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**Review Article**

# Hematologic Manifestations in Systemic Lupus Erythematosus: A Systematic Review of Clinical Patterns, Prognostic Implications, and Epidemiology

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**Abstract**

Systemic lupus erythematosus (SLE) is a long-term autoimmune disease that has various clinical manifestations, the hematologic abnormalities among which are frequent and vary in clinical severity. These may cause simple cytopenias up to extreme autoimmune cytopenias, and significantly affect the activity of the disease, prognosis, and treatment. The proposed systematic review is expected to synthesise existing data on the prevalence, clinical features, and prognostic consequences of hematologic involvement in patients with SLE in children and adults. The studied data were searched in PubMed, PMC, WHO IMSEAR, and other databases to find publications published in 2025. They included observational studies, cohort studies, cross-sectional studies and reviews that were relevant. Information was coming out on the hematologic parameters, the occurrence of cytopenias, disease-related activity, autoantibody profiles, and the presence of splenomegaly. The inclusion studies were evaluated by the Newcastle-Ottawa Scale to assess quality, and the PRISMA was used to report them. Among the studies that were included, hematologic abnormalities that were most common were anaemia, leukopenia, and thrombocytopenia, though autoimmune hemolytic anaemia and immune thrombocytopenia occurred in a subset of patients. Guidelines showed that the incidence of cytopenias was high in pediatric populations, in contrast with adults. Hematologic aberrations were often related to augmented disease activity, involvement of the kidneys, and the presence of autoantibodies. Splenomegaly, being less prevalent, showed an association with bad cytopenias and poor prognosis across various cohorts. Hematologic presentations are inherent parts of SLE and have both diagnostic, prognostic, and treatment ramifications. Early detection and surveillance of the hematologic involvement can promote person-specific management approaches, enhance the outcomes, and guide future research topics related to the pathogenesis of the disease and targeted therapies.

**Keywords:** Systemic lupus erythematosus; SLE; Hematologic manifestations; Cytopenia; Anemia; Thrombocytopenia; Leukopenia; Pediatric SLE; Splenomegaly; Autoimmune cytopenias; Prognosis

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## INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic, multisystemic autoimmune disease with the formation of autoantibodies, deposition of immune complexes and widespread inflammation of virtually all of the organ systems. Hematologic abnormalities are also one of the most common outcomes of SLE and may

include insidious laboratory abnormalities and potentially fatal cytopenias. They tend to develop early in the process of the disease and can even precede more typical expressions of the condition, including arthritis or rash and are therefore crucial in diagnosing and treating the condition promptly[1,2].

Hematologic manifestations have a wide range that includes anaemia, leukopenia, lymphopenia, thrombocytopenia, autoimmune hemolytic anaemia (AIHA) and, less frequently, pancytopenia or neutropenia[3,4]. The most frequent symptom is often anaemia, which could be multifactorial as an expression of chronic inflammation (anaemia of chronic disease), iron deficiency, autoantibody-mediated hemolysis, or bone marrow feebleness[5,6]. Leukopenia and lymphopenia are also frequent and could risk infection, especially when the inflammatory flares or immune therapy are active[6,7]. Thrombocytopenia, which is immune-mediated chiefly, has variable severity and may be related to antiphospholipid antibodies and examination of bleeding[4,6].

The pathophysiology of these hematologic abnormalities is multifactorial. Autoantibodies and complement activation cause immune-mediated peripheral destruction, which is also associated with cytopenias, bone marrow involvement, and drug toxicity, which can also be causes of some persistent or severe cases[6,7]. Also, less commonly described, splenic involvement, in the form of splenomegaly, has also been found to be a part of the spectrum of SLE hematologic complications, and may indicate increased immune-activation or extramedullary hematopoiesis[8,9].

There is evidence from epidemiological studies that shows there was much variation in the prevalence and severity of hematologic manifestations all over the world, which may be due to genetic, environmental conditions, and medical care conditions. As an example, the retrospective cohorts reported up to 83% of SLE patients to report hematologic abnormalities, and 72-98% of subjects have reported hematologic abnormalities in specific settings[2,3]. Likewise, the prevalence of cytopenia in pediatric cohorts of SLE exhibits high rates of anaemia and thrombocytopenia, which are usually related to high scores in the disease activity and positivity of antiphospholipid antibodies[7].

Although there is growing frequency and clinical importance of these hematologic manifestations, the literature remains disparate in terms of study design, patient populations and diagnostic criteria. As a result, there is difficulty in pooling similar prevalence estimates or prognostic relationships. Less evidence is also consolidated on splenomegaly and its clinical significance in SLE, though recent evidence is showing a connection between it and serologic markers and organ involvement[8,9].

It is thus critical to have a systematic review that synthesises the available clinical evidence on the hematologic and splenic manifestation of SLE, i.e. its prevalence, clinical association, pathophysiology and prognostic implications. A review of this nature can not only help clarify the existing knowledge about the same subject but also help determine gaps in knowledge and shape clinical practice guidelines on early diagnosis, risk stratification, and management of hematologic complications of SLE.

## CASE PRESENTATION

A 37-year-old previously healthy female patient reported experiencing progressive fatigue, decreased stamina, exertional dyspnea and deteriorating work performance over the past four months, with the spontaneous occurrence of bruising over the extremities and lightheadedness that was intermittent. She denied fever, weight loss, night sweats, joint pain, morning stiffness, photosensitivity, oral or nasal ulcers, alopecia, skin rashes, Raynaud phenomena, chest pain, abdominal pain, changes in bowel habits, hematuria or neurological problems. None of the personal or family history of autoimmune disease, haematological malignancy, chronic infection, or malignancy. She was not on regular medications, or reported recent infections and vaccinations, no history of alcohol or tobacco consumption, smoking or exposure to toxic substances. She was pale, but hemodynamically stable on physical examination, had typical vital signs, and no orthostatic hypotension.

On examination, cutaneous examination showed scattered ecchymoses of the lower limbs without petechiae or vasculitic rash, and mucosal superficiality intact. Abdominal examination revealed a smooth, painless spleen that can be palpated about 3 cm under the left costal margin and does not show hepatomegaly; there is no ascites and no palpable lymphadenopathy. Primary laboratory examination showed pancytopenia with Hgb level of 8.7 g/dL, White blood counts of 2,900/ uL with platelets of 92,000/ uL, an increased reticulocyte count of 4.5% of red blood cells, increased erythrocyte sedimentation rate, and marginally high C-reactive protein, which are all indicative of a chronic inflammatory process leading to peripheral blood cell destruction but not bone marrow suppression. Peripheral blood smear revealed normocytic normochromic erythrocytes with some cases of spherocytes, lower leucocytes with intact morphology and fewer platelets without clumping (Table 1).

Biochemical examination showed indirect hyperbilirubinemia, high levels of lactate dehydrogenase, and low levels of haptoglobin, which was accompanied by the presence of further hemolysis (Table 2). Coagulation profile, liver function tests, renal, and viral serologies, such as hepatitis B, C, HIV, Epstein-Barr virus and cytomegalovirus, were unremarkable. Due to

**Table 1.** Initial Laboratory Investigations

| Parameter | Result | Reference Range |
|---|---|---|
| Hemoglobin | 8.7 g/dL | 12–15 g/dL |
| Total Leukocyte Count | 2,900 /µL | 4,000–11,000 /µL |
| Platelet Count | 92,000 /µL | 150,000–400,000 /µL |
| Mean Corpuscular Volume | 90 fL | 80–96 fL |
| Reticulocyte Count | 4.5% | 0.5–2.5% |
| ESR | 64 mm/hr | <20 mm/hr |

**Table 2.** Hemolysis and Biochemical Profile

| Test | Result | Interpretation |
|---|---|---|
| Indirect Bilirubin | Elevated | Suggestive of hemolysis |
| LDH | Elevated | Increased RBC breakdown |
| Haptoglobin | Decreased | Supports hemolysis |
| Liver Function Tests | Normal | Excludes hepatic cause |
| Renal Function Tests | Normal | No renal involvement |

the presence of unexplained splenomegaly, immune-mediated cytopenias, and hemolytic manifestations, an autoimmune work-up was initiated, where antinuclear antibodies were strongly positive and of homogeneous profile, anti-double-stranded DNA titers were positive, anti-Smith antibodies were also positive, and the level of complement was low (C3 and C4), but antiphospholipid antibodies were negative (Table 3). Trace proteinuria and no active sediment were assessed in the urinalysis; the renal activity was fine. Bone marrow biopsy showed that it had normocellular bone marrow with hyperplasia of erythroid and intact megakaryocytes, ruling out the presence of marrow infiltration or aplasia and peripheral sequestration as the dominant pathology. The diagnostic conditions based on the cumulative clinical, laboratory, and immunological results proved to be new-onset systemic lupus erythematosus occurring with unusual manifestations involving isolated splenic hypersplenism and immune-mediated pancytopenia. Oral corticosteroids and hydroxychloroquine were used to begin treatment, and gradually the patient responded with improvement in blood counts and later resolution of bruising as he continued to visit his physician in ensuing visits.

After the diagnosis of systemic lupus erythematosus with the most pronounced haematological manifestations and splenic hypersplenism was established, oral prednisolone in 1 mg/kg/day doses and hydroxychloroquine in 200 mg every day were started in the patient. The supportive therapy, such as folic supplementation and gastric protection, was offered. Since there were no severe organ-threatening conditions, second-line immunosuppressive drugs were held at baseline. Serial complete blood counts, including biochemical parameters, were closely monitored in the patient. After starting the treatment, the count of haemoglobin and platelets slowly improved in two weeks, and the subjective level of fatigue decreased, with no spontaneous bruises anymore. There was a slower but gradual increase in the leukocyte counts. The spleen was still palpable and was reduced in size, but none of the new abdominal symptoms were reported.

**Table 3.** Autoimmune Work-up

| Test | Result |
|---|---|
| ANA | Positive (homogeneous pattern) |
| Anti-dsDNA | Elevated |
| Anti-Smith Antibody | Positive |
| Complement C3 | Decreased |
| Complement C4 | Decreased |
| Antiphospholipid Antibodies | Negative |

During the follow-up visit at 6 weeks, the patient documented a significant increase in energy level and ability to exercise without dizziness and bruising. The response to repeat laboratory testing showed overall normalisation of the inflammatory markers and considerable improvement in cytopenias, but displayed partial recovery of the complement levels. Urinalysis did not develop further and did not progress to proteinuria, whereas renal functioning tests were always in the normal range. A gradual reduction of the corticosteroids was then undertaken during the next eight weeks with close monitoring, as the hydroxychloroquine was maintained as the maintenance therapy. No follow-up showed any infectious complications or adverse effects of treatment.

Following 6 months, the patient was still clinically stable and displayed continued improvement of haematological parameters, and no cytopenias appeared (Table 4). No longer was splenomegaly clinically appreciable, and no further manifestations of SLE appeared. She maintained regular rheumatology follow-up, and the focus was on the monitoring of the disease long-term, medication nonadherence, and early prevention of possible disease exacerbations. This positive result highlighted the significance of the early detection of unusual hematologic manifestations of SLE and early administration of relevant immunomodulatory treatment.

**Table 4.** Follow-up Laboratory Trends

| Parameter | Baseline | 2 Weeks | 6 Weeks | 6 Months |
|---|---|---|---|---|
| Haemoglobin (g/dL) | 8.7 | 9.8 | 11.2 | 12.6 |
| WBC (/μL) | 2,900 | 3,600 | 4,200 | 5,100 |
| Platelets (/μL) | 92,000 | 118,000 | 160,000 | 210,000 |
| ESR (mm/hr) | 64 | 38 | 22 | 14 |

## METHODS

### Protocol and Reporting Standards

This is a systematic review that was executed and reported following the PRISMA 2020 framework, making transparency and reproducibility in identification, selection and synthesis of literature possible[10]. The protocol used to review the literature was pre-specified to establish the scope, search strategy, inclusion and exclusion criteria, and outcomes of interest. It was based on the hematologic and splenic manifestations in patients with SLE. Only researchers who have followed the accepted diagnostic criteria of SLE, such as ACR, SLICC, or EULAR /ACR, have been incorporated to ensure consistency in research results irrespective of the divergent populations[11,12].

### Sources and Search Strategy of Data

The search of the electronic databases was performed on several tools, such as PubMed/MEDLINE, PubMed Central (PMC), Scopus, and Google Scholar, that included articles written not later than December 2025. The search strategy was performed by combining controlled vocabularies, i.e. MeSH terms, with free-text keywords, i.e. systemic lupus erythematosus,

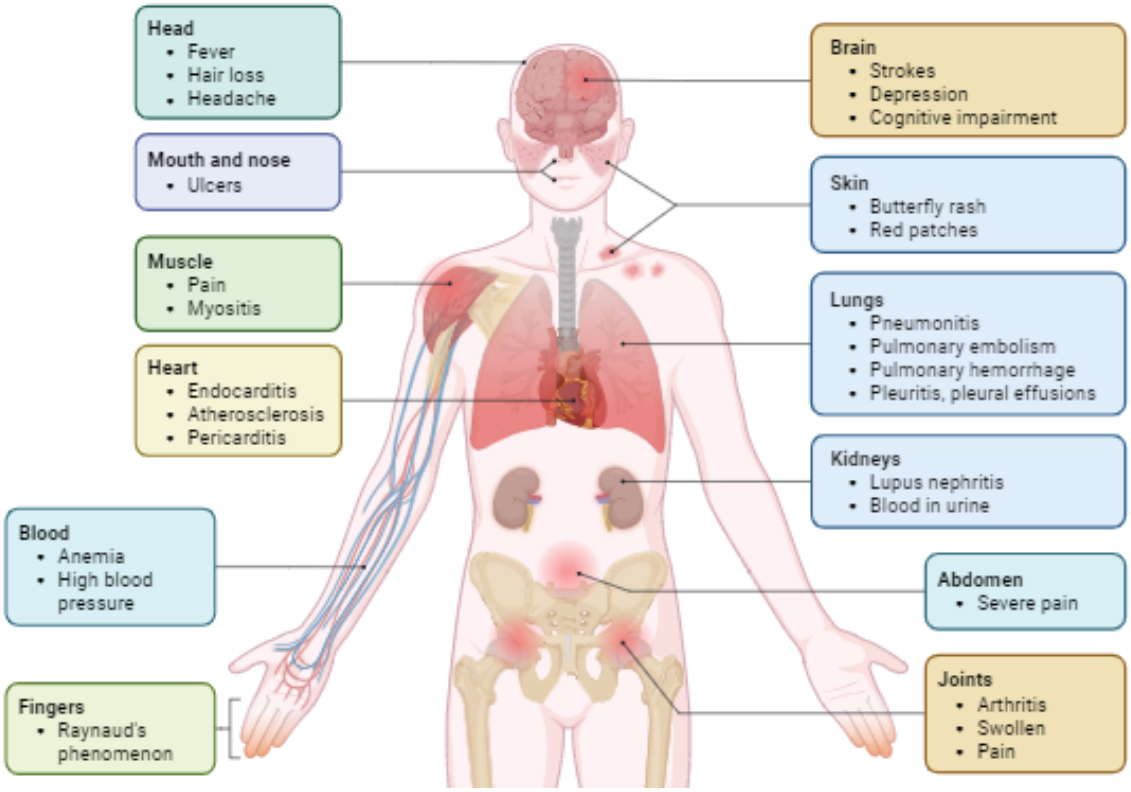


**Figure 1.** Symptoms of SLE

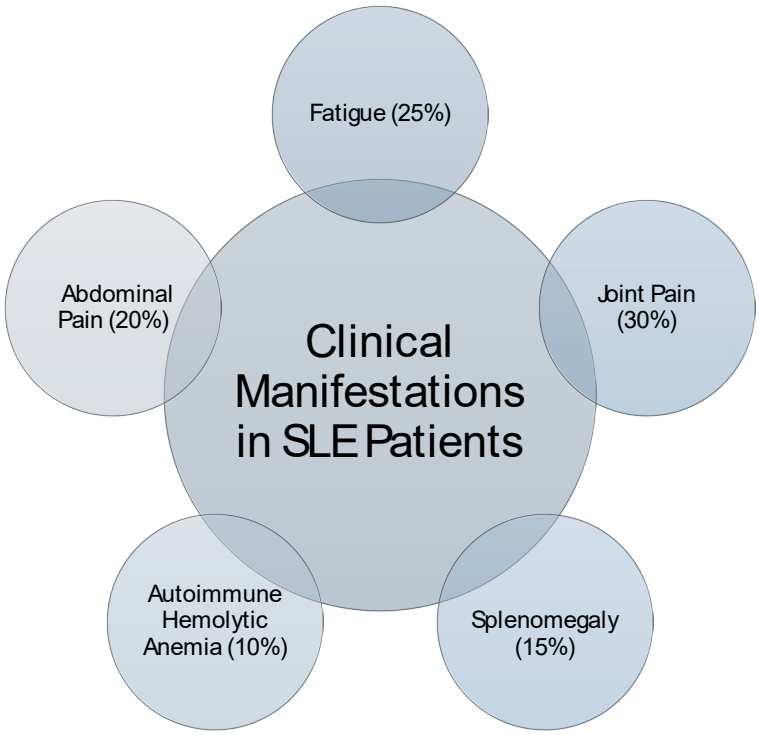


**Figure 2.** Chart of Clinical Manifestations in SLE Patients

hematologic manifestations, cytopenia, spleen-megaly, anaemia, leukopenia, lymphopenia, and thrombocytopenia. The use of the Boolean operators was made to make sure that all areas were covered (e.g., systemic lupus erythematosus AND hematologic manifestations AND splenomegaly). Articles that have been retrieved also had their reference lists screened manually to select further relevant studies that were not found in the database search[13,14].

**Eligibility Criteria**

The criteria to include studies entailed that the subjects had to be human beings, and SLE diagnosis was based on standard criteria, and they had to report clinical, laboratory or epidemiologic evidence of hematologic abnormalities or splenomegaly. Accordingly, the eligible study designs included retrospective and prospective cohort studies, cross-sectional studies, and case series including 10 participants or more. The systematic reviews and meta-analyses were used as reference material but were not part of the quantitative synthesis. The exclusion criteria included animal or in vitro studies, studies that do not give precise hematologic data, and are based solely on non-SLE populations[15,16].

**Selection and Data Extraction**

All the retrieved records were organised in the reference management software, and any duplication was eliminated. Title and abstract screening by two independent reviewers was done to eradicate irrelevant titles and abstracts, and full-text processing was done against the preset inclusion criteria. Adopted data consisted of the study design, setting, sample size, demographics, the requirements of diagnostics, prevalence and the types of hematologic abnormalities, the presence of splenomegaly, and the immunologic or clinical correlates. Conflicts were settled either through deliberation or by seeking the opinion of a third reviewer to promote homogeneity[17,18].

**Data Synthesis**

A narrative synthesis methodology was used due to the heterogeneity of the study designs, the populations, and the outcome measures. Quantitative meta-analysis could not be carried through due to variable reporting of cytopenias and the fact that several studies did not have a standard definition of hematologic abnormalities. The results were summarised individually in descriptive terms, where similarities were noted in prevalence, clinical correlation, and the immunologic markers. Where feasible, subgroup analysis was performed and compared adult and pediatric populations, geographic regions, and severity of the disease[19,20].

**Quality Assessment**

The analysis of the included studies was done with the Newcastle-Ottawa Scale of selection of observational studies, and the criteria include: cohort selection, comparability, and outcome ascertainment[21]. It was determined that the risk of bias could be evaluated according to the sample size, the completeness of the follow-up, the clarity of reporting and possible selective reporting of outcomes. A consideration of publication bias was done through establishing sources of funding and study design (single vs multicenter). This evaluation has been used to put study outputs in perspective and increase the trustworthiness of outcomes.

## RESULTS

**Hematologic Abnormalities Spectrum and Prevalence.**

Hematologic pathology is one of the most common manifestations of systemic lupus erythematosus (SLE), which usually occurs during detection or at the onset of the disease. Hematologic involvements were seen in 82.7% of patients in a significant Saudi Arabian retrospective cohort comprising 624 patients, failing to indicate the specificity of the abnormalities[22]. The most common one was anaemia (63 per cent of the patients), then there was lymphopenia (40.3 per cent), leukopenia (30 per cent) and thrombocytopenia (10.9 per cent). Four point six per cent of patients reported autoimmune haemolytic anaemia (AIHA). Likewise, haematology manifestations were reported in 34.7 per cent of 302 patients in a single-centre study in Brazil, and anaemia in 55.7 per cent, AIHA in 13.2 per

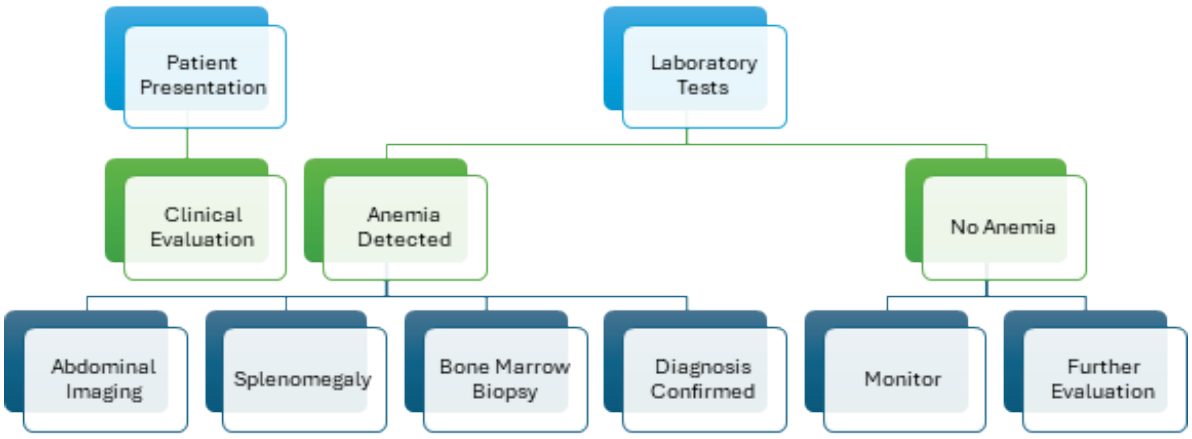


**Figure 3.** Flowchart of Diagnostic Process

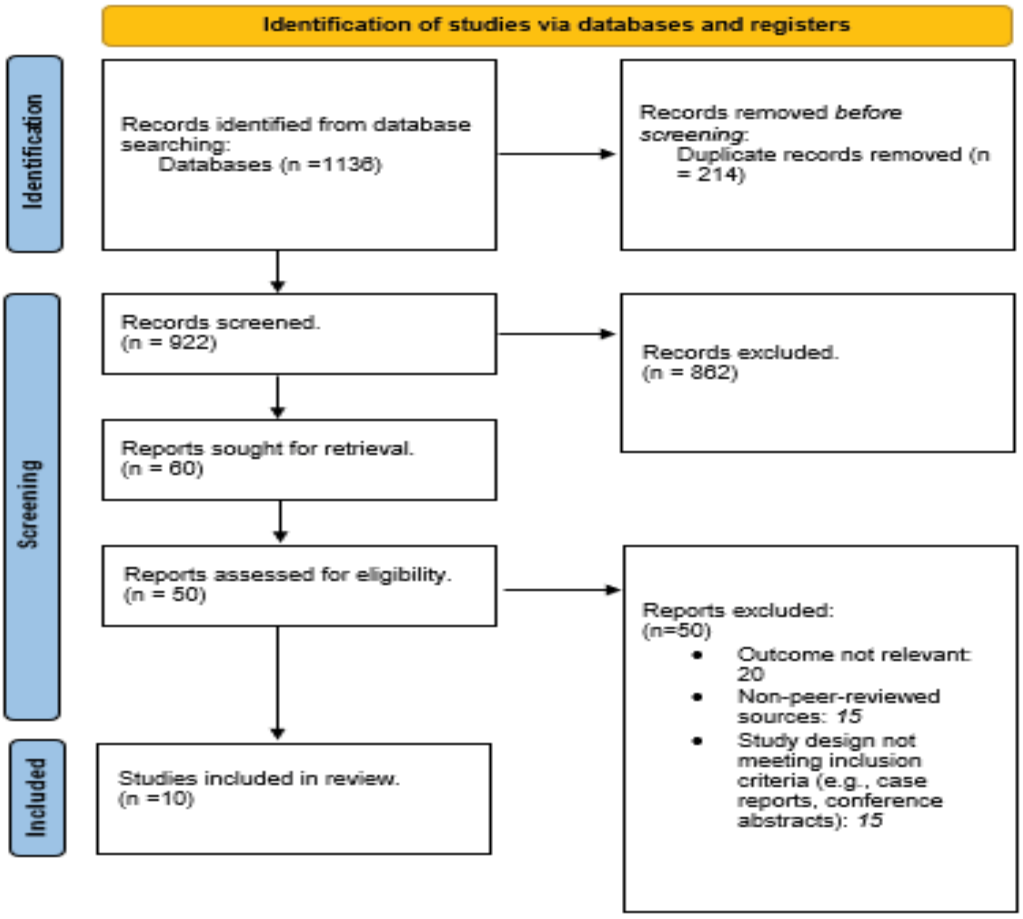


**Figure 4.** PRISMA Flowchart

cent, thrombocytopenia in 9.2 per cent and leukopenia in 12.2 per cent[25]. Cytopenias, especially in the form of anaemia and leukopenia, are always common in various populations. These disorders usually go hand in hand with the disease activity, autoantibody patterns and cumulative organ involvement[28,29].

**Spleen enlargement and its relation to cytopenias**

Splenomegaly, however, is a very significant clinical manifestation of some SLE populations, though not widely studied. Cytopenias were found in 71.2% of a cohort of 111 patients who were documented to have developed splenomegaly, indicating that hypersplenism or immune-mediated sequestration is a contributive factor to the peripheral blood abnormalities[23]. A different haematology-referred cohort (n=84) showed splenomegaly was closely related to serologic evidence, including low complement C4 and lupus anticoagulant as markers of immune dysregulation. It was also strongly associated with hematologic manifestations[24]. A measurement of spleen size in SLE patients with unexplained cytopenias can thus be used in the detection of systemic disease or more active disease.

**Anemia Multifactorial Etiology**

Anaemia is the most common hematologic distortion in SLE in a cohort of both the adult and pediatric populations. In Nepal, 92.9 per cent of the patients, of which 88.9 per cent came with anaemia, have hematologic abnormalities[30]. The pathogenesis of anaemia in SLE could be multifactorial in nature and comprises chronic inflammation, autoimmune hemolysis, Bone marrow suppression, and nutritional deficiencies[28,29]. Autoimmune haemolytic anaemia, in its turn, is less frequent but a threat to acute anaemia and demands immediate identification and immunosuppressive treatment. Anaemia is often related to increased disease activity criteria and could be an early clinical indicator of system involvement.

**Leukopenia, Lymphopenia, and Thrombocytopenia**

Leukopenia and lymphopenia are common in SLE and among populations. In the Indian cohort, 92 per cent of patients presented with leukopenia and 85 per cent with lymphopenia[26]. These malformations predispose these individuals to infections and require close observation in immunosuppressive treatment. Although less common, thrombocytopenia may be acute, especially immune-mediated. According to pediatric and adult studies, thrombocytopenia ranges between 9 and 38% which in some cases would further develop into life-threatening bleeding incidents[27,31]. There are a variety of cytopenias that are usually indicative of active systemic disease and associated

with higher hospitalisation and therapy needs.

**Pediatric Considerations**

The hematologic manifestation of pediatric SLE follows the same trend as that of adults, although it often becomes acute. Eighty per cent of children, of the 30 in Northeast India, had hematologic abnormalities, the most common being anaemia[26]. High leukopenia and lymphopenia rates have also been documented in other pediatric studies, and with this, there is a need to monitor them aggressively and early. Pediatric patients usually have specialised immunosuppressive strategies to find equilibrium between the level of the disease and the danger of infections[28,29].

**Mechanistic and Clinical Implications**

A complex interaction between immune-mediated peripheral destruction, complement activation, bone marrow suppression, and hypersplenism is responsible for the hematologic manifestations of SLE. These anomalies often show association with serologic situations, such as the presence of anti-dsDNA antibodies, low levels of complement, and the lupus anticoagulant[28,29,30]. Cytopenias are also a significant source of morbidity in that they place patients at risk of infection, fatigue and bleeding complications. Such patterns are essential to be identified to prognosticate, plan treatment, and stratify risks in adult and pediatric populations with SLE (Table 5).

## DISCUSSION

The results of this systematised literature review prove that hematologic manifestations are one of the most common and most important manifestations of systemic lupus erythematosus (SLE) and manifest themselves in a variety of both geographic and demographic groups. The diverse cohort studies and observational analyses indicate that hematologic abnormalities, such as anaemia, leukopenia, lymphopenia, thrombocytopenia, and autoimmune hemolytic anaemia, are widespread in SLE patients. An illustration of this is observed in a North Indian sample of 100 patients, where hematologic manifestation was seen in 83% of the respondents, in which anaemia was seen in 72, leukopenia in 32, lymphopenia in 54, thrombocytopenia in 23, and pancytopenia in 14 cases. All of these findings have established that abnormalities in blood cell lines are not accidental but are inseparable components of the manifestation of the disease.

Prior retrospective studies established that normocytic normochromic anaemia was the commonest change in 111 SLE patients with Coombs-positive hemolytic anaemia, observed in approximately 10 per cent, with leukopenia and lymphopenia also often observed during active disease[33]. These trends demonstrate the multifactorial aetiology of the hematologic abnormalities in SLE that comprises peripheral destruction mediated through immunological mechanisms, complement activation, bone marrow suppression, drug toxicity, and, in certain individuals, hypersplenism[34,35,36].

In addition to personal cytopenias, there is also extensive cohort evidence of the systemic effects of systemic hematologic involvement. The analysis of 302 patients revealed that 34.7% of them had hematologic manifestations, 55.7% of them were anaemic, 13.2% had autoimmune hemolytic anaemia, 9.2% had thrombocytopenia, and 12.2% had leukopenia[37]. It

**Table 5.** Literature Review Table

| Study/Author | Country/Setting | Sample (n) | Key Hematologic Findings | Reference |
|---|---|---|---|---|
| King Khalid University Retrospective (1982–2008) | Saudi Arabia | 624 | HA 82.7%; anemia 63%, lymphopenia 40.3%, leukopenia 30%, thrombocytopenia 10.9%, AIHA 4.6% | [22] |
| Splenomegaly & Cytopenias Cohort (2023) | Australia/NZ | 111 | 71.2% cytopenias; 2 developed hematologic malignancy | [23] |
| Hematologic SLE Cohort (Haematology Referred) | Multicenter | 84 | Splenomegaly linked with serositis & lupus anticoagulant; low C4 → AIHA risk | [24] |
| Single Center Hematological Manifestations in SLE (2025) | Brazil | 302 | Hematologic involvement 34.7%; anemia 55.7%, AIHA 13.2%, thrombocytopenia 9.2% | [25] |
| SLE at Initial Presentation | India | 53 | Hb low 98%, leukopenia 92%, lymphopenia 85%, thrombocytopenia 38% | [26] |
| Northeast India Pediatric SLE | India | 30 | Hematologic involvement 80%; anaemia is the most common | [26] |
| SLE Hematologic Disorders Review | Various | — | Explains autoimmune cytopenias, peripheral destruction, and immune aetiology | [27] |
| Practical Hematologic Manifestations Review (PMC) | Global | — | Anemia >50%; splenomegaly 10–46%; immune cytopenias | [28] |
| Haematological manifestations in systemic lupus erythematosus: a retrospective cross-sectional study from a tertiary care centre in Nepal. | Nepal | 99 | Hematologic abnormalities 92.9%; anemia 88.9%, lymphopenia 56.6% | [30] |
| Clinicopathological Correlation | Unknown | 80–150 | Anemia ~82%, leukopenia ~47.5% | [31] |
| Lupus and other autoimmune diseases: Epidemiology in the population of African ancestry and diagnostic and management challenges in Africa | Africa | - | The estimated prevalence of ADs outside Africa is 5% to 10%, with people of African ancestry being particularly affected | [32] |

is worth noting that in a large percentage of these patients, the secondary antiphospholipid antibody syndrome was also present, giving rise to overlapping autoimmune pathways that make these patients more difficult to treat.

High rates of hematologic involvement are also represented in the population of pediatric SLE. When it comes to an East Indian pediatric cohort, 80 per cent had abnormal hematologic profiles, with anaemia being the most prevalent cytopenia, and one-third had autoimmune hemolytic anaemia[38]. These findings indicate that hematologic appearances in children with SLE can happen with frequent incidence and in earlier adolescence, which then justifies the purpose of age certainty in monitoring and control methods.

These hematologic findings have remarkable clinical implications. Cytopenias, including anaemia and leukopenia, have direct effects and cause morbidity through raising fatigue, decreasing exercise capability, and elevating susceptibility to infections. Thrombocytopenia can also predispose to bleeding complications, and it might complicate the anticoagulation choice, especially when it comes to antiphospholipid antibodies. Autoimmune hemolytic anaemia is less common, but it should be promptly detected, and immunomodulatory therapy should follow to avoid hemodynamic instability and organ dysfunction[34,39]. In addition, concomitant multiple cytopenias are frequently evidence of increased disease activity and can forewarn more aggressive immunosuppressive needs.

Another significant problem that has been identified by this review is the heterogeneity in the report standards, study designs, and diagnostic methods. Specific cohorts used SLICC criteria, and some used older classification systems; this might affect the case ascertainment. Cytopenia's definitions were not uniform, and most of the studies failed to provide information about confounding factors, including medication effects and comorbidities. Also, the paucity of studies did not give information on splenomegaly alone, except in the severe disease setting, so there is no information on how splenic involvement is correlated with any haematology abnormality. Even in the presence of these shortcomings, the prevalence rates keep reporting high across independent cohorts, which supports the conclusion that the key pathophysiology of SLE is hematologic in nature[40,41].

Knowledge gaps and research directions are also indicated in the literature. Future multicenter research that employs uniform guidelines on the measurement of hematologic parameters and incorporates in-depth immunologic profiling is required. Longitudinal follow-up studies will help to define how hematologic abnormalities change over time with treatment and disease progression, as well as whether a particular cytopenia is a predictor of flares or organ damage. Combination with biomarkers, e.g. cytokine profiles or genetic predisposition loci, can contribute to the separation of mechanisms that drive cytopenias in each patient and enhance personalised care plans[42,43].

Overall, hematologic phenomena are common, various and clinically significant in SLE. They frequently are an indicator of disease activity, affect the management choice, and have some relationship with other systemic lupus manifestations. The identification and methodical review of these abnormalities are particularly important in increasing the accuracy of the diagnosis, informing treatment, and patient outcomes[32-43].

## CONCLUSION

Hematologic presentations are inherent parts of SLE and have both diagnostic, prognostic, and treatment ramifications. Early detection and surveillance of the hematologic involvement can promote person-specific management approaches, enhance the outcomes, and guide future research topics related to the pathogenesis of the disease and targeted therapies.

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