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*Review Article*

# Association between platelet-to-lymphocyte ratio and gastrointestinal bleeding in Henoch-Schönlein purpura: a meta-analysis

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**Abstract:** Objective: To assess systematically the association between platelet-to-lymphocyte ratio (PLR) and gastrointestinal bleeding in Henoch-Schönlein purpura (HSP) patients. Methods: Comprehensive online searches were conducted in mainstream Chinese and English online databases to collect studies related to gastrointestinal bleeding in patients with HSP and extract the reported PLR data. The literature was independently evaluated by two investigators according to predefined criteria, and the quality of the included studies was assessed by the Newcastle-Ottawa Scale (NOS). Meta-analysis was conducted using RevMan 5.3 software. The mean difference (MD) with 95% confidence interval (CI) was used as the effect size. Heterogeneity was evaluated by Cochran's Q test and the I-squared statistic. Publication bias was assessed using funnel plots and Egger's test. Results: Twenty-three studies were ultimately enrolled, consisting of 3,797 patients. The NOS scores ranged from 5 to 7 (median: 5). The meta-analysis results showed that PLR exhibited a significant positive association with gastrointestinal involvement in HSP patients (23 studies; MD = 26.24, 95% CI: [22.56, 29.92]; Z = 13.97, $I^2$ = 19%, P<0.001) and also showed a significant positive correlation with gastrointestinal bleeding in HSP patients (14 studies; MD = 26.17, 95% CI: [22.22, 30.13]; Z = 12.98, $I^2$ = 27%, P<0.001). These findings were consistent in both pediatric and adult populations. Conclusions: An elevated PLR serves as a risk indicator for gastrointestinal involvement and gastrointestinal bleeding in HSP patients.

**Keywords:** Platelet-to-lymphocyte ratio, Henoch-Schönlein purpura, gastrointestinal bleeding, systematic review, meta-analysis

## Introduction

Henoch-Schönlein purpura (HSP) is the most prevalent systemic small-vessel vasculitis in children and is less commonly seen in adults. Its main clinical manifestations include skin purpura, gastrointestinal mucosal bleeding, arthralgia, and renal injury [1]. Gastrointestinal involvement is common among patients, and severe cases can develop gastrointestinal bleeding (GIB) [2]. Studies have indicated that the incidence of GIB among pediatric HSP patients can range from 20% to 50%, and bleeding symptoms are often insidious. Without timely identification and intervention, some children may progress to acute abdominal conditions such as intussusception or even perforation [3]. Specific laboratory indicators for predicting GIB in pediatric HSP patients remain lacking. Therefore, identifying objective and readily available biomarkers that can provide early warning of GIB in children with HSP are needed to improve patient prognosis.

In recent years, the platelet-to-lymphocyte ratio (PLR) has garnered increasing attention from researchers, which is a derivative index derived from routine blood tests that comprehensively reflects the body's inflammatory state and immune imbalance. The pathologic basis of HSP involves extensive small-vessel vasculitis, and the inflammatory response can simultane-

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ously promote platelet activation and proliferation as well as a relative depletion of lymphocytes, leading to an abnormal elevation of PLR. Existing evidence suggests that in children with HSP, PLR level is significantly associated with disease activity and a risk of organ involvement [4]. To date, several clinical studies have investigated the association between PLR and GIB in HSP. Multiple studies have shown that PLR has predictive efficacy for GIB, but the reported predictive performances vary considerably, largely due to single-center designs and small sample sizes, and a unified conclusion is lacking. To clarify the overall association between PLR and GIB in HSP, this study aims to conduct a systematic review and meta-analysis of relevant domestic and international literature, thereby providing a robust evidence-based reference for clinicians to identify early high-risk populations.

## Materials and methods

### *Literature search strategy*

The databases searched included CNKI, Wanfang Data Knowledge Service Platform, VIP Chinese Journal Service Platform, PubMed, Wiley, and Google Scholar. A combination of MeSH terms and free-text terms was used for the search. The Chinese search strategy was: ('platelet-to-lymphocyte ratio' OR PLR) AND (Henoch-Schönlein purpura OR HSP) AND (gastrointestinal bleeding OR digestive tract bleeding OR esophageal bleeding OR gastric bleeding OR intestinal bleeding OR hematemesis OR melena) AND (prediction OR influence). The English search strategy was: ((platelet to lymphocyte ratio) OR (PLR)) AND ((Henoch Schonlein purpura) OR (Henoch Schoenlein purpura) OR (HSP) OR (allergic purpura) OR (anaphylactoid purpura) OR (Henoch purpura) OR (Henoch-Schoenlein purpura) OR (Henoch-Schonlein purpura) OR (purpura hemorrhagica) OR (nonthrombocytopenic purpura) OR (nonthrombopenic purpura) OR (rheumatoid purpura) OR (Schoenlein-Henoch purpura) OR (Schonlein-Henoch purpura) OR (Schonlein Henoch purpura) OR (hemorrhagic vasculitis) OR (IgA vasculitis)) AND ((((esophageal) OR (duodenal) OR (intestinal) OR (colonic) OR (rectal) OR (gastrointestinal) OR (gastrointestinal tract) OR (GI tract) OR (digestive tract)) AND ((hemorrhage) OR (bleeding))) OR (gastrorrhagia) OR (hematochezia) OR (abdominal pain) OR (colicky pain) OR (haematemesis)). The PROSPERO registration number is CRD420261413920.

### *Inclusion and exclusion criteria*

Inclusion criteria: According to meta-analysis principles [5], the inclusion criteria for this study were formulated. (1) Study population: Patients of any age with a diagnosis of HSP. (2) Outcome measures: A subset of patients in the included studies must have been clearly diagnosed with gastrointestinal involvement or bleeding. (3) Laboratory examination: The PLR of patients must have been reported as a continuous variable. (4) Publication period: Studies published in the past 20 years (2007-2026), to capture the most relevant and contemporary evidence [6]. (5) Sample size: The number of patients included was more than 60, to minimize the disproportionate influence of very small studies on the pooled estimates and to ensure a minimum level of statistical reliability [7].

Exclusion criteria: (1) Reviews (including meta-analyses), non-cohort, non-case-control studies, and non-human clinical studies. (2) Inappropriate research methods, study results not involving PLR, or failure to report results grouped by gastrointestinal involvement or bleeding. (3) Duplicate publications, articles whose full text could not be obtained, or articles with incomplete data. (4) Anomalous data. (5) Unpublished dissertations that have not undergone peer review, or abstracts, indices, and other non-original research articles. (6) Low quality research articles.

### *Data extraction and quality evaluation*

Literature collection, data extraction, and quality assessment were independently conducted by two researchers, with cross-checking performed afterward. Any disagreements were resolved through consensus discussion or third-party adjudication. The extracted content was as follows: (1) Basic information (first author, publication year, article title, source, study type); (2) Study population (sample size, age, disease diagnostic criteria, inclusion and exclusion criteria); (3) Outcome measures (definition of outcome, assessment tool, number of patients with and without gastrointestinal involve-

ment/bleeding); (4) Others (statistical methods, risk factors involved). For quality assessment of cohort studies and case-control studies, NOS [8] was adopted. This scale consists of two parts, respectively applicable to evaluating cohort studies and case-control studies. Every section comprises 8 items, covering three dimensions: selection of study groups, comparability of groups, and ascertainment of exposure or outcome. A semi-quantitative star system is used to rate the risk of bias, with one point awarded for meeting the criterion, for a maximum of 9 points. Literature with a total score ≥6 was considered to be of high quality, and literature with a total score <4 was considered to be of low quality and eliminated.

*Statistical methods*

Statistical analyses were carried out employing Review Manager 5.3. For studies that reported PLR as median (interquartile range), the method described by Wan et al. [9] was used to estimate the mean and standard deviation. For continuous variables, the mean difference (MD) or standardized mean difference (SMD) served as the effect size estimate. For dichotomous variables, the odds ratio (OR) was adopted as the effect size measure, with its 95% confidence interval (CI) computed. Inter-study heterogeneity was evaluated via the Cochran's Q test and quantified by the $I^2$ statistic. When heterogeneity was within acceptable limits ($P>0.1$, $I^2<50\%$), a fixed-effects model was applied. In the presence of substantial heterogeneity ($P<0.1$, $I^2>50\%$), the reasons for heterogeneity were explored. If no clinical heterogeneity was identified, a random-effects model was adopted. When clinical heterogeneity existed, subgroup analyses were conducted according to its origin, or sensitivity analyses were performed by alternating between random-effects and fixed-effects models and by omitting studies one by one. If heterogeneity was too high, a descriptive analysis was performed. Funnel plots were generated and Egger's test was conducted to evaluate publication bias. A *P*-value <0.05 was considered significant.

## Results

*Search results*

A preliminary search yielded 641 relevant articles, of which 173 were retrieved from CNKI, 15 from Wanfang, 26 from VIP, 18 from PubMed, 368 from Wiley, and 41 from Google Scholar. First, 35 duplicates were removed using EndNote X9 literature management software. Subsequently, one record identified as ineligible by automation tools was excluded, and 10 articles were excluded due to inappropriate article types (n = 8) or missing information (n = 2), leaving 595 articles. During the screening phase, 15 reviews and meta-analyses, 14 non-human clinical studies, and 10 articles with inappropriate research methods were excluded. Following review of titles and abstracts, a further 393 articles were excluded. The full texts of the remaining 163 articles were obtained. Two articles for which the full text could not be accessed were excluded. The remaining 161 articles were read in full, resulting in the exclusion of 118 articles that did not address HSP, gastrointestinal involvement, or bleeding, or did not measure PLR; 3 articles that did not report PLR grouped by gastrointestinal involvement or bleeding; 5 articles with duplicate data (dissertations subsequently published as journal articles) or anomalous data; 12 articles (dissertations) of low quality (NOS score <4) or without peer review. Ultimately, 23 studies met the inclusion criteria [10-32]. The literature screening workflow is shown in **Figure 1**.

*Basic characteristics and NOS scores of included studies*

The 23 studies ultimately enrolled consisted of 11 English-language and 12 Chinese-language publications, encompassing 3797 patients in total. The NOS scores ranged from 5 to 7 (median: 5). The quality of the included studies was considered moderate to high. The basic characteristics and NOS scores of the enrolled studies are presented in **Table 1**.

*Meta-analysis of PLR and gastrointestinal involvement in HSP*

All 23 studies [10-32] reported data on gastrointestinal involvement in HSP patients and PLR data for different subgroups. PLR values were reported in the same unit (ratio) across all included studies, and therefore the mean difference (MD) was used as the appropriate effect size measure. The heterogeneity test results showed that the Cochran's Q test was not statistically significant ($P = 0.20$), and $I^2 = 19\% <50\%$; therefore, the studies were consid-

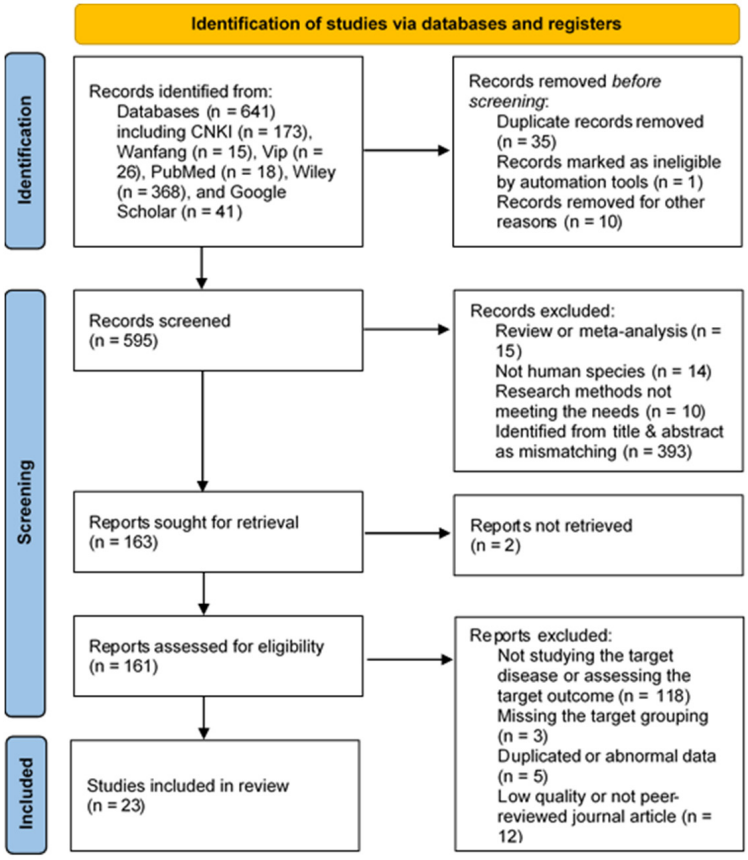



**Figure 1.** PRISMA flow diagram.

ered to show acceptable homogeneity, and a fixed-effects model was applied. The pooled analysis revealed a significant association between PLR and gastrointestinal involvement in HSP patients, with elevated PLR identified as a risk factor for gastrointestinal involvement in HSP patients. The pooled effect size was significant (Z = 13.97, P<0.001), as illustrated in **Figure 2**.

Subgroup analysis was performed to address patient population as a potential confounding factor. The studies were divided into a children group, an adult group, and a mixed children and adult group. Homogeneity within the three subgroups was good (P = 0.26, 0.20, 0.86; $I^2$ = 16%, 35%, 0%), and fixed-effects models were applied for all subgroups. The findings indicated that PLR demonstrated an association with gastrointestinal involvement in HSP in children (Z = 13.18, P<0.001), adults (Z = 4.07, P<0.001), and the mixed group (Z = 2.88, P = 0.004). The inter-subgroup difference was not significant (Cochran's Q = 3.50, P = 0.17, $I^2$ = 42.9%), as depicted in **Figure 3**. Therefore, elevated PLR served as a risk factor for gastrointestinal involvement in HSP patients, with minimal heterogeneity in the magnitude of its effect across different age groups.

Subgroup analysis was also performed to address publication source as a potential confounding factor. The studies were divided into a Chinese group and an English group. Homogeneity within the subgroups was good (P = 0.10, 0.47; $I^2$ = 35%, 0%), and fixed-effects models were applied for all subgroups. The findings indicated that PLR demonstrated an association with gastrointestinal involvement in HSP in both Chinese research articles (Z = 8.74, P<0.001) and English research articles (Z = 10.91, P<0.001). The inter-subgroup difference was not significant (Cochran's Q = 0.28, P = 0.60, $I^2$ = 0%), as depicted in **Figure 4**.

*Meta-analysis of PLR and gastrointestinal bleeding in HSP*

Among the 23 studies, 14 articles [11, 13, 14, 17, 18, 20, 24-28, 30-32] provided data on GIB in HSP patients and PLR data for different subgroups (one article reported data separately for both pediatric and adult patients). The heterogeneity test results showed good homogeneity among the studies (P = 0.13, $I^2$ = 27%), and a fixed-effects model was applied. The pooled analysis revealed a significant association between PLR and GIB in HSP patients, with elevated PLR constituting a risk factor for GIB in HSP patients. The pooled effect size was significant (Z = 12.98, P<0.001), as shown in **Figure 5**.

Subgroup analysis was performed to address patient population as a potential confounding factor. The studies were divided into a children group, an adult group, and a mixed children and adult group. Homogeneity within the children group and the mixed group was good (P = 0.29, 0.86; $I^2$ = 16%, 0%), while the two studies in the

**Table 1.** Basic characteristics of included studies

| First Author & Publication Year | Language | Patient Population | Sample Size [Total (GI/C)] | Grouping | | NOS Scores (Points) |
|---|---|---|---|---|---|---|
| | | | | GI Group | Control Group | |
| Caglayan 2025 [10] | English | Children | 109 (34/75) | HSP with severe GII | HSP with non-severe GII | 5 |
| Cai 2023 [11] | Chinese | Children | 82 (41/41) | HSP with GIB | HSP without GIB | 5 |
| Ekinci 2019 [12] | English | Children | 214 (77/137) | HSP with severe GII | HSP without severe GII | 6 |
| Gayret 2016 [13] | English | Children | 119 (41/78) | HSP with GIB | HSP without GIB | 6 |
| Hong 2018 [14] | English | Children | 141 (15/126) | HSP with GIB | HSP without GIB | 5 |
| Hu 2020 [15] | Chinese | Children | 117 (38/64) | HSP with GII | HSP without GII | 6 |
| Karadag 2020 [16] | English | Children | 215 (50/165) | HSP with severe GII | HSP without GII | 5 |
| Liao 2019 [17] | Chinese | Children | 121 (33/88) | HSP with GIB | HSP without GIB | 5 |
| Liu 2022 [18] | Chinese | Children | 308 (74/234) | HSP with GIB | HSP without GIB | 6 |
| Liu 2023 [19] | English | Children | 79 (39/40) | HSP with GII | HSP without GII | 5 |
| | | Adults | 79 (26/53) | HSP with GII | HSP without GII | |
| Ma 2021 [20] | Chinese | Children | 51 (9/42) | HSP with GIB | HSP without GIB | 7 |
| | | Adults | 60 (16/44) | HSP with GIB | HSP without GIB | |
| Oksel 2024 [21] | English | Children | 207 (100/107) | HSP with GII | HSP without GII | 5 |
| Onder 2022 [22] | English | Children | 95 (22/73) | HSP with GII | HSP without GII | 5 |
| Shao 2023 [23] | English | Adults | 158 (26/132) | HSP with GII | HSP without GII | 5 |
| Wang 2016 [24] | Chinese | Adults | 115 (28/87) | HSP with GIB | HSP without GIB | 5 |
| Wang 2023 [25] | Chinese | Children | 124 (48/76) | Abdominal HSP with GIB | Abdominal HSP without GIB | 5 |
| Wei 2024 [26] | Chinese | Children+Adults | 113 (42/71) | Abdominal HSP with GIB | Abdominal HSP without GIB | 5 |
| Yang 2024 [27] | English | Children | 112 (62/50) | HSP with GIB | HSP without GIB | 5 |
| Yin 2025 [28] | Chinese | Children | 516 (112/404) | HSP with GIB | HSP without GIB | 5 |
| Yuan 2021 [29] | English | Children | 115 (34/81) | HSP with GII | HSP without GII | 5 |
| Zhang 2018 [30] | Chinese | Children | 201 (28/173) | HSP with GIB | HSP without GIB | 6 |
| Zhang 2022 [31] | Chinese | Children | 248 (111/137) | HSP with GIB | HSP without GIB | 6 |
| Zhang 2024 [32] | Chinese | Children+Adults | 98 (67/31) | Abdominal HSP with GIB | Abdominal HSP without GIB | 5 |

Note: GI: gastrointestinal involvement or bleeding group; C: control group; HSP: Henoch-Schönlein purpura; GII: gastrointestinal involvement; GIB: gastrointestinal bleeding.

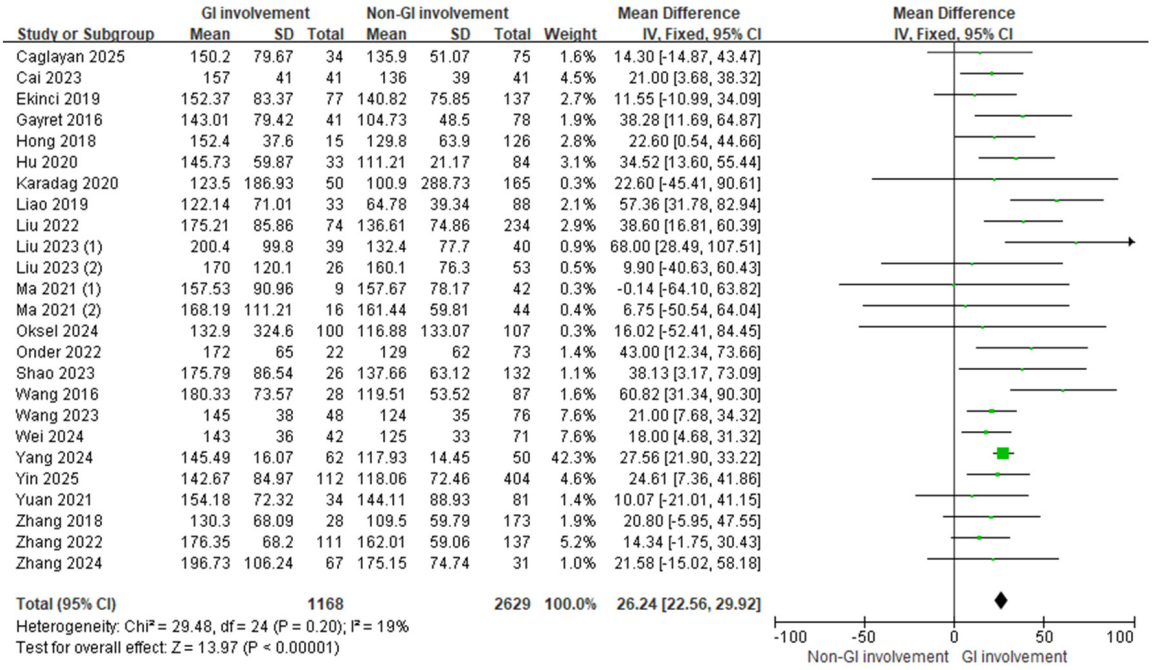



**Figure 2.** Forest plot of platelet to lymphocyte ratio and gastrointestinal involvement.

adult group showed some heterogeneity (P = 0.10, $I^2$ = 63%); nevertheless, a fixed-effects model was applied. The results showed that PLR had an association with GIB in HSP in children (Z = 12.28, P<0.001), adults (Z = 3.70, P<0.001), and the mixed group (Z = 2.88, P = 0.004). The inter-subgroup difference was not significant (Cochran's Q = 4.53, P = 0.10, $I^2$ = 55.9%), as shown in **Figure 6**. Therefore, elevated PLR also serves as a risk factor for GIB in HSP patients, and the magnitude of its effect is relatively consistent across different age groups.

Subgroup analysis was also performed to address publication source as a potential confounding factor. The studies were divided into a Chinese group and an English group. Homogeneity within the groups was good (P = 0.09, 0.66; $I^2$ = 38%, 0%), and a fixed-effects model was applied. The results showed that PLR had an association with GIB in HSP in both Chinese research articles (Z = 8.17, P<0.001) and English research articles (Z = 10.11, P<0.001). The inter-subgroup difference was not significant (Cochran's Q = 0.68, P = 0.41, $I^2$ = 0%), as shown in **Figure 7**.

*Publication bias*

Funnel plots were employed to assess publication bias. In this study, funnel plots were generated for both the analysis of PLR and gastrointestinal involvement and the analysis of PLR and GIB. The funnel plots were generally symmetric, indicating a low likelihood of publication bias, as presented in **Figures 8**, **9**. Egger's test was also conducted, and no significant results were found for either the analysis of PLR and gastrointestinal involvement or the analysis of PLR and GIB (P>0.05).

## Discussion

By integrating data derived from 23 studies involving a combined total of 3797 cases, this meta-analysis provided the first comprehensive evaluation to date of the association between PLR and gastrointestinal complications among HSP patients. The pooled results revealed significantly higher PLR levels in HSP patients with gastrointestinal involvement and GIB compared to those without such complications. This finding supports the potential clinical utility of PLR, a simple and readily available derivative index from routine blood tests, in the early identification of high-risk gastrointestinal events in HSP patients. This investigation strictly followed the inclusion and exclusion criteria for literature screening. The quality of the enrolled research articles was identified as moderate or above, which confers good reliability.

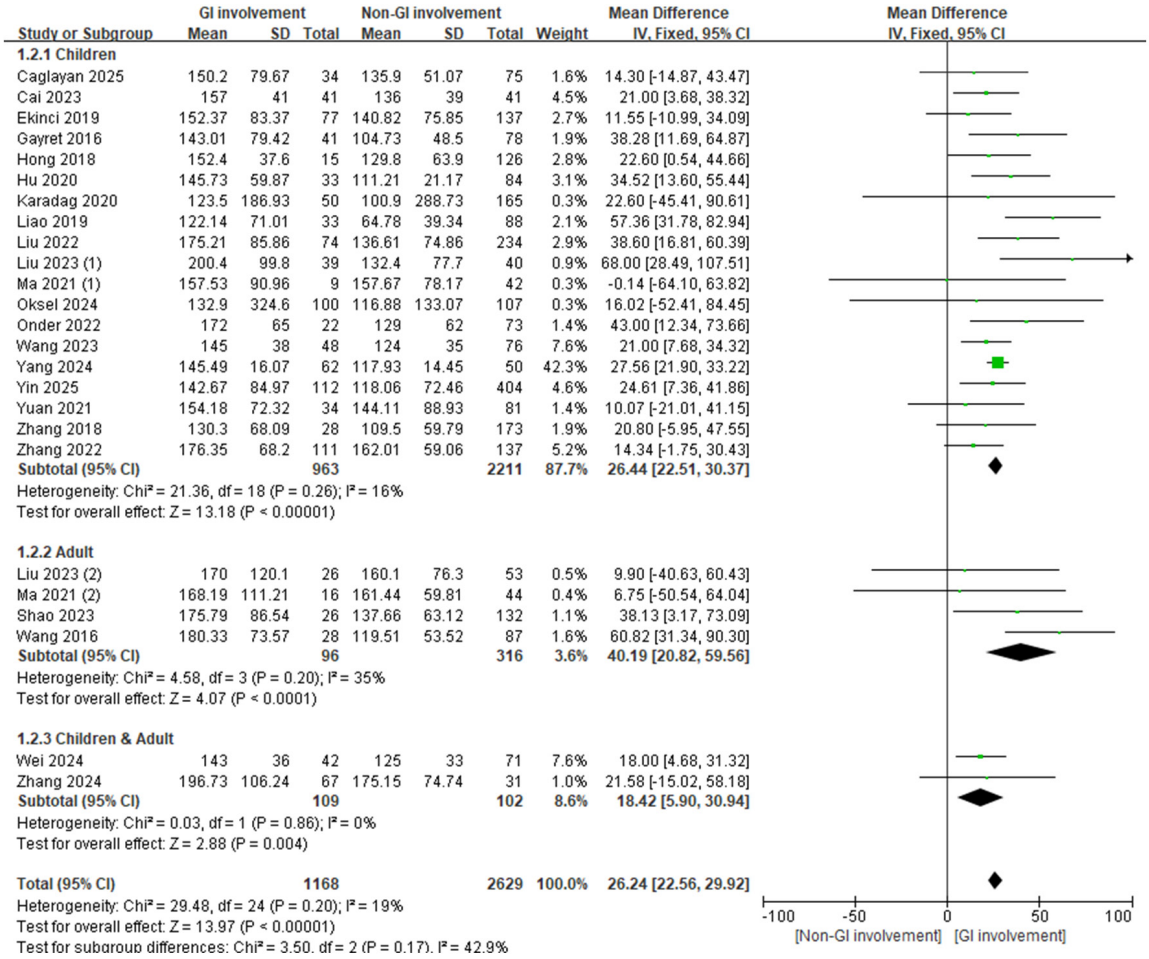



**Figure 3.** Forest plot of platelet to lymphocyte ratio and gastrointestinal involvement (population subgroup analysis).

The association between PLR and gastrointestinal complications in HSP has a plausible pathophysiologic basis. HSP is fundamentally an immune complex-mediated systemic small-vessel vasculitis, in which inflammation plays a central role in its initiation and progression [33]. The deposition of IgA-containing immune complexes in the walls of small blood vessels triggers complement activation, leading to vascular endothelial injury and increased vascular permeability, particularly in the gastrointestinal tract, where the rich capillary network is highly susceptible to inflammatory damage. In this inflammatory milieu, platelets act not only as key effector cells in hemostasis but also as active participants in amplifying the inflammatory cascade. Activated platelets express adhesion molecules such as P-selectin and glycoprotein IIb/IIIa, release pro-inflammatory mediators including platelet-derived growth factor, thromboxane A2, and serotonin, and facilitate leukocyte recruitment to sites of vascular injury [34]. This platelet-mediated inflammatory amplification may be particularly relevant in the gastrointestinal vasculature, where the dense microvascular network is vulnerable to immune complex deposition.

Concurrently, children with HSP often exhibit an imbalance in T lymphocyte subsets and immune dysfunction, manifesting as a relative decrease in peripheral blood lymphocyte counts [35]. The reduction in lymphocyte counts may reflect the migration of lymphocytes to sites of active inflammation, immune-mediated lymphocyte apoptosis, or the immunosuppressive effects of systemic inflammation. The ratio of platelet count to lymphocyte count (PLR) can comprehensively reflect the dual state of pro-inflammatory pathway activation and immune

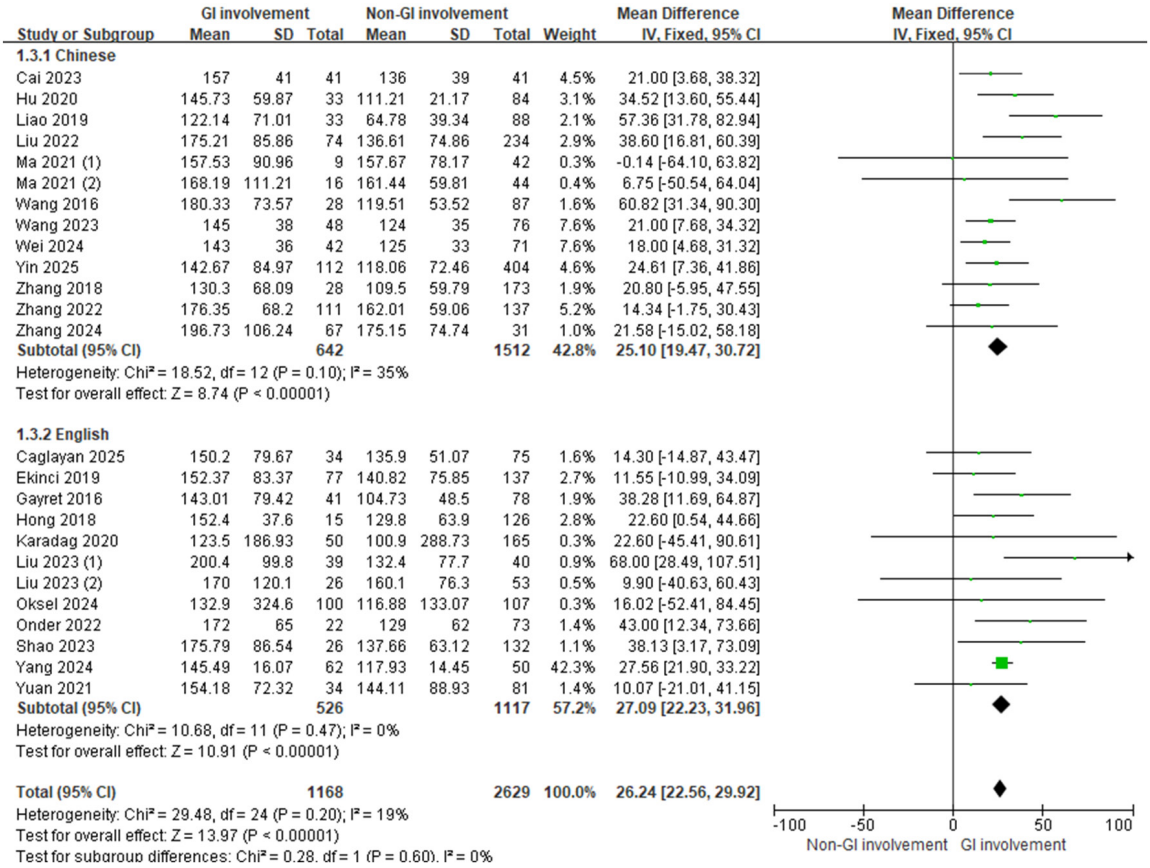



**Figure 4.** Forest plot of platelet to lymphocyte ratio and gastrointestinal involvement (publication source subgroup analysis).

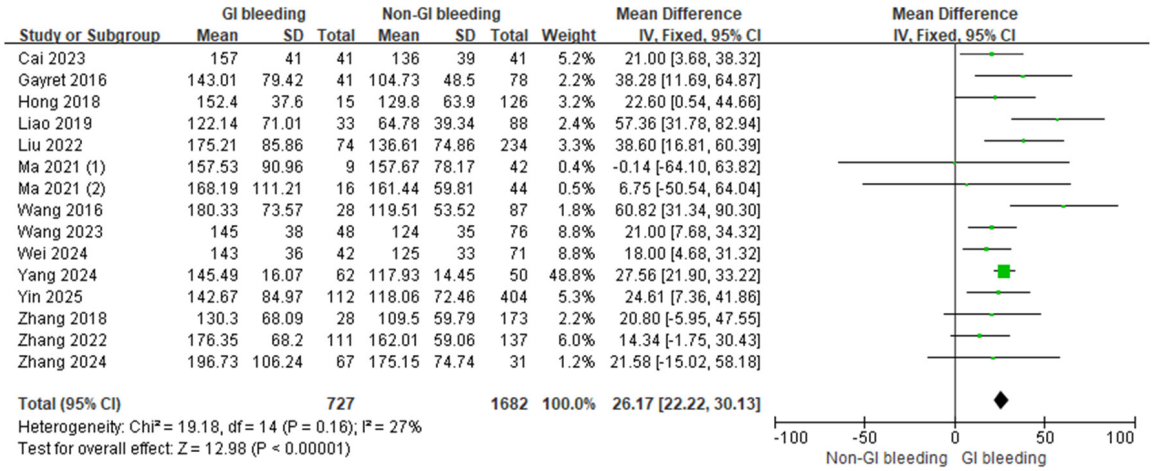



**Figure 5.** Forest plot of platelet to lymphocyte ratio and gastrointestinal bleeding.

response suppression. Consequently, an elevated PLR may, to some extent, indicate more severe vascular inflammatory injury and a higher risk of visceral organ involvement [36]. This study confirms a robust positive association between PLR and gastrointestinal involvement and bleeding in both pediatric and adult patients with HSP. It is important to emphasize,

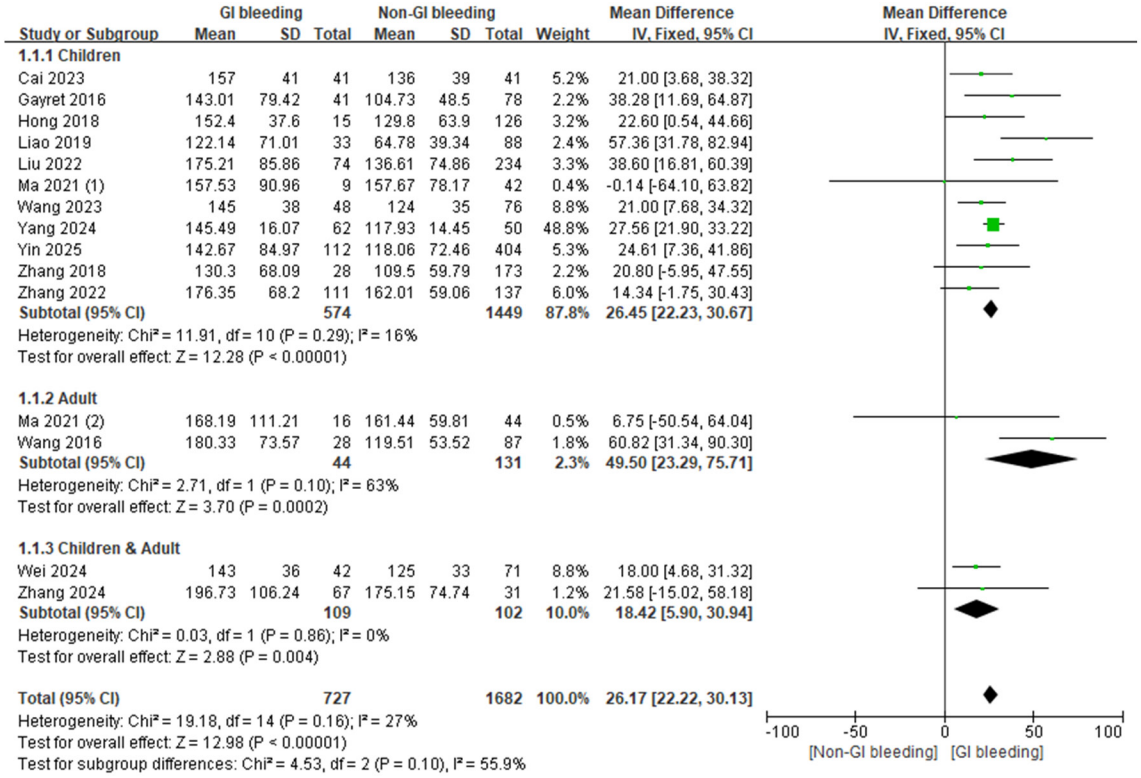



**Figure 6.** Forest plot of platelet to lymphocyte ratio and gastrointestinal bleeding (population subgroup analysis).

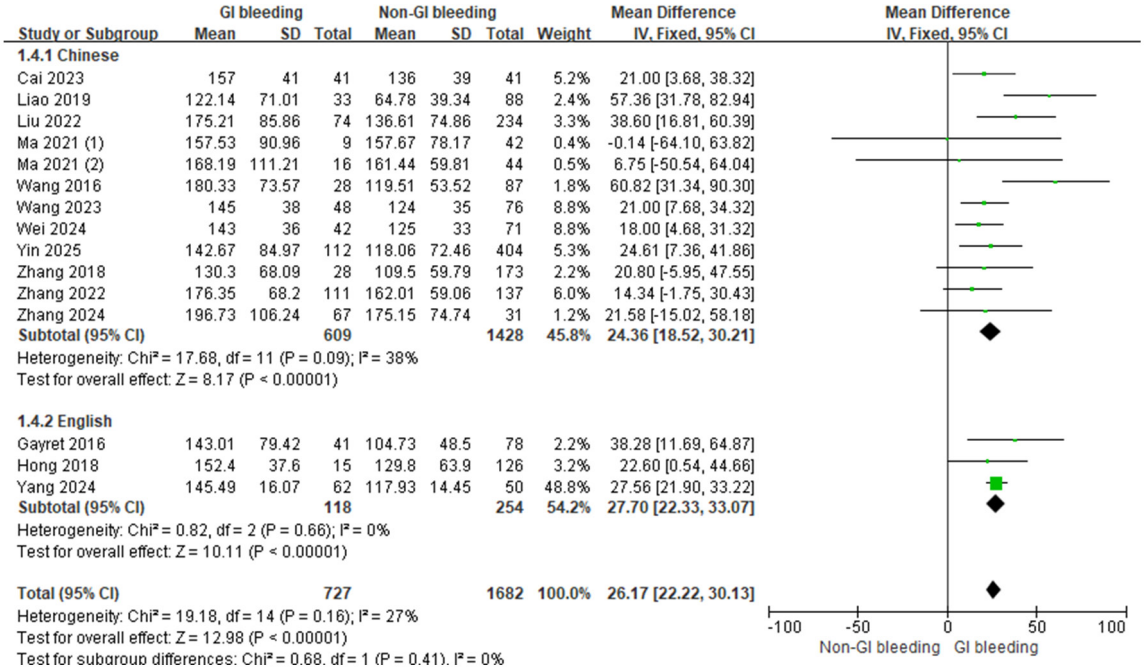



**Figure 7.** Forest plot of platelet to lymphocyte ratio and gastrointestinal bleeding (publication source subgroup analysis).

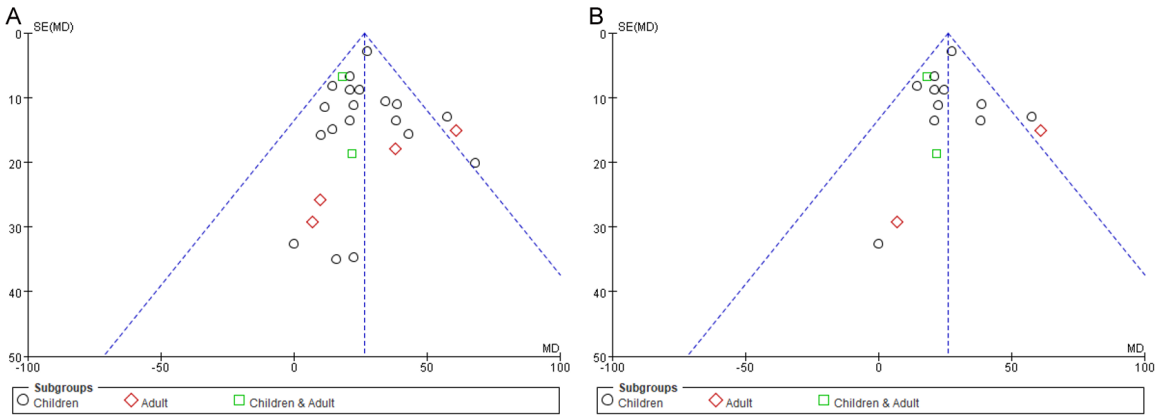



**Figure 8.** Funnel plots of platelet to lymphocyte ratio and gastrointestinal involvement (A), and of platelet to lymphocyte ratio and gastrointestinal bleeding (B) with population subgroup.

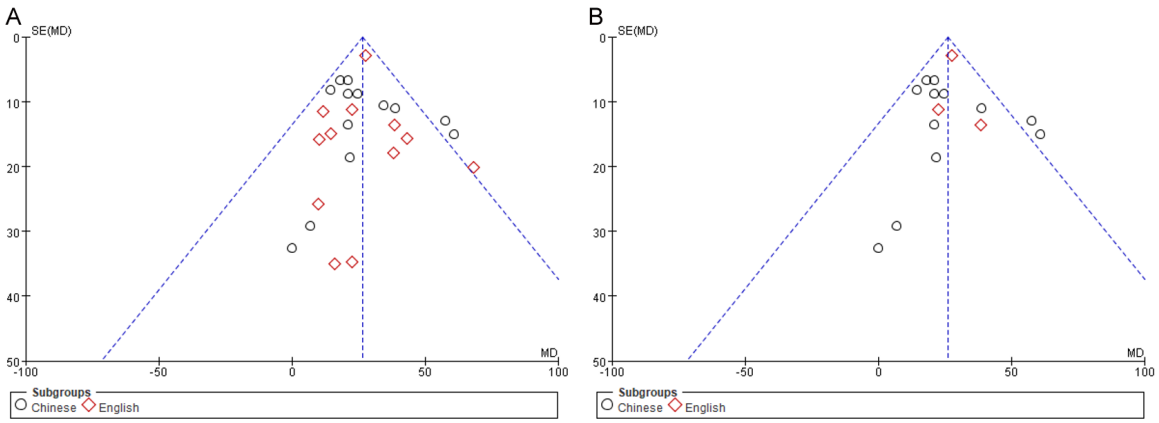



**Figure 9.** Funnel plots of platelet to lymphocyte ratio and gastrointestinal involvement (A), and of platelet to lymphocyte ratio and gastrointestinal bleeding (B) with publication source subgroup.

however, that the cross-sectional and retrospective designs of the included studies preclude definitive determination of whether PLR elevation precedes or follows the onset of gastrointestinal injury; the observed association should be interpreted as correlational rather than causal or temporal.

PLR is one of several peripheral blood-derived inflammatory indices that have been investigated in the context of HSP. The neutrophil-to-lymphocyte ratio (NLR), another widely studied marker, primarily reflects the neutrophilic inflammatory response and has been shown to be elevated in HSP patients with severe organ involvement, including those with gastrointestinal bleeding. Several of the studies included in the present meta-analysis simultaneously evaluated NLR and PLR, consistently reporting that both markers were significantly elevated in patients with gastrointestinal involvement or bleeding. While both PLR and NLR incorporate lymphocyte counts in their denominators, PLR uniquely captures the platelet-mediated component of inflammation, which is particularly relevant in HSP given the central role of platelet activation in IgA vasculitis. Combining PLR and NLR may provide a more comprehensive assessment of the inflammatory state than either marker alone.

C-reactive protein (CRP), a classical acute-phase reactant, has also been evaluated as a marker for gastrointestinal complications in HSP. While CRP is widely available and reflects systemic inflammation, it lacks specificity for

vascular inflammation and does not capture the immune cell imbalance that characterizes HSP. Moreover, CRP levels can be influenced by various confounding factors, including concurrent infections and tissue injury unrelated to vasculitis. In contrast, PLR is derived from a routine complete blood count, is readily available at no additional cost, and integrates information from both the inflammatory (platelet) and immune (lymphocyte) pathways. The monocyte-to-lymphocyte ratio (MLR) has also been explored in HSP, though the evidence remains limited. Future studies directly comparing the diagnostic and prognostic performance of PLR, NLR, CRP, and MLR in the same cohort would help clarify the relative utility of these biomarkers and whether their combination improves risk stratification.

Although the overall heterogeneity in our meta-analysis was low to moderate ($I^2$ = 19% for gastrointestinal involvement; $I^2$ = 27% for GIB), several factors may contribute to the variability observed across studies. First, differences in study design (cross-sectional, case-control, and retrospective cohort) may have influenced the observed effect estimates, as each design has inherent strengths and limitations in terms of selection bias, recall bias, and temporal clarity. Second, the diagnostic criteria for gastrointestinal involvement and bleeding varied across studies; some studies defined GIB based on the presence of melena or hematemesis, while others included occult blood positivity or endoscopic findings, potentially leading to differential misclassification. Third, the methods and timing of PLR measurement were inconsistent; some studies measured PLR at admission, while others used values obtained at various points during the disease course, and the use of different laboratory analyzers may have introduced inter-laboratory variability.

Fourth, the patient populations differed across studies in terms of age distribution, disease severity, and treatment status, all of which may have influenced PLR values. Notably, the adult subgroup in the GIB analysis exhibited higher heterogeneity ($I^2$ = 63%), which may be attributed to the small number of studies (only two) and the greater clinical heterogeneity of adult HSP, which is known to have a different disease phenotype and severity profile compared to pediatric HSP. Fifth, a substantial proportion of the included studies (12 of 23) were published in Chinese journals, which may introduce language and regional bias; however, our subgroup analysis comparing Chinese-language and English-language publications did not reveal significant differences in effect estimates (P = 0.60, 0.41). Finally, for studies that reported PLR as median (interquartile range), the conversion to mean and standard deviation using the method of Wan et al. [9] may have introduced estimation errors, particularly in studies with small sample sizes or non-normal distributions.

The findings of this meta-analysis carry several clinical implications. First, PLR, as a simple and inexpensive derivative index derived from a routine complete blood count, is universally available in clinical settings without requiring additional tests or specialized equipment. Its significant association with gastrointestinal involvement and bleeding in HSP patients suggests that it may serve as a convenient auxiliary reference indicator for clinicians, particularly in resource-limited settings where advanced biomarker testing may not be readily accessible. Second, the consistent associations observed across both pediatric and adult populations suggest that PLR may have broad applicability in HSP patients regardless of age. Third, clinicians managing HSP patients with elevated PLR should remain vigilant for potential gastrointestinal complications, including close monitoring for signs of abdominal pain, melena, and hematemesis, and consider early endoscopic evaluation when clinically indicated.

However, it is important to emphasize that PLR should not be used as a standalone diagnostic or screening tool. The observational nature of the included studies means that the observed associations do not establish causality or temporal precedence. Furthermore, a universally accepted PLR cut-off value in HSP has not yet been established, and the optimal threshold may vary by age group, disease severity, and ethnic background. PLR values should therefore be interpreted in conjunction with clinical symptoms, physical examination findings, and other laboratory values. The integration of PLR into a comprehensive risk assessment model, incorporating clinical features and other biomarkers, may improve the accuracy of risk

stratification for gastrointestinal complications in HSP.

The limitations of this study include the following: the majority of the included studies employed cross-sectional or retrospective designs, which preclude the establishment of temporal relationships or causal inferences between PLR and gastrointestinal outcomes; the overall quality of the included studies was only moderate; some studies lacked clarity in reporting diagnostic criteria and patient characteristics, making it difficult to accurately assess study quality and to screen accordingly. Furthermore, the reporting formats for PLR in the included studies were inconsistent (varying units and different measures of dispersion, including standard deviation and interquartile range), which may have introduced some measurement and statistical biases and contributed to heterogeneity among the studies. Future research should focus on further validating the temporal relationship between dynamic changes in PLR and the degree of gastrointestinal injury in HSP patients, as well as exploring the optimal clinical cut-off values for different age groups.

In summary, current evidence consistently indicates that an elevated PLR is a risk factor for gastrointestinal involvement and GIB in HSP patients. Clinicians may consider PLR as a convenient auxiliary reference indicator and, in conjunction with clinical symptoms, improve the recognition and management of high-risk patients. It is recommended that future studies further standardize testing protocols and conduct prospective validation to provide higher-quality evidence for improving the prognosis of patients with HSP.

## Disclosure of conflict of interest

None.

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