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## ORIGINAL ARTICLE

# Analysis of the Formation of Eosinophilic Pleural Effusion

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### SUMMARY

***Background:*** **Eosinophilic pleural effusion constitutes a captivating and salient subject for exploration. In order to find out the reasons behind the formation of eosinophilic pleural effusion, we chose this study, hoping to gradually unravel its mysterious veil.**
***Methods:*** **The absolute value and percentage of eosinophils in healthy people (normal control group), the peripheral blood of patients, and the pleural effusion of patients were compared to analyze their characteristics.**
***Results:*** **In patients with eosinophilic pleural effusion, the absolute level of eosinophils was significantly greater than that in healthy people (normal control group) ($p < 0.0001$) and the percentage of eosinophils was significantly greater than that in healthy people ($p < 0.0001$) as well, even within the biological reference interval.**
***Conclusions:*** **Peripheral blood eosinophil counts were higher in patients with eosinophilic pleural effusion than in normal controls, even within the biological reference interval. Moreover, the overall absolute level of eosinophilic cells in the pleural effusion fluid was consistent with that in the peripheral blood. Combining these characteristics, the mechanism of eosinophilic pleural effusion formation was deduced.**
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### KEYWORDS

eosinophilic pleural effusion, eosinophils, percentage of eosinophils, peripheral blood eosinophils, pleural effusion

### INTRODUCTION

Eosinophilic pleural effusions (EPEs) are defined as pleural effusions in which the ratio of eosinophilic pleural effusions to leukocytes exceeds 10%, accounting for approximately 5% - 16% of exudative pleural effusions, with or without an increase in peripheral blood eosinophils [1]. Originally defined by Harmsen in 1894, it is associated with a variety of causes, including, but not limited to, parasitic infections, allergic diseases, drug reactions, and certain malignancies, 14% - 25% of which remain idiopathic eosinophilic pleural effusion. Eosinophilic pleural effusion, a special type of pleural effusion, has various clinical manifestations and lacks specificity, making early diagnosis difficult. Patients often present with chest pain, chest tightness, cough, fatigue, asthma, and dyspnea, and some patients present with fever and other related nonspecific clinical symp-

toms; thus, clinicians need to rely more on detailed clinical observations and comprehensive laboratory examinations when encountering such patients to make diagnoses and judgments. The increase in eosinophils in pleural effusion may be related to various pathological mechanisms [2,3] and, sometimes, medical factors [4-7]. Further exploration and study of these mechanisms are highly important for understanding the pathophysiology of EPE. Most studies suggest that the pathogenesis of EPE may involve the release of certain cytokines, such as interleukin-5 (IL-5), granulocyte/macrophage colony-stimulating factor, and interleukin-3 (IL-3). These factors increase the production and survival of eosinophils, stimulate the proliferation and differentiation of eosinophils through corresponding pathways, and increase eosinophil accumulation in the pleural effusion [8].

Therefore, what are the characteristics of eosinophil absolute values and eosinophil percentages in the peripheral blood of patients with eosinophilic pleural effusion? Eosinophil levels are elevated upon stimulation with interleukin 3, interleukin 5, and granulocyte/macrophage colony-stimulating factors. Eosinophils produced by the bone marrow are increased in the thoracic cavity, and the association between the thoracic cavity and the bone marrow is based on blood circulation. Based on this logic, do peripheral blood eosinophil levels increase in patients with eosinophilic pleural effusion? With this question in mind, we conducted research to verify this hypothesis.

## MATERIALS AND METHODS

### Patients

We entered the clinical research center of our hospital (the time period was from January 2001 to January 2024), selected "eosinophilic percentage (%) - pleural effusion > 10" as the search condition, and obtained data from 672 patients at the initial screening. If the same patient had multiple test data points, the data from the first test were used to eliminate interference after drug treatment. Simultaneously, complete clinical data, including medical history, physical examination, laboratory examination results, and definitive diagnostic data, were verified, and 495 valid data points were confirmed. There were 248 cases 10 years ago and 247 cases in the last 10 years. There were 342 male patients and 153 female patients, with a male-to-female ratio of 2.24:1, which was higher than that reported by Minfang Li's study (approximately 1.8:1) in 2021 [9]. The median age (25% quartile, 75% quartile) was 57 (43,92). Moreover, their etiology can be classified into the eight categories shown in Table 1 below. The top three etiologies were malignant tumors (43.6%), infections (22.6%), and idiopathic eosinophilic pleural effusions (15.5%). The main cause of the tumors was consistent with that reported in the literature [10,11].

### Abbreviation

To facilitate the description and illustration of the subsequent sections of this article, the relevant phrases have been abbreviated for convenience. A reference table is provided, as shown in Table 2 below.

### Methods

To better analyze the eosinophil count and percentage level of eosinophil pleural effusion, we selected 451 healthy male and 381 healthy female people as the normal control group to study the characteristics of eosinophil pleural effusion through comparisons under different conditions to explore its formation principle, specifically as follows:

1. The eosinophil count and percentage were compared between the 495 patients and the normal controls.
2. To verify the increase in eosinophils in patients compared with healthy people in the biological reference interval, the eosinophilic counts and percentages were compared.
3. To investigate whether there was a gender difference, the eosinophil counts and percentages in the biological reference interval were compared with those in the normal control group.
4. The pathological level of eosinophilic pleural effusion was confirmed by comparing the eosinophil counts and percentages in the pleural effusion, peripheral blood, and normal control groups.

### Statistical analysis

GraphPad Prism 9.5 was used for descriptive statistical analysis of the eosinophil count and eosinophil percentage in patients and healthy controls (negative control group). Normality tests were used to analyze the normality of the data to determine whether parametric tests were used. The Shapiro-Wilk test was used for the normality test. The Mann-Whitney test was used for two groups of unpaired nonparametric *t*-tests, and the Kruskal-Wallis test was used for comparisons of more than two groups.

## RESULTS

A comparison of data from 495 patients with eosinophilic pleural effusion and the normal control group revealed that the data did not conform to a normal distribution, except for peripheral blood hemoglobin with eosinophilic pleural effusion. The values of PB-EOS%, PB-EOS#, PB-WBC, and PB-PLT in patients with eosinophilic pleural effusion were as follows: 5.1 (2.8, 8.8), 0.4 (0.2, 0.6), 7.1 (5.6, 9.2), and 250 (191, 326), respectively; these values were extremely significantly higher than those of the normal control group ($p < 0.0001$). In contrast, the values of PB-RBC, PB-HGB, and PB-HCT (4.1 (3.6, 4.6), 122 (107, 136), and 36.9 (32.6, 40.8)) were significantly lower than those of the normal control group ($p < 0.0001$). For details, see Table 3 below.

**Table 1. Etiological distribution of eosinophilic pleural effusion.**

| ID | Etiology | Totality | Percentage (%) |
|---|---|---|---|
| 1 | Blood/Air in pleural space | 12 | 2.4 |
| 2 | Infection | 112 | 22.6 |
| 3 | Uremic pleuritis | 19 | 3.8 |
| 4 | Malignancy | 216 | 43.6 |
| 5 | Autoimmune disorder | 8 | 1.6 |
| 6 | Tuberculosis | 39 | 7.8 |
| 7 | Parasitic EPE | 12 | 2.4 |
| 8 | Idiopathic EPE | 77 | 15.5 |
| Total | | 495 | 100 |

**Table 2. Each analysis index abbreviation comparison table.**

| Item | Abbreviation |
|---|---|
| Percentage of eosinophils in peripheral blood | PB-EOS% |
| Percentage of eosinophils in negative control group | NCG-EOS% |
| Eosinophilic counts in peripheral blood | PB-EOS# |
| Eosinophilic counts in negative control group | NCG-EOS# |
| White blood cell in peripheral blood | PB-WBC |
| White blood cell in negative control group | NCG-WBC |
| Red blood cell in peripheral blood | PB-RBC |
| Red blood cell in negative control group | NCG-RBC |
| Hemoglobin in peripheral blood | PB-HGB |
| Hemoglobin in negative control group | NCG-HGB |
| Hematocrit in peripheral blood | PB-HCT |
| Hematocrit in negative control group | NCG-HCT |
| Platelet in peripheral blood | PB-PLT |
| Platelet in negative control group | NCG-PLT |
| Peripheral blood | PB |
| Negative control group | NCG |
| Eosinophilic pleural effusions | EPE |
| Eosinophilic percentage | Eos% |
| Eosinophilic count (absolute values) | Eos# |
| Pleural effusion | PE |
| Body fluid | BF |
| Parasite | PA |
| Non-significant | ns |

To verify Adelman's theory of “with or without an increase in peripheral blood eosinophils” [1], a design was used to compare the level of eosinophils in the biological reference interval between patients with eosinophilic pleural effusion and normal controls to analyze the characteristics of the two groups, as shown in Table 4 below. The biological reference interval of the eosinophilic percentage is 0.4 - 8%, and the biological reference interval of the eosinophilic count (absolute value) is 0.02 - 0.52. The data were derived from the Refer-

**Table 3. Comparison of the peripheral blood of patients with that of normal controls.**

| Item | Number of values | Shapiro-Wilk test | Minimum | 25% percentile | Median | 75% percentile | Maximum | p-value |
|---|---|---|---|---|---|---|---|---|
| PB-EOS% | 495 | < 0.0001 | 0 | 2.8 | 5.1 | 8.8 | 58.7 | < 0.0001 |
| NCG-EOS% | 832 | < 0.0001 | 0.4 | 1.2 | 1.9 | 3 | 10.7 | |
| PB-EOS# | 495 | < 0.0001 | 0 | 0.2 | 0.4 | 0.6 | 11.3 | < 0.0001 |
| NCG-EOS# | 832 | < 0.0001 | 0 | 0.1 | 0.1 | 0.2 | 0.5 | |
| PB-WBC | 495 | < 0.0001 | 1.3 | 5.6 | 7.1 | 9.2 | 67.3 | < 0.0001 |
| NCG-WBC | 832 | < 0.0001 | 3.5 | 4.9 | 5.7 | 6.7 | 9.4 | |
| PB-RBC | 495 | < 0.0001 | 1.6 | 3.6 | 4.1 | 4.6 | 8.3 | < 0.0001 |
| NCG-RBC | 832 | < 0.0001 | 3.8 | 4.5 | 4.8 | 5.1 | 5.7 | |
| PB-HGB | 495 | 0.083 | 53 | 107 | 122 | 136 | 179 | < 0.0001 |
| NCG-HGB | 832 | < 0.0001 | 116 | 133 | 143.5 | 153 | 167 | |
| PB-HCT | 495 | 0.0127 | 13.5 | 32.6 | 36.9 | 40.8 | 51.9 | < 0.0001 |
| NCG-HCT | 832 | < 0.0001 | 36.2 | 41.3 | 44.1 | 46.8 | 50 | |
| PB-PLT | 495 | < 0.0001 | 14 | 191 | 250 | 326 | 830 | < 0.0001 |
| NCG-PLT | 832 | < 0.0001 | 126 | 200 | 229 | 264 | 347 | |

**p-values were derived from the two-tailed *t*-test via the Mann-Whitney test.**

**Table 4. Comparison of peripheral blood samples from healthy controls within a biological reference interval.**

| Item | Number of values | Shapiro-Wilk test | Minimum | 25% percentile | Median | 75% percentile | Maximum | p-value |
|---|---|---|---|---|---|---|---|---|
| PB-EOS% | 355 | < 0.0001 | 0 | 2 | 3.8 | 5.5 | 7.9 | < 0.0001 |
| NCG-EOS% | 832 | < 0.0001 | 0.4 | 1.2 | 1.9 | 3 | 10.7 | |
| PB-EOS# | 355 | < 0.0001 | 0 | 0.2 | 0.3 | 0.4 | 1.1 | < 0.0001 |
| NCG-EOS# | 832 | < 0.0001 | 0 | 0.1 | 0.1 | 0.2 | 0.5 | |
| PB-WBC | 355 | < 0.0001 | 1.3 | 5.7 | 7.1 | 9.2 | 67.3 | < 0.0001 |
| NCG-WBC | 832 | < 0.0001 | 3.5 | 4.9 | 5.7 | 6.7 | 9.4 | |
| PB-RBC | 355 | < 0.0001 | 1.6 | 3.6 | 4.1 | 4.6 | 8.3 | < 0.0001 |
| NCG-RBC | 832 | < 0.0001 | 3.8 | 4.5 | 4.8 | 5.1 | 5.7 | |
| PB-HGB | 355 | 0.195 | 53 | 106 | 121 | 134 | 179 | < 0.0001 |
| NCG-HGB | 832 | < 0.0001 | 116 | 133 | 143.5 | 153 | 167 | |
| PB-HCT | 355 | 0.0408 | 13.5 | 32.5 | 36.7 | 40.3 | 51.9 | < 0.0001 |
| NCG-HCT | 832 | < 0.0001 | 36.2 | 41.3 | 44.1 | 46.8 | 50 | |
| PB-PLT | 355 | < 0.0001 | 14 | 189 | 256 | 331 | 830 | < 0.0001 |
| NCG-PLT | 832 | < 0.0001 | 126 | 200 | 229 | 264 | 347 | |

**p-values were derived from the two-tailed *t*-test via the Mann-Whitney test.**

ence Interval for Blood Cell Analysis, Health Industry Standard WS/T 405-2012 of the People's Republic of China.

Table 4 shows that each indicator has the same trend as Table 3. Specifically, the values of PB-EOS%, PB-EOS#, PB-WBC, and PB-PLT in patients with eosinophilic pleural effusion are 5.1 (2.8, 8.8), 0.4 (0.2, 0.6), 7.1 (5.6, 9.2), and 250 (191, 326), respectively, which are extremely significantly greater than those of the normal control group ($p < 0.0001$). In contrast, the values of PB-RBC, PB-HGB, and PB-HCT, which are 4.1 (3.6, 4.6), 122 (107, 136), and 36.9 (32.6, 40.8), respectively, are significantly lower than those of the normal control group ($p < 0.0001$). These data indicate that in patients

**Table 5. Comparison of males and females with normal controls within biological reference intervals.**

| Gender | Item | Number of values | Shapiro-Wilk test | Minimum | 25% percentile | Median | 75% percentile | Maximum | p-value |
|---|---|---|---|---|---|---|---|---|---|
| Male | PB-EOS% | 243 | 0.0006 | 0 | 2.4 | 4.2 | 5.6 | 7.9 | < 0.0001 |
| | NCG-EOS% | 451 | < 0.0001 | 0.4 | 1.3 | 2.1 | 3.2 | 8 | |
| | PB-EOS# | 243 | < 0.0001 | 0 | 0.17 | 0.29 | 0.4 | 1.08 | < 0.0001 |
| | NCG-EOS# | 451 | < 0.0001 | 0.02 | 0.07 | 0.12 | 0.19 | 0.51 | |
| | PB-WBC | 243 | < 0.0001 | 1.31 | 5.69 | 7.14 | 9.32 | 27.19 | < 0.0001 |
| | NCG-WBC | 451 | < 0.0001 | 3.5 | 4.98 | 5.82 | 6.78 | 9.41 | |
| Female | PB-EOS% | 112 | < 0.0001 | 0 | 1.325 | 2.95 | 5.2 | 7.8 | < 0.0001 |
| | NCG-EOS% | 381 | < 0.0001 | 0.4 | 1.2 | 1.7 | 2.8 | 10.7 | |
| | PB-EOS# | 112 | < 0.0001 | 0 | 0.1 | 0.18 | 0.34 | 0.76 | < 0.0001 |
| | NCG-EOS# | 381 | < 0.0001 | 0.02 | 0.07 | 0.09 | 0.15 | 0.52 | |
| | PB-WBC | 112 | < 0.0001 | 2.43 | 5.555 | 6.88 | 8.575 | 67.3 | < 0.0001 |
| | NCG-WBC | 381 | < 0.0001 | 3.5 | 4.76 | 5.6 | 6.59 | 9.16 | |

**p-values were derived from the two-tailed *t*-test via the Mann-Whitney test.**

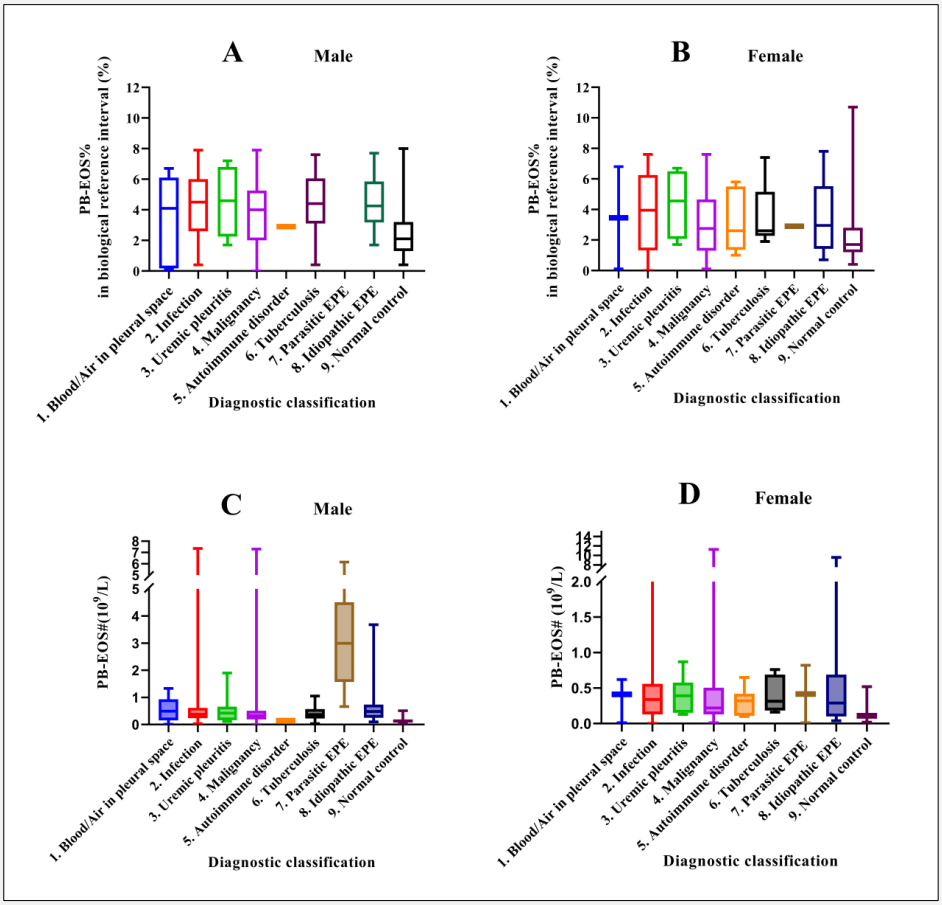


**Figure 1. Eos% in the biological reference interval and EOS# of the 8 categories of etiology.**

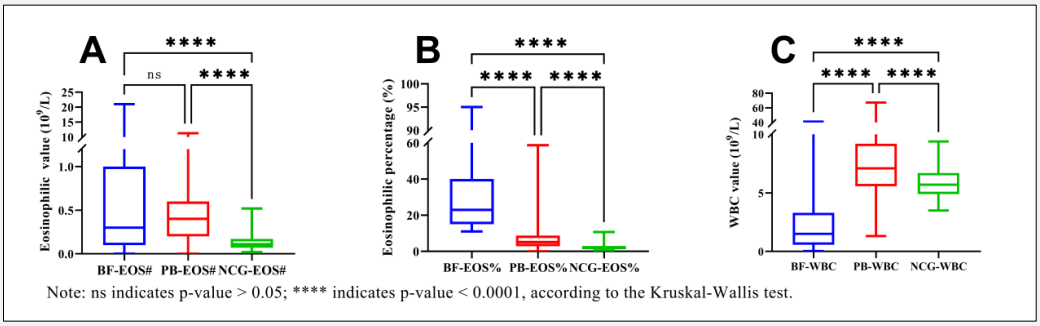


**Figure 2. Comparison of EOS%, EOS#, and WBC counts in the pleural fluid, peripheral blood, and normal controls.**

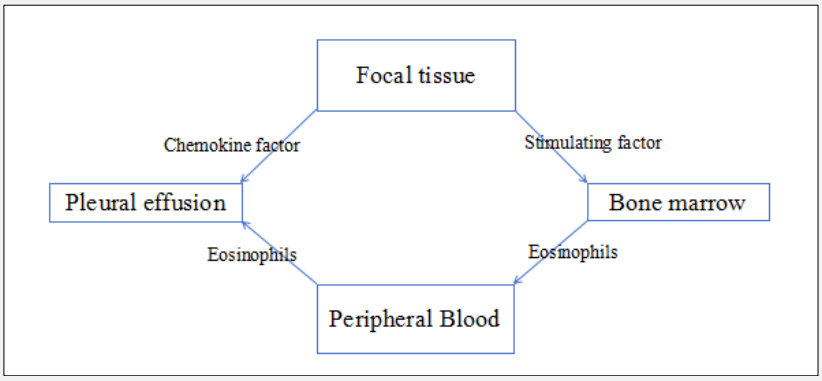


**Figure 3. Schematic diagram of the formation of eosinophilic pleural effusion.**

with eosinophilic pleural effusion, the so-called eosinophils (PB-EOS# and PB-EOS%) do not increase (that is, within the range of the biological reference interval); in fact, the level of eosinophils is higher than that of normal controls, but this level is within the biological reference interval, which makes it difficult to detect.

To clarify whether there were gender differences in the biological reference interval, patients in the biological reference interval were divided into male and female groups, and the two groups were compared with the normal control group. The PB-EOS%, PB-EOS#, and PB-WBC of the two groups showed the same regularity and were all higher than those of the normal control group. Moreover, the corresponding values for males were greater than those for females: 4.2 (2.4, 5.6), 0.29 (0.17, 0.4), and 7.14 (5.69, 9.32) vs. 2.95 (1.325, 5.2), 0.18 (0.1, 0.34), and 6.88 (5.555, 8.575), respectively. The details are shown in Table 5.

In the biological reference interval, the percentage of eosinophils was greater than that in the normal control group. To clarify more detailed changes, the percentage of eosinophils with different etiologies was calculated by gender as a grouping variable, as shown in Figure 1 (A for males and B for females). The percentages of eosinophils in patients with 8 etiologies were greater than those in the normal control group, and the differences were more significant in the male parasite group. The data here were zero (there is no bar chart), indicating that the percentage of eosinophils in male parasite patients was greater than the biological reference interval threshold of 8%. To gain a clearer understanding of the level of eosinophils, we further calculated the count of eosinophils in the male and female groups, as shown in Figure 1 (C for male and D for female). According to the graph, the eosinophil count in the male parasite group was greater than that in the other etiological groups, whereas this feature was not obvious in the female group.

To understand the process of accumulation and elevation of eosinophils in the thoracic cavity, it is necessary to compare the eosinophil count, as well as the percentage of eosinophils, in the pleural effusion, peripheral blood, and normal control samples under the three conditions. Since the percentage eosinophils is based on white blood cells, the comparison of white blood cells is also essential. All the data are shown in A, B, and C in Figure 2.

As shown in the figure, there was no significant difference in the eosinophilic count between the PEs and peripheral blood (ns, non-significant), whereas the other states were extremely significantly different ($p < 0.0001$).

## DISCUSSION

Eosinophilic pleural effusions (EPE) are defined as pleural effusions in which the ratio of eosinophilic pleural effusions to leukocytes exceeds 10%, accounting for approximately 5% - 16% of exudative pleural effusions, with or without an increase in blood eosinophilic effusions. As mentioned previously, to complete the delivery of eosinophils in eosinophilic pleural effusion, blood is required as a transport channel. Following this logic, we compared the percentages of eosinophils, eosinophils, white blood cells, red blood cells, hemoglobin, hematocrit, and platelets in the peripheral blood of patients with those in patients with eosinophilic pleural effusion. The percentage of eosinophils in the peripheral blood of patients was 5.1 (2.8, 8.8), which was significantly greater ($p < 0.0001$) than that in the negative control group (1.9 (1.2, 3.0)). The eosinophil count in the peripheral blood of the former was 0.4 (0.2, 0.6), which was significantly greater ($p < 0.0001$) than that of the negative control group (0.1, 0.2). This result is obvious and in line with Adelman's description of "with an increase in blood eosinophilic effusions" [1]. More detailed data are described in Table 3.

To test Adelman's claim of "without an increase in blood eosinophilic effusions" [1], we compared eosinophilic levels in the biological reference interval between patients and negative control groups. The percentage of eosinophils in the peripheral blood of patients with eosinophilic pleural effusion was significantly greater ($p < 0.0001$) than that in the negative control group (3.8 (2, 5.5) compared with 1.9 (1.2, 3.0)). The former had an eosinophil count of 0.3 (0.2, 0.4) and was significantly greater ($p < 0.0001$) than the latter, which had an eosinophil count of 0.1 (0.1, 0.2). In other words, in the biological reference interval, the peripheral blood level (percentage and count) of patients is also higher than that of healthy people; however, in this state, because the level is within the range of 8% of the biological reference interval, it cannot be easily detected. To capture the more subtle differences between the two groups, the collected patients were distinguished by gender, and the differences were compared between the two groups. For males, the percentage of eosinophils in the peripheral blood of patients was 4.2% (2.4, 5.6%), which was significantly greater ($p < 0.0001$) than the 2.1% (1.3, 3.2%) of the healthy population in the biological reference range (0.4% - 8%). The eosinophil count in patients was 0.29 (0.17, 0.4), and that in healthy people was 0.12 (0.07, 0.19). The former was significantly greater than the latter ($p < 0.0001$). For females, a similar pattern was observed, with an eosinophilic percentage of 2.95 (1.33, 5.2) compared with 1.7 (1.2, 2.8) and an eosinophilic count of 0.18 (0.1, 0.34) compared with 0.09 (0.07, 0.15); both differences were statistically significant ($p < 0.0001$). For more details, see Table 4 and Table 5. In short, patients with eosinophilic pleural effusion also have higher than normal levels of eosinophils within the biological reference interval. During the whole process, the increase in eosinophils was stimulated and the generation of erythroid cells was inhibited, which was the reason why several indexes that related to erythroid cells, such as RBC, HGB, and HCT, were lower than those of the normal control group.

In addition to analyzing the characteristics of eosinophilic pleural effusion from the perspective of gender, it is also necessary to analyze the characteristics of eosinophils under each etiology. A total of 495 patients with a definite diagnosis of PE were included, and the etiology was divided into eight categories: 1) blood/air in the pleural space, 2) infection, 3) uremic pleuritis, 4) malignancy, 5) autoimmune disorder, 6) tuberculosis, 7) parasitic EPE, and 8) idiopathic EPE. Among the eight categories of etiology, malignant tumors are still the main cause, accounting for 43.6% of the total cases, which is consistent with previous reports [10-12]; the details can be found in Table 1. Notably, tuberculosis infection and parasitic infection are two common categories of infection, so they are separated from infection and divided into two groups [13-15]. The cases of parasitic infection identified in this study were all caused by lung fluke infection. It is also an operation that has obtained unexpected effects. The percentage of eosinophils in the peripheral blood of male patients with parasitic infection was greater than 8%, and the percentage of eosinophils in the peripheral blood of male patients with parasitic infection was also significantly greater ($p < 0.0001$) than that in the peripheral blood of the other groups, which can be intuitively reflected in Figure 1.

To understand the cause of eosinophilic pleural effusion, we also needed to compare the eosinophilic levels of pleural effusion and peripheral blood and their changes relative to those of normal controls. Following this logic, we compared the EOS%, EOS#, and WBC counts in the pleural effusion, peripheral blood, and normal control samples, as shown in Figure 2. Combined with the characteristics of the data, we conducted a nonparametric analysis (using the Kruskal-Wallis test) and detected extremely significant differences, except for EOS# in the pleural effusion and peripheral blood. As reported by Ioannis Kalomenidis et al., VCAM-1 contributes to the accumulation of eosinophils in the pleural

effusion, which may explain why there is no difference in the EOS# between the pleural effusion and peripheral blood [16].

Based on these statistical data, we deduced the formation process of eosinophilic pleural effusion. Specifically, it will be explained with reference to Figure 3.

The lesions produce eosinophilic stimulators and chemokines due to the eight etiologies summarized above, and the stimulating factors enter the bone marrow to stimulate the bone marrow to produce eosinophils at higher-than-normal levels and release these eosinophils into the blood; then, the eosinophils enter the pleural effusion under the action of chemokines. Since the overall level of eosinophils was greater than that of the normal control group, eosinophils with higher-than-normal levels arrived at the chest cavity from the bone marrow through the peripheral blood during the formation of eosinophils in the pleural effusion and peripheral blood, and there was no difference in the EOS# between the pleural effusion and peripheral blood. Thus, eosinophils can be inferred to enter and exit the pleural effusion freely. This "freedom" also verified that the level of eosinophils in the peripheral blood of patients with eosinophilic pleural effusion was greater than that of normal controls, because if this requirement could not be met, the role of chemokines in the pleura must be to ensure that the number of eosinophils entering is greater than the number of eosinophils flowing out to increase the number of eosinophils in the chest. The level of white blood cells in the chest was lower than that in the peripheral blood, indicating that the chest blocks the entry of other cells, with the exception of eosinophils, into the chest, resulting in an increase in eosinophils in the pleural effusion. This process was most significant in male patients with parasitic infections.

At this point, we clearly identified the reason for the eosinophilic increase in eosinophilic pleural effusion through the changes in EOS#, EOS%, and WBC levels in the pleural cavity, peripheral blood, and normal control groups.

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**Ethical Approval:**
The study protocol was approved by the Tongji Medical College Ethics Committee for Research in Health at Tongji Hospital and complied with all relevant national regulations and institutional policies. As a retrospective study, visa-free informed consent was adopted.

**Declaration of Interest:**
The authors declare that they have no affiliations with or involvement in any organization or entity with any financial interest in the subject matter or materials discussed in this manuscript.

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