Journal of Clinical and Nursing Research, 2024, Volume 8, Issue 11
http://ojs.bbwpublisher.com/index.php/JCNR
Online ISSN: 2208-3693
Print ISSN: 2208-3685

Clinical Implications of Eosinopenia in Adult Brucellosis Patients

Luxuan Yang[1,2], Dan Xiao[1,2], Chuanwu Zhu[1,2], Haiyan Wang[1,2], Wenyong Zhang[1,2], Jianguo Chang[1,2], Meiqin Liu[1,2], Xiujuan Shen[1,2]*

[1]Department of Infection Disease, The Affiliated Infectious Diseases Hospital of Soochow University, Suzhou 215000, Jiangsu Province, China
[2]Department of Infection Disease, The Fifth People's Hospital of Suzhou, Suzhou 215000, Jiangsu Province, China

***Corresponding author:** Xiujuan Shen, 530024067@qq.com

Abstract: *Objective:* To analyze the differences between eosinopenia and non-eosinopenia brucellosis patients in depth. *Methods:* Medical records of brucellosis patients admitted to the Affiliated Infectious Diseases Hospital of Soochow University between January 1, 2013, and December 31, 2023, were reviewed retrospectively. Patients were categorized into an eosinopenia group and a non-eosinopenia group based on pre-treatment eosinophil levels. A nonparametric test was performed to estimate the differences between the two groups. *Results:* Among the 125 patients, 66 (52.80%) experienced eosinopenia. Patients with eosinopenia were older (52.09 ± 15.63 years vs. 46.08 ± 16.39 years, $P = 0.024$), had a higher proportion of hypertension (21.21% vs. 6.78%, $P = 0.024$), and exhibited a greater likelihood of complications (75.76% vs. 35.59%, $P = 0.000$), particularly hematological (68.18% vs. 23.73%, $P = 0.000$) and relapse (19.70% vs. 6.78%, $P = 0.040$). The eosinopenia group also showed higher levels of ALT (29.00 vs. 20.00, $P = 0.003$), AST (29.00 vs. 22.00, $P = 0.037$), and LOS (17.50 vs. 12.00, $P = 0.000$). Among certain inflammatory indicators related to brucellosis, the eosinopenia group demonstrated lower levels, such as MPV (9.75 vs. 10.70, $P = 0.000$), MLR (0.28 vs. 0.36, $P = 0.002$), and SIRI (0.67 vs. 1.03, $P = 0.004$). *Conclusion:* Brucellosis patients with eosinopenia differed in clinical manifestations and prognosis. Monitoring eosinophils may provide better prognostic assessment and suggest potential new treatment options.

Keywords: Brucellosis; Eosinopenia; Complication; Prognosis

Online publication: November 26, 2024

1. Introduction

Brucellosis, one of the most neglected zoonotic diseases globally, poses a major threat to human health and increases social burden. In recent years, the epidemiology of human brucellosis has changed significantly, with its geographical spread continuously expanding, especially in Asia [1]. In China, brucellosis cases have rapidly

increased since the mid-1990s, making it one of the infectious diseases with the highest reported morbidity rates. The morbidity rate rose from 0.07 per 100,000 in 1995 to 4.95 per 100,000 in 2021, with the number of deaths ranking among the top 10 legally reported infectious diseases of categories A and B [2]. This infectious disease urgently requires increased attention and comprehensive research.

Brucellosis often has a long and recurrent course, affecting multiple systems and organs. Hematologic abnormalities are frequently observed and are associated with infection, hypersplenism, phagocytosis, myelosuppression, diffuse anticoagulation, and autoimmune hemolysis [3]. However, current research primarily focuses on diseases such as cytopenia, hemophagocytic lymphohistiocytosis, and myelofibrosis [3-6], with limited studies on eosinophils. A recent study on brucellosis [7] suggests that eosinopenia is a significant laboratory finding, indicating that the role of eosinophils may have been underestimated.

The correlation between eosinopenia and the severity of infectious diseases has recently been observed in influenza, COVID-19, and varicella [8–10]. This study aimed to analyze the clinical characteristics of brucellosis patients with eosinopenia to identify possible mechanisms and therapeutic targets.

2. Materials and methods

2.1. Ethics statement

This study adheres to medical ethics standards and was reviewed and approved by the Ethical Review Committee of the Affiliated Infectious Diseases Hospital of Soochow University (No: K2024-007-01). All treatments and procedures were performed with informed consent obtained from patients' family members.

2.2. Patient recruitment

Brucellosis patients admitted to the Affiliated Infectious Diseases Hospital of Soochow University between January 1, 2013, and December 31, 2023, were considered eligible for enrollment. The inclusion criteria were as follows: (1) aged ≥ 18 years; (2) confirmed to have brucellosis by blood culture (blood samples cultured for more than seven days) or a positive serological test (Standard Tube Agglutination Test ≥ 1:100); (3) received standardized treatment; and (4) had complete clinical data. The exclusion criteria were as follows: (1) patients previously diagnosed with brucellosis; (2) patients for whom relapse could not be distinguished from reinfection; (3) patients with a history of infections, trauma, or surgery in the past month; (4) patients who were pregnant or lactating; and (5) patients with acute or chronic hepatitis, nephritis, tumors, blood system diseases, immune system diseases, or other severe organ diseases.

2.3. Data collection

Collected data included demographic characteristics, clinical data, therapeutic schedule, and outcomes.

(1) Demographic characteristics: Information on age, gender, epidemiological history, past medical history, personal history, symptoms, and complications was collected. All complications were determined based on the patient's symptoms and established laboratory tests and imaging examinations. Hematological abnormalities were defined as follows: blood hemoglobin < 120 g/L for males and < 110 g/L for females, leukocyte count < 4×10^9/L, platelet count < 150×10^9/L, and leukocyte count > 10×10^9/L, corresponding to anemia, leukopenia, thrombocytopenia, and leukocytosis, respectively. Osteoarticular, respiratory, and genitourinary involvements were identified through imaging examinations, while

gastrointestinal involvement was indicated by elevated alanine aminotransferase or aspartate aminotransferase levels and related clinical symptoms.

(2) Clinical data: Blood test results and inflammatory indicators were recorded. All blood samples were collected on the day of admission or the following morning after fasting. Blood test parameters included serum leukocyte count (WBC, 10^9/L), serum hemoglobin (HGB, g/L), serum platelet count (PLT, 10^9/L), serum neutrophil count (NE, 10^9/L), serum lymphocyte count (LY, 10^9/L), serum monocyte count (MON, 10^9/L), serum eosinophil count (EOS, 10^9/L), mean platelet volume (MPV, fL), red cell distribution width (RDW, %), alanine aminotransferase level (ALT, U/L), aspartate aminotransferase level (AST, U/L), gamma-glutamyl transferase level (GGT, U/L), alkaline phosphatase level (ALP, U/L), albumin (ALB, g/L), total bilirubin (TBIL, μmol/L), serum creatinine (Cr, μmol/L), glucose (GLU, mmol/L), serum C-reactive protein level (CRP), serum procalcitonin level (PCT), and microbiological results. Inflammation indices were calculated as follows: neutrophil-lymphocyte ratio (NLR) = NE ÷ LY; platelet–lymphocyte ratio (PLR) = PLT ÷ LY; monocyte–lymphocyte ratio (MLR) = MON ÷ LY; systemic immune-inflammation index (SII) = PLT × NE ÷ LY; systemic inflammation response index (SIRI) = NE × MON ÷ LY; CALLY index = ALB × LY ÷ (CRP × 10).

(3) Therapeutic schedule: Information on treatment duration, therapeutic drugs, and length of stay (LOS) was recorded. Most patients received a 6-week course of treatment, which was extended based on symptoms if necessary. Patients with osteoarticular involvement received a 12-week course of treatment. Main therapeutic drugs included doxycycline (DOX), rifampicin (RIF), sulfamethoxazole/trimethoprim (SMZ/TMP), and ceftriaxone (CRO).

(4) Outcomes: Outcomes were recorded as sequelae and relapse. Sequelae were considered present if the patient continued to experience discomfort related to brucellosis after Brucella had been eliminated from the body. If a patient exhibited symptoms associated with brucellosis during post-treatment follow-up, blood culture and serological tests were refined to confirm relapse.

2.4. Grouping criterion

Due to the absence of patients with elevated eosinophils, patients were categorized into eosinopenia and non-eosinopenia groups based on whether their pre-treatment eosinophil count was less than 0.05×10^9/L.

2.5. Statistical analysis

Data were analyzed using SPSS (IBM SPSS Statistics 23.0). Measurement data conforming to a normal distribution were expressed as mean ± standard deviation (SD), with a *t*-test used for comparison between groups. Measurement data with a non-normal distribution were expressed as median (IQR), with comparisons between groups performed by the Mann–Whitney U test. Categorical data were expressed as percentages (%), with comparisons between groups performed by chi-squared test or Fisher exact test. $P < 0.05$ was considered to indicate statistical significance.

3. Results

3.1. General characteristics

A total of 125 brucellosis patients were enrolled in the study, with 86 (68.80%) male and 39 (31.20%) female.

The average age was 49.26 ± 16.21 years. Among them, 66 patients (52.80%) experienced eosinopenia. Patients with eosinopenia tended to be older (52.09 ± 15.63 years vs. 46.08 ± 16.39 years, $P = 0.024$) and exhibited a higher proportion of hypertension (21.21% vs 6.78%, $P = 0.024$). No significant differences were noted between the eosinopenia and non-eosinopenia groups regarding diagnostic time, epidemiological history, personal history, or symptoms (**Table 1**).

Table 1. Comparison of epidemiological and clinical features between the eosinopenia group (Group 1) and the non-eosinopenia group (Group 2).

Variables	Total (n = 125)	Group 1 (n = 66)	Group 2 (n = 59)	P value
Male	86 (68.80%)	48 (72.73%)	38 (64.41%)	0.339
Age (years)	49.26 ± 16.21	52.09 ± 15.63	46.08 ± 16.39	0.024
With epidemiology history	25 (20.00%)	11 (16.67%)	14 (23.73%)	0.375
Time consumed in diagnosis (months)	1.00 (0.50, 3.00)	1.00 (0.50, 2.00)	2.00 (0.75, 3.00)	0.058
Past history				
Hypertension	18 (14.40%)	14 (21.21%)	4 (6.78%)	0.024
Diabetes	10 (8.00%)	8 (12.12%)	2 (3.39%)	0.101
Personal history				
Smoking	19 (15.20%)	12 (18.18%)	7 (11.86%)	0.455
Drinking	5 (4.00%)	3 (4.55%)	2 (3.39%)	0.674
Symptoms				
Fever	115 (92.00%)	61 (92.42%)	54 (91.53%)	1.000
Weakness	115 (92.00%)	59 (89.39%)	56 (94.92%)	0.332
Arthralgia	101 (80.80%)	51 (77.27%)	50 (84.75%)	0.365
Sweating	75 (60.00%)	44 (66.67%)	31 (52.54%)	0.143
Muscle ache	55 (44.00%)	29 (43.94%)	26 (44.07%)	1.000
Lack of appetite	46 (36.80%)	28 (42.42%)	18 (30.51%)	0.196
Lymphadenopathy	15 (12.00%)	8 (12.12%)	7 (11.86%)	0.795
Cough	4 (3.20%)	2 (3.03%)	2 (3.39%)	1.000

3.2. Complications between eosinopenia and non-eosinopenia groups

The eosinopenia group showed a significantly higher probability of complications (75.76% vs. 35.59%, $P = 0.000$). Hematological complications were more prevalent in the eosinopenia group (68.18% vs. 23.73%, $P = 0.000$), with significantly higher rates of anemia (39.39% vs, 13.56%, $P = 0.001$) and leukopenia (37.88% vs. 8.47%, $P = 0.001$). There was no difference between groups for thrombocytopenia or leukocytosis. No differences were found for osteoarticular, respiratory, gastrointestinal, or genitourinary complications, though some complications occurred exclusively in the eosinopenia group (**Table 2**).

Table 2. Comparison of complications between the eosinopenia group (Group 1) and non-eosinopenia group (Group 2) [*n* (%)]

Variables	Total (*n* = 125)	Group 1 (*n* = 66)	Group 2 (*n* = 59)	*P* value
With complications	71 (56.80%)	50 (75.76%)	21 (35.59%)	0.000
Hematological involvement	59 (47.20%)	45 (68.18%)	14 (23.73%)	0.000
Anemia	34 (27.20%)	26 (39.39%)	8 (13.56%)	0.001
Leukopenia	30 (24.00%)	25 (37.88%)	5 (8.47%)	0.000
Thrombocytopenia	9 (7.20%)	9 (13.64%)	0 (0.00%)	/
Leukocytosis	6 (4.80%)	3 (4.55%)	3 (5.08%)	1.000
Osteoarticular involvement	15 (12.00%)	10 (15.15%)	5 (8.47%)	0.283
Spondylodiscitis	11 (8.80%)	6 (9.09%)	5 (8.47%)	1.000
Sacroiliitis	2 (1.60%)	2 (3.03%)	0 (0.00%)	/
Osphyarthrosis	1 (0.8%)	1 (1.52%)	0 (0.00%)	/
Gonarthritis	1 (0.8%)	1 (1.52%)	0 (0.00%)	/
Gastrointestinal involvement	13 (10.40%)	9 (13.64%)	4 (6.78%)	0.251
Transaminase elevation	12 (9.60%)	8 (12.12%)	4 (6.78%)	0.373
Hepatosplenomegaly	1 (0.8%)	1 (1.52%)	0 (0.00%)	/
Respiratory involvement	3 (2.40%)	3 (4.55%)	0 (0.00%)	/
Hydrothorax	2 (1.60%)	2 (3.03%)	0 (0.00%)	/
Pneumonia	1 (0.8%)	1 (1.52%)	0 (0.00%)	/
Genitourinary involvement	1 (0.8%)	1 (1.52%)	0 (0.00%)	/
Epididymo-orchitis	1 (0.8%)	1 (1.52%)	0 (0.00%)	/
Two systems involvement	16 (12.80%)	14 (21.21%)	2 (3.39%)	0.003
More than two systems involvement	2 (1.60%)	2 (3.03%)	0 (0.00%)	/

3.3. Laboratory findings between eosinopenia and non-eosinopenia groups pre-treatment

Laboratory data taken before treatment initiation revealed that the eosinopenia group had lower WBC (4.44 [3.68, 6.43] vs. 5.70 [4.81, 6.70], $P = 0.007$), HGB (125.00 [108.50, 133.00] vs. 134.00 [127.00, 141.00], $P = 0.000$), PLT (169.00 [129.00, 228.75] vs. 220.00 [197.00, 274.00], $P = 0.001$), and MON (0.37 [0.29, 0.48] vs. 0.52 [0.40, 0.81], $P = 0.000$), but higher ALT (29.00 [16.00, 41.50] vs. 20.00 [11.00, 28.00], $P = 0.003$) and AST (29.00 [19.00, 39.00] vs. 22.00 [15.00, 33.00], $P = 0.037$) levels than the non-eosinopenia group. There were no significant differences in other indicators (**Table 3**).

Table 3. Comparison of laboratory findings between eosinopenia group (Group 1) and non-eosinopenia group (Group 2) pre-treatment

Variables	Total (*n* = 125)	Group 1 (*n* = 66)	Group 2 (*n* = 59)	*P* value
WBC ($\times 10^9$/L)	5.60 (4.09, 6.64)	4.44 (3.68, 6.43)	5.70 (4.81, 6.70)	0.007
HGB (g/L)	131.00 (116.00, 137.50)	125.00 (108.50, 133.00)	134.00 (127.00, 141.00)	0.000
PLT ($\times 10^9$/L)	208.00 (156.50, 265.50)	169.00 (129.00, 228.75)	220.00 (197.00, 274.00)	0.001
NE ($\times 10^9$/L)	3.06 (2.11, 4.32)	2.70 (1.78, 4.48)	3.09 (2.43, 4.30)	0.062
LY ($\times 10^9$/L)	1.51 (1.06, 1.83)	1.47 (1.05, 1.81)	1.61 (1.07, 1.90)	0.207
MON ($\times 10^9$/L)	0.43 (0.33, 0.66)	0.37 (0.29, 0.48)	0.52 (0.40, 0.81)	0.000
ALT (U/L)	21.00 (13.00, 37.50)	29.00 (16.00, 41.50)	20.00 (11.00, 28.00)	0.003
AST (U/L)	28.00 (18.00, 35.00)	29.00 (19.00, 39.00)	22.00 (15.00, 33.00)	0.037
GGT (U/L)	47.00 (33.00, 107.00)	47.00 (30.50, 94.00)	45.00 (36.00, 107.00)	0.089
ALP (U/L)	90.00 (78.00, 114.00)	90.00 (72.50, 110.50)	100.00 (79.00, 151.00)	0.145
Tbil (μmol/L)	11.50 (7.65, 14.90)	12.10 (7.65, 16.20)	11.50 (6.80, 12.20)	0.238
GLU (mmol/L)	5.42 (4.96, 6.37)	5.44 (5.11, 6.56)	5.30 (4.96, 5.82)	0.094
Creatinine (μmol/L)	56.35 (50.10, 69.48)	56.70 (49.25, 69.35)	55.80 (52.00, 72.00)	0.855
h-CRP (mg/L)	21.10 (10.40, 44.00)	20.80 (8.05, 33.95)	22.70 (11.15, 45.85)	0.376
PCT (ng/mL)	0.02 (0.00, 0.25)	0.00 (0.00, 0.45)	0.02 (0.00, 0.05)	0.221

3.4. Microbiological results, treatment, and outcomes

All patients in the study were referred after testing positive for blood cultures or having a serum agglutination test (SAT) ≥ 1:100 at an external facility. The eosinopenia group had a higher proportion of diagnoses based on blood culture (89.39% vs 72.88%, $P = 0.021$). The duration of treatment did not differ significantly between groups; however, the length of stay was longer for patients with eosinopenia (17.50 [14.75, 23.00] vs. 12.00 [9.00, 14.00], $P = 0.000$). The eosinopenia group also showed a higher likelihood of relapse (19.70% vs. 6.78%, $P = 0.040$), though no differences were found for cases of multiple relapses or sequelae between the two groups (**Table 4**).

Table 4. Summary of microbiological, treatment, and outcome data between the eosinopenia group (Group 1) and non-eosinopenia group (Group 2)

Variables	Total (*n* = 125)	Group 1 (*n* = 66)	Group 2 (*n* = 59)	*P* value
Microbiological results				0.021
Blood culture	102 (81.60%)	59 (89.39%)	43 (72.88%)	
STA ≥ 1:100	23 (18.40%)	7 (10.61%)	16 (27.12%)	
Treatment				
Treatment duration (weeks)	6.00 (6.00, 8.00)	6.00 (6.00, 8.00)	6.00 (6.00, 6.00)	0.558
LOS (days)	14.00 (12.00, 21.00)	17.50 (14.75, 23.00)	12.00 (9.00, 14.00)	0.000

Table 4 (Continued)

Variables	Total (*n* = 125)	Group 1 (*n* = 66)	Group 2 (*n* = 59)	*P* value
Antibiotic combinations				
DOX+RIF	89 (71.20%)	44 (66.67%)	45 (76.27%)	0.323
DOX+ SMZ/TMP	20 (16.00%)	11 (16.67%)	9 (15.25%)	1.000
DOX+RIF +CRO	15 (12.00%)	10 (15.15%)	5 (8.47%)	0.283
Others	1 (0.80%)	1 (1.52%)	0 (0.00%)	/
Outcome				
With relapse	17 (13.60%)	13 (19.70%)	4 (6.78%)	0.040
With twice relapse	6 (4.80%)	5 (7.58%)	1 (1.69%)	0.212
With more than twice relapse	1 (0.80%)	1 (1.51%)	0 (0.00%)	/
With sequelae	9 (7.20%)	7 (10.61%)	2 (3.39%)	0.170

3.5. Inflammatory markers between eosinopenia and non-eosinopenia groups pre-treatment

Analysis of inflammatory markers suggested potential differences associated with brucellosis. The eosinopenia group displayed lower levels in certain markers, including MPV (9.75 [9.20, 10.73] vs. 10.70 [10.00, 12.60], $P = 0.000$), MLR (0.28 [0.19, 0.34] vs. 0.36 [0.23, 0.50], $P = 0.002$), and SIRI (0.67 [0.37, 1.69] vs. 1.03 [0.64, 1.87], $P = 0.004$). No differences were observed for RDW, NLR, PLR, SII, or the CALLY index (**Table 5**).

Table 5. Comparison of inflammatory markers between eosinopenia group (Group 1) and non-eosinopenia group (Group 2) pre-treatment

Variables	Total (*n* = 125)	Group 1 (*n* = 66)	Group 2 (*n* = 59)	*P* value
MPV (fL)	10.10 (9.40, 11.50)	9.75 (9.2, 10.73)	10.70 (10.00, 12.60)	0.000
RDW (%)	11.90 (10.90, 15.10)	13.45 (10.65, 15.40)	11.10 (11.10, 14.00)	0.144
NLR	2.05 (1.19, 3.53)	1.92 (0.96, 4.35)	2.06 (1.24, 3.34)	0.469
PLR	81.60 (68.61, 121.90)	84.85 (66.68, 121.59)	81.60 (68.79, 128.97)	0.663
MLR	0.29 (0.21, 0.44)	0.28 (0.19, 0.34)	0.36 (0.23, 0.50)	0.002
SII	424.08 (243.94, 781.71)	383.85 (178.90, 727.84)	444.46 (283.73)	0.055
SIRI	0.89 (0.49, 1.80)	0.67 (0.37, 1.69)	1.03 (0.64, 1.87)	0.004
CALLY index	0.20 (0.10, 0.38)	0.20 (0.12, 0.32)	0.26 (0.07, 0.53)	0.768

4. Discussion

Brucellosis is a zoonotic infectious disease caused by Brucella bacteria, which profoundly and multifacetedly impacts individuals and society. Asia bears the highest burden of human brucellosis among continents, creating a serious public health problem. Traditional agricultural practices, lifestyles, and the consumption of fresh dairy products, such as raw milk, contribute to this high prevalence [1], particularly notable in China and deserving increased attention [2].

The review of relevant literature [7,11,12] indicates that brucellosis complicated by eosinopenia is not uncommon, yet remains insufficiently studied. Eosinopenia frequently occurs in brucellosis, especially during the early acute phase, and is thought to aid in diagnosis. This study confirms these findings and further identifies distinct clinical characteristics and potential mechanisms in patients with eosinopenia.

Brucellosis is easily misdiagnosed, often progressing to a chronic phase due to its atypical clinical symptoms. Early diagnosis is both highly needed and challenging to achieve. Hematological complications, such as anemia, leukopenia, and thrombocytopenia, are frequently observed in brucellosis [4] and other infectious diseases [13], thus limiting their diagnostic utility. When combining previous studies with findings from this research, eosinopenia emerges as an effective and convenient diagnostic aid.

This study further found that eosinopenia patients were older and had significantly higher risks of relapse and complications. Prior research on brucellosis [14-17] associates advanced age with increased risks of relapse and complications, potentially suggesting a poorer prognosis. This correlation adds reliability to the results of this study. While it remains unclear whether the age-related decline in the number and function of T cells, B cells, and NK cells—attributable to weakened immune function [18,19]—increases the likelihood of bone marrow suppression and eosinopenia during infections, eosinopenia may be considered a risk factor for poor prognosis.

A comparison with historical data reveals a higher proportion of hypertension in the eosinopenia group, attributed to the group's greater mean age rather than a direct association with brucellosis. Nonetheless, a potential connection between hypertension and brucellosis warrants blood pressure monitoring during treatment and further investigation into underlying mechanisms.

Eosinopenia was closely associated with complications, particularly hematological ones, in this study. The relevant literature does not address correlation studies between these two variables. However, based on available data, it can be hypothesized that eosinopenia results from infection, with the degree of decline potentially linked to the quantity and virulence of Brucella abortus, thereby influencing disease prognosis. This finding establishes a significant relationship between eosinopenia and complications.

A slight increase in transaminase elevation among the eosinopenia group was observed, though without statistical significance. However, significant elevations in ALT and AST levels suggest potential liver damage in this group. Another study [20] indicates that eosinophils accumulate in injured liver tissue during immune-mediated damage, secreting IL-4 locally to stimulate hepatocyte proliferation and support liver regeneration. It is possible that a low eosinophil count may adversely impact liver cell regeneration, though this study primarily involves liver tissue rather than blood, highlighting the need for expanded studies to clarify the mechanism.

While no differences in clinical symptoms, treatment duration, or antibiotic combinations were observed, the eosinopenia group experienced longer lengths of stay (LOS). Communication with attending physicians suggests that the older age and higher complication probability in the eosinopenia group necessitated cautious evaluation of treatment efficacy and side effects. Consequently, multiple evaluations and treatment adjustments, if necessary, extended the treatment duration.

Although CRP and PCT levels showed no differences between groups, an unexpected finding emerged regarding other inflammatory markers. Literature comparisons [21-27] identify potential diagnostic markers, with significant differences observed in MPV, MLR, and SIRI between groups. MPV, commonly used to gauge platelet function, typically decreases in severe brucellosis [21-23], aligning with this study's findings. Some research [23,24] indicates that high MLR levels are predictive of an elevated risk for osteoarticular and genitourinary involvement, although one study [21] contests this, suggesting controversy. In this study, MON

levels were significantly lower in the eosinopenia group, with no LY differences, possibly explaining the low MLR. Limited research on SIRI and brucellosis exists; one study [27] suggests SIRI lacks diagnostic value, indicating a need for further studies.

This study found that the eosinopenia group exhibited both low SIRI and low MLR in association with a poorer prognosis. It can be speculated that the eosinopenia group may exhibit lower inflammation levels, which contrasts with theories positing that severe inflammation correlates with disease severity. Given that indexes were measured approximately one-month post-infection, it can be inferred that inflammation initially peaks following Brucella infection and subsequently declines without influencing disease progression, likely due to brucellosis-related immune evasion.

This study serves as an initial exploration of the clinical manifestations of brucellosis in eosinopenia patients. While it does not elucidate underlying mechanisms, it lays a foundation for future research and provides substantive support for the diagnosis and treatment of brucellosis.

5. Conclusion

Eosinopenia is a common manifestation of brucellosis. Brucellosis patients with eosinopenia exhibited differences in clinical indicators and prognosis, though not in clinical symptoms. This suggests that eosinophils may serve as a risk factor for assessing prognosis. Monitoring eosinophils could improve prognosis assessment and present potential new treatment options.

Funding

This work was supported by the Suzhou Clinical Centre for Respiratory Infectious Diseases (Szlcyxzx202108) and the Suzhou Youth Science and Technology Project (KJXW2023050).

Disclosure statement

The authors declare no conflict of interest.

References

[1] Liu Z, Gao L, Wang M, et al., 2024, Long Ignored But Making A Comeback: A Worldwide Epidemiological Evolution of Human Brucellosis. Emerg Microbes Infect, 13(1): 2290839. https://doi.org/10.1080/22221751.2023.2290839

[2] National Health Commission of the People's Republic of China, 2023, China Health Statistical Yearbook (2022). China Union Medical College Press, Beijing.

[3] Jin M, Fan Z, Gao R, et al., 2023, Research Progress on Complications of Brucellosis. Front Cell Infect Microbiol, 13: 1136674. https://doi.org/10.3389/fcimb.2023.1136674

[4] Kaya S, Elaldi N, Deveci O, et al., 2018, Cytopenia in Adult Brucellosis Patients. Indian J Med Res, 147(1): 73–80. https://doi.org/10.4103/ijmr.IJMR_542_15

[5] Wang JN, Li BJ, Yuan J, et al., 2023, Brucellosis Complicated by Myelofibrosis: Report of Five Cases and Review of Literature. Int J Clin Exp Pathol, 16(7): 164–171.

[6] Guo M, Zhang L, Chen K, et al., 2024, Hemophagocytic Lymphohistiocytosis Associated with Brucellosis. Diagn Microbiol Infect Dis, 109(3): 116289. https://doi.org/10.1016/j.diagmicrobio.2024.116289

[7] Çelik M, Arslan Y, Topcu E, et al., 2024, Investigation of Hematologic Findings Related to Brucellosis in Anatolian Region. Saudi Med J, 45(5): 495–501. https://doi.org/10.15537/smj.2024.45.5.20230847

[8] Samarasinghe AE, Melo RC, Duan S, et al., 2017, Eosinophils Promote Antiviral Immunity in Mice Infected with Influenza A Virus. J Immunol, 198(8): 3214–3226. https://doi.org/10.4049/jimmunol.1600787

[9] Rosenberg HF, Foster PS, 2021, Eosinophils and COVID-19: Diagnosis, Prognosis, and Vaccination Strategies. Semin Immunopathol, 43(3): 383–392. https://doi.org/10.1007/s00281-021-00850-3

[10] Yang L, Zhang W, Shen X, et al., 2022, Predictive Role of Blood Eosinophils in Adult Varicella Patients. Epidemiol Infect, 150: e127. doi:10.1017/S095026882200111X

[11] Jiao PF, Chu WL, Ren GF, et al., 2015, Expression of Eosinophils Be Beneficial to Early Clinical Diagnosis of Brucellosis. Int J Clin Exp Med, 8(10): 19491–19495.

[12] Guo MY, Xu K, Cao Y, et al., 2021, Clinical Analysis of Brucellosis in 49 Cases in North Anhui Area. Journal of Tropical Diseases and Parasitology, 19(4): 219.

[13] Gnanaraj J, Parnes A, Francis CW, et al., 2018, Approach to Pancytopenia: Diagnostic Algorithm for Clinical Hematologists. Blood Rev, 32(5): 361–367. https://doi.org/10.1016/j.blre.2018.03.001

[14] Deniz S, Baykam N, Celikbas A, et al., 2015, Screening Household Members of Acute Brucellosis Cases in Endemic Areas and Risk Factors for Brucellosis. Vector Borne Zoonotic Dis, 15(8): 468–472. https://doi.org/10.1089/vbz.2014.1723

[15] Nematollahi S, Ayubi E, Karami M, et al., 2017, Epidemiological Characteristics of Human Brucellosis in Hamadan Province During 2009–2015: Results from the National Notifiable Diseases Surveillance System. Int J Infect Dis, 61: 56–61. https://doi.org/10.1016/j.ijid.2017.06.002

[16] Sayin Kutlu S, Kutlu M, Tuzun T, et al., 2018, Spondylodiscitis: A Common Complication of Brucellosis. J Infect Dev Ctries, 12(7): 550–556. https://doi.org/10.3855/jidc.10557

[17] Demirdal T, Sen P, 2020, Risk Factors for Focal Involvement in Brucellosis. Diagn Microbiol Infect Dis, 97(1): 115003. https://doi.org/10.1016/j.diagmicrobio.2020.115003

[18] Cisneros B, García-Aguirre I, Unzueta J, et al., 2022, Immune System Modulation in Aging: Molecular Mechanisms and Therapeutic Targets. Front Immunol, 13: 1059173. https://doi.org/10.3389/fimmu.2022.1059173

[19] Liu Z, Liang Q, Ren Y, et al., 2023, Immunosenescence: Molecular Mechanisms and Diseases. Signal Transduct Target Ther, 8(1): 200. https://doi.org/10.1038/s41392-023-01451-2

[20] Aoki A, Hirahara K, Kiuchi M, et al., 2021, Eosinophils: Cells Known for Over 140 Years with Broad and New Functions. Allergol Int, 70(1): 3–8. https://doi.org/10.1016/j.alit.2020.09.002

[21] Cift A, Yucel MO, 2018, Comparison of Inflammatory Markers Between Brucella and Non-Brucella Epididymo-Orchitis. Int Braz J Urol, 44(4): 771–778. https://doi.org/10.1590/s1677-5538.ibju.2018.0004.0

[22] Sen P, Demirdal T, Nemli SA, 2019, Predictive Value of Inflammation Markers in Brucellosis. Arch Iran Med, 22(11): 640–645.

[23] Aydin E, Karadag MA, Cecen K, et al., 2016, Association of Mean Platelet Volume and the Monocyte/Lymphocyte Ratio with Brucella-Caused Epididymo-Orchitis. Southeast Asian J Trop Med Public Health, 47(3): 450–456.

[24] Balın ŞÖ, Tartar AS, Akbulut A, 2018, The Predictive Role of Haematological Parameters in the Diagnosis of Osteoarticular Brucellosis. Afr Health Sci, 18(4): 988–994. https://doi.org/10.4314/ahs.v18i4.19

[25] Islam MM, Satici MO, Eroglu SE, 2024, Unraveling the Clinical Significance and Prognostic Value of the

Neutrophil-to-Lymphocyte Ratio, Platelet-to-Lymphocyte Ratio, Systemic Immune-Inflammation Index, Systemic Inflammation Response Index, and Delta Neutrophil Index: An Extensive Literature Review. Turk J Emerg Med, 24(1): 8–19. https://doi.org/10.4103/tjem.tjem_198_23

[26] Zhang J, Zhao Q, Liu S, et al., 2024, Clinical Predictive Value of the CRP-Albumin-Lymphocyte Index for Prognosis of Critically Ill Patients with Sepsis in Intensive Care Unit: A Retrospective Single-Center Observational Study. Front Public Health, 12: 1395134. https://doi.org/10.3389/fpubh.2024.1395134

[27] Şah V, Baran Aİ, 2024, Can Hematological Inflammatory Indices Be Used to Differentiate Modic Type 1 Changes from Brucella Spondylodiscitis? Medicina (Kaunas), 60(7): 1131. https://doi.org/10.3390/medicina60071131