistical analysis.

Results: The NHR and PHR of patients in the silicosis group were significantly higher than those in the control group, with the highest levels in advanced stages (Stage II–III). All lung function parameters were significantly reduced in silicosis patients. NHR and PHR showed significant negative correlations with lung function decline (FVC%, FEV_1%, FEV_1/FVC), and these correlations strengthened in patients with advanced silicosis.

Conclusion: NHR and PHR are elevated and inversely correlate with worsening lung function in patients with silicosis, especially in advanced disease. These ratios are readily available, cost-effective biomarkers for monitoring inflammation-lipid dysregulation and functional decline during the progression of silicosis. Incorporating NHR and PHR into routine occupational health screening for workers exposed to silica could help in risk stratification and disease management.

Silicosis, a serious occupational lung disease prevalent among ceramic workers, miners, and construction workers, is driven by persistent inflammation and pulmonary fibrosis following exposure to crystalline silica exposure. Recently, it has been discovered that the activation of inflammatory cells and lipid metabolism disorder initiated by silica

are the key pathological mechanisms of silicosis (*1*). Currently, no effective strategies exist to delay silicosis progression; therefore, exploring biomarkers related to its pathological processes is essential for facilitating reliable monitoring and, guiding clinical management. C-reactive protein (CRP), Interleukin-6 (IL-6), and erythrocyte sedimentation rate (ESR) are well-known inflammatory markers of silicosis. However, they cannot be obtained from routine, low-cost blood tests that are widely used in clinical practice, limiting their application in the health surveillance of silica-exposed workers. Furthermore, these markers do not reflect lipid metabolic dysregulation, which is central to the pathogenesis of silicosis. In contrast, hematological ratios combining leukocytes with high-density lipoprotein (HDL) could function as representative indices for evaluating lipid metabolism disorder and the level of inflammatory response level (*2*). Moreover, these ratios serve as cost-effective and technically accessible biomarkers for subclinical inflammation detection, requiring only routine blood parameters. Several recent studies have suggested that the Neutrophil-HDL Ratio (NHR) could quantify the imbalance between inflammatory components and lipid parameters, and has been widely used in many inflammatory conditions, including malignancies, acute coronary syndromes, cerebrovascular events and neurodegenerative disorders (*3*). Similarly, the Platelet-HDL Ratio (PHR) correlates thrombo-inflammatory burden with lipid metabolic dysregulation, wherein elevated values denote heightened thrombo-inflammatory activity and altered lipid metabolism homeostasis (*4*). Several studies with NHR and PHR have demonstrated that these blood indices could detect inflammation-lipid dysregulation status in interstitial lung disease (ILD); however, no reports have been published on patients with silicosis, and even less is known about the relationship between these parameters and pulmonary functional decline during silicosis progression.

Therefore, this cross-sectional study aimed to determine whether patients with silicosis exhibit an activated inflammatory response and lipid metabolic disorder using potential biomarkers obtained from regular blood tests. Moreover, we evaluated their relationships with the pulmonary functional parameters.

Data were collected from medipopulationcal records of the Jiangsu Provincial Center for Disease Control and Prevention, spanning between January 2021 and December 2024. The study included ceramic workers engaged in raw material crushing, trimming, glazing, and product polishing, who were referred for occupational health screening due to suspected silicosis. A definitive diagnosis requires multidisciplinary consensus, a history of silica dust exposure, and radiological confirmation according to the International Labour Organization (ILO) classification criteria (*5*). Exclusion criteria eliminated individuals with acute infections [clinical diagnosis or laboratory evidence (e.g., body temperature >38 °C, leukocyte count $>10\times10^9$/L) of any acute infection (e.g., respiratory, urinary) within the past 4 weeks], chronic inflammatory conditions (a history of systemic autoimmune or chronic inflammatory disorders, e.g., rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel disease), chronic infectious diseases [e.g., chronic viral hepatitis B or C, tuberculosis, or human immunodeficiency virus (HIV) infection], metabolic and endocrine disorders leading to lipid metabolic disturbance (e.g., diabetes mellitus, hyperlipidemia), malignancies, or use of medications affecting hematological parameters. The study analyzed 160 patients with silicosis, and 123 silica-exposed controls (radiographically healthy ceramic workers) from Jiangsu Province, China. Based on ILO pneumoconiosis classifications, the silicosis severity distribution was as follows: 106 patients (66.25%) had silicosis at Stage I (early stage of silicosis), and 54 patients (33.75%) had Stage II–III silicosis (advanced stage of silicosis). Peripheral blood samples were collected to calculate NHR and PHR from complete blood counts and HDL measurements. We assessed pulmonary function parameters including forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_1), and FEV_1/FVC. Data analysis was performed using SPSS version 25.0 (IBM Corp., NY, USA). Continuous variables were classified as mean±standard deviation (parametric) or median (interquartile range) (non-parametric). For intergroup differences, parametric data underwent one-way analysis of variance (ANOVA) test while non-parametric distributions were analyzed via Kruskal-Wallis test. Two-group comparisons were performed using Student's *t*-tests (parametric) or Mann-Whitney *U*-tests (non-parametric). Spearman's correlation coefficient was used to measure bivariate associations. Statistical significance was indicated as $P<0.05$. For the significant Kruskal-Wallis test results (e.g., for NHR,

PHR, lung function), post-hoc pairwise comparisons were conducted to identify the differences between two groups (e.g., Control *vs*. Stage I, Control *vs*. Stage II–III and Stage I *vs*. Stage II–III). To decrease the risk of type I error due to these multiple comparisons, a Bonferroni-corrected *P* threshold of <0.017 (0.05/3) was applied.

All participants were male workers aged ≥60 years, with detailed sociodemographic information presented in Table 1. Compared to controls, patients with silicosis exhibited significantly prolonged silica exposure duration (*P*=0.00) and higher smoking prevalence (*P*=0.01). No intergroup differences were observed in age or alcohol consumption patterns. Routine hematological analysis (Table 2) revealed that the total leukocyte count did not differ between the three groups studied (Control, Stage I, and Stage II–III). Moreover, a non-significant increasing trend was observed in the neutrophil counts. Similarly, the platelet count was notably elevated in the Stage I and Stage II–III silicosis groups compared to that in the control group, but intergroup differences lacked statistical significance. Moreover, lymphocyte counts and HDL concentrations did not differ between controls and either patients groups, although downward trends were observed for lymphocytes and HDL concentrations in both parameters in the Stage I and Stage II–III silicosis groups.

A significant increase in NHR levels was observed in patients with silicosis [3.07 (2.07, 3.94) in Stage I and 3.08 (2.60, 4.72) in Stage II–III] compared with controls [2.82 (2.04, 3.73)]. There was a significant

TABLE 1. Basic sociodemographic characteristics of the study population.

Characteristics	Overall (*n*=283)	Control (*n*=123)	Silicosis (*n*=160)	*P*
Age (years)	70.26±5.40	70.06±5.38	70.41±5.44	0.58
Duration of exposure (years)	24.00 (10.00, 32.00)	9.00 (5.00, 15.00)	29.50 (25.00, 36.00)	0.00
Smoking status, *n* (%)				0.01
Smoker	136 (48.06)	48 (39.02)	88 (55.00)	
Non-smoker	147 (51.94)	75 (60.98)	72 (45.00)	
History of alcohol intake, *n* (%)				0.47
Yes	115 (40.64)	47 (38.21)	68 (42.50)	
No	168 (59.36)	76 (61.79)	92 (57.50)	

Note: Data are presented as median (interquartile range), mean±standard deviation, or *n* (%). Significance was set as *P*<0.05.

TABLE 2. Laboratory data of the study population.

Characteristics	Control (*n*=123)	Stage Ⅰ silicosis (*n*=106)	Stage II–III silicosis (*n*=54)	*P*
Laboratory findings				
WBC (x10^9/L)	6.46 (5.43, 7.67)	6.60 (5.40, 7.90)	6.95 (5.81, 8.25)	0.16
Neutrophils (x10^9/L)	3.90 (3.07, 4.70)	3.98 (3.24, 4.92)	4.23 (3.46, 5.30)	0.06
Lymphocytes (x10^9/L)	2.14 (1.62, 2.67)	2.05 (1.47, 2.77)	1.99 (1.63, 2.40)	0.59
Platelet (x10^9/L)	175.00 (147.00, 208.00)	190.50 (147.75, 221.00)	188.50 (153.50, 221.50)	0.10
HDL (mmol/L)	1.37 (1.19, 1.60)	1.33 (1.11, 1.64)	1.30 (1.14, 1.52)	0.29
NHR (10^9/mmol)	2.82 (2.04, 3.73)	3.07 (2.07, 3.94)	3.08 (2.60, 4.72)*	0.02
PHR (10^9/mmol)	120.13 (95.62, 154.17)	134.04 (103.30, 187.62)	145.70 (112.40, 167.28)*	0.02
Lung functions				
FVC (% pred)	78.00 (73.00 , 85.00)	75.50 (66.75, 85.00)	68.00 (54.75, 78.25)*,**	0.00
FEV1 (% pred)	77.00 (72.00, 83.00)	73.00 (67.53, 81.50)*	65.00 (46.00, 73.25)*,**	0.00
FEV1/FVC%	85.00 (80.00, 91.00)	78.98 (63.75, 86.12) *	65.93 (56.00, 79.25)*,**	0.00

Note: Data are presented as median (interquartile range), or mean±standard deviation. *P*<0.05: *P* from the Kruskal-Wallis *H* test across the three groups (Control, Stage I, Stage II–III).
Abbreviation: HDL=high-density lipoprotein; NHR=neutrophil to HDL ratio; PHR=platelet to HDL ratio; FVC=forced vital capacity; FEV1=forced expiratory volume in 1 second.
* *P*<0.017 (Bonferroni-corrected): compared to the controls in the Mann-Whitney *U* test;
** *P*<0.017 (Bonferroni-corrected): compared to Stage Ⅰ silicosis in the Mann-Whitney *U* test.

difference between all the three cohorts (P=0.02). The post-hoc pairwise comparisons revealed that NHR was elevated in both Stage I (P=0.03) and Stage II–III patients (P=0.01) compared with controls. However, only the difference between Stage II–III patients and controls was statistically significant after Bonferroni correction (P<0.017). No significant difference in NHR was observed between Stage I and Stage II–III patients. PHR also exhibited a significant progressive increase in patients with silicosis [134.04 (103.30, 187.62) in Stage I and 145.70 (112.40, 167.28) in Stage II–III] and showed a significant difference among all the groups studied (P=0.015). The post-hoc pairwise comparisons revealed that compared to controls, PHR was elevated in both Stage I (P=0.04) and Stage II–III patients (P=0.01) compared with controls. Similarly, only the difference between Stage II–III patients and controls was statistically significant after Bonferroni correction. All pulmonary function parameters (FEV1%, FVC% and FEV1/FVC) showed significant overall differences among the three groups (all P=0.00). Post-hoc pairwise comparisons revealed that compared with controls, patients with silicosis exhibited significant functional impairment. Compared with controls, Stage I patients had lower FVC% (P=0.04), FEV_1% (P=0.00), and FEV_1/FVC (P=0.00). Stage II-III patients also showed more significant pulmonary dysfunction in all parameters than controls (all P=0.00). Furthermore, advanced-stage patients manifested more severe functional deterioration than early-stage counterparts, with lower FVC% (P=0.00), FEV_1% (P=0.00) and FEV_1/FVC (P=0.00).

Spearman's correlation analysis results (Table 3) demonstrated that NHR presented significant negative correlations with FVC (r=−0.25, P=0.00), FEV_1 (r=−0.26, P=0.00) and FEV1/FVC (r=−0.22, P=0.01). Similarly, PHR was negatively correlated with FVC (r=−0.20, P=0.01), FEV_1 (r=−0.24, P=0.00), and FEV1/FVC (r=−0.24, P=0.00). Hence, these findings suggested significant correlations between hematological ratios and disease severity in patients with silicosis. Notably, the strength of these correlations intensified in patients with Stage II–III silicosis. Especially, NHR-FEV1/FVC (r=−0.39, P=0.00) and PHR-FEV_1 (r=−0.41, P=0.00) correlations in Stage II–III silicosis were nearly equivalent to −0.4, reflecting the stronger impact of neutrophilic or lymphatic inflammation and HDL dysfunction on spirometry deterioration in advanced-stage of silicosis.

DISCUSSION

Current evidence confirms the clinical utility of accessible hematological indices, including NHR, PHR, systemic inflammation indices and aggregate inflammation markers as minimally invasive inflammatory biomarkers (*3*–*4*). However, their roles in the pathogenesis of silicosis remain unexplored. Crucially, no previous investigations have been established to explore the relationship between NHR or PHR and respiratory functional decline in cohorts with silicosis. This cross-sectional analysis demonstrated that novel hematological ratios integrating immune cells with lipid parameters-specifically NHR and PHR cav serve as sensitive indicators associated with pulmonary function tests in ceramic workers with silicosis. Quantitative analysis demonstrated significantly elevated NHR and PHR in patients with silicosis compared with silica-exposed controls. These ratios exhibited stage-dependent amplification, with Stage II–III patients, displaying the most pronounced elevations (Stage I: moderate increase; Stage II–III: marked enhancement). Notably, these ratios demonstrated negative correlations with pulmonary function parameters such as FEV_1%, FVC% and FEV_1/FVC ratio, which suggested that the

TABLE 3. Correlations between NHR, PHR and pulmonary function in silicosis.

Characteristics		FVC (% pred)		FEV1 (% pred)		FEV1/FVC%	
		r	*P*	*r*	*P*	*r*	*P*
Silicosis (*n*=160)	NHR	−0.25	0.00	−0.26	0.00	−0.22	0.01
	PHR	−0.20	0.01	−0.24	0.00	−0.24	0.00
Stage II–III (*n*=54)	NHR	−0.28	0.04	−0.34	0.01	−0.39	0.00
	PHR	−0.33	0.02	−0.41	0.00	−0.25	0.07

Note: Significance was set as P<0.05.
Abbreviation: NHR=neutrophil to high-density lipoprotein ratio; PHR=platelet to HDL ratio; FVC=forced vital capacity; FEV1=forced expiratory volume in 1 second.

inflammation-lipid imbalance occurred in parallel with pulmonary function impairment.

Silica particle phagocytosis mediated by alveolar macrophages initiates of the pathogenic cascade of silicosis, which ultimately leads to pulmonary fibrosis (*1*). The activation of inflammatory cells and lipid metabolism disorder initiated by silica are key pathological mechanisms of silicosis. Neutrophils, the most abundant leukocyte subtype, promoted diseases progression by secreting pro-inflammatory cytokines, reactive oxygen species and proteolytic effectors. Researchers have found that the neutrophils counts are significantly upregulated not only in the serum but also in the lung tissues of patients with silicosis (*6*). In contrast, lymphocytes, another abundant subtype of leukocytes, were significantly lower in the serum of patients with silicosis. This may be because lymphocytes primarily mediated immune responses primarily through T cells, particularly the regulatory T (Treg) cells, which could suppress inflammation and promote silicosis development (*7*). In addition to their roles in thrombosis, platelets are now recognized to have immune regulatory and inflammatory signaling properties. Platelet activation can promote inflammatory cytokine secretion, stimulate leukocyte recruitment and disrupt endothelial function, all of which contribute to pulmonary inflammation and fibrosis (*8*). Moreover, elevated platelet-activation factor (PAF) in silicosis plasma and amelioration of silica-induced fibrosis by PAF antagonists in models collectively delineate the thrombo-inflammatory contributions. Critically, neutrophils, platelets and lymphocytes exhibited integrated roles in the pathogenesis of silicosis, with dynamic lipid remodeling, particularly LDL elevation and HDL reduction. HDL, traditionally associated with lipid metabolism, is now recognized as a key mediator involved in anti-inflammatory, antioxidant and antifibrotic responses. Current evidence indicates its significant association with idiopathic pulmonary fibrosis onset, progression and prognosis (*8*). Our data showed that although total leukocyte and platelet counts exhibited no intergroup differences, patients with advanced stages of silicosis presented with non-significant neutrophilia. Moreover, although lymphocyte counts and HDL concentrations did not reveal any differences between controls and patients with silicosis, downward trends were observed in the Stage I and Stage II–III silicosis groups.

The novel inflammatory-metabolic indices NHR and PHR, which integrate neutrophil/platelet (inflammatory markers) with HDL (anti-inflammatory/antioxidant marker) ratios, reflected the imbalance between inflammatory processes and lipid metabolism. In this study, NHR was significantly upregulated in patients with silicosis compared to silica-exposed controls, with significant differences observed across all the three cohorts, showing an increasing trend from Stage I silicosis to Stage II–III silicosis. A possible explanation for this might be that early radiographic stages are characterized by localized, low-grade inflammation at silica deposition sites, whereas advanced stages manifest with inflammation-lipid dysregulation proportional to the cumulative silica burden. PHR could serve as a validated quantitative indicator for assessing thrombo-inflammatory responses and lipid metabolic disorder across diverse pathologies. Clinical studies have confirmed the diagnostic and prognostic utility of PHR in chronic respiratory pathologies, including chronic obstructive pulmonary disease (COPD) and acute pulmonary vascular events such as embolism (*9*). Our analysis demonstrated that PHR also presented a significant progressive increase in patients with silicosis. Although the difference did not reach statistical significance, Stage II–III patients exhibited higher PHR values than their Stage I counterparts. Additionally, we found that single indices showed no significant differences, while combined ratios (NHR and PHR) showed significant differences between the groups, indicating the superiority of hematological parameters combining inflammatory cells with HDL in the evaluation of inflammatory response and lipid dysregulation during silicosis.

Significant inverse correlations were observed between hematological indices and pulmonary function parameters in the silicosis group. Even though the strength of correlation coefficients was modest (r ranged from -0.2 to -0.4), their practical significance remained important. This is because low-cost and readily available indicators make it easier for high-risk patients with silicosis to be quickly identified and treated early. Moreover, in patients with advanced stage silicosis, the correlations were stronger ($r \approx -0.4$ for several pairs). The gradual strengthening of correlations in Stage II–III silicosis suggests more pronounced associations between inflammation-lipid dysregulation, as assessed by NHR and PHR, and the decline in spirometry parameters with disease progression. These findings are consistent with those of Kang HY et al., who indicated that several systemic immune inflammation indices presented significant

inverse correlations with pulmonary function in a silicosis cohort (*10*).

Our findings provide important guidance for clinical practice. First, the discovery of blood combined biomarkers, including NHR and PHR, opens a new perspective on the potential pathological mechanisms of silicosis, suggesting a potential role of lipid metabolism and inflammatory responses. Second, NHR and PHR not only correlated with lung dysfunction but also associated with the presence and progression of silicosis. These findings both optimize the diagnostic process of silicosis and suggest new therapeutic targets for future treatment strategies.

The findings in this report are subject to at least 4 limitations. First, the absence of detailed information on potential confounders, including workplace exposure histories, quantitative dust measurements, and smoking status limited the potential confounders analysis and the independent prognostic capacity of NHR and PHR. Second, silica-exposed controls were not matched to silicosis cases on key potential confounders such as age, duration of silica exposure or smoking status, thus, matched designs for potential confounders would be included in subsequent studies. Third, all participants in the study were male workers aged ≥60 years, therefore, generalizability of our findings to female silicosis patients, younger individuals, or workers from other silica-exposed industries (e.g., mining, construction) should be apporached with caution and warrants further exploration. Finally, since the sample size of the advanced-stage subgroup was small, larger cohort studies arre required to confirm the stability and reliability of the stronger correlations in this subgroup.

NHR and PHR are accessible, low-cost blood-based biomarkers that reflect the degree of inflammation-lipid dysregulation in silicosis patients and are significantly associated with the decline in pulmonary function, with a stronger correlation observed particularly in advanced-stage silicosis. Based on these findings, it is recommended that NHR and PHR be incorporated into the routine occupational health surveillance system for silica-exposed workers, serving as supplementary indicators of chest imaging and pulmonary function tests.

Conflicts of interest: No conflicts of interest.

Ethical statement: All participants provided written informed consent prior to participation. Ethical approval was obtained from Jiangsu Provincial Center for Disease Control and Prevention (Approval JSJK2022-B002-01) and all procedures adhered to the Helsinki Declaration.

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