Leukocyte Variations in Gestational Hypertension: An Immunohematological Review

Chinedu-madu Jane Ugochi

Department of Haematology, Faculty of medical laboratory science, Federal university Otuoke, Otuoke, Bayelsa State, Nigeria

Abstract

Gestational hypertension (GH) is a hypertensive condition of pregnancy characterised by the emergence of hypertension after 20 weeks of gestation, in the absence of proteinuria or systemic organ involvement. Although preeclampsia has been thoroughly studied, the modifications in leukocytes associated with gestational hypertension have garnered relatively little attention, despite common immunological mechanisms. Physiological pregnancy is characterised by leukocytosis, mostly induced by neutrophilia, alongside relative lymphopenia, which indicates an innate–adaptive immunological transition. In GH, data suggests further enhancement of these alterations, encompassing raised total white blood cell (WBC) counts, an increased neutrophil-to-lymphocyte ratio (NLR), monocyte activation, and heightened systemic inflammatory indices. These results correspond with a condition of increased maternal systemic inflammation, endothelial dysfunction, and placental maladaptation. This review integrates contemporary literature regarding leukocyte changes in GH, examines the processes responsible for leukocyte-mediated vascular injury, assesses the therapeutic relevance of haematological indices, and underscores future research trajectories. Leukocyte-derived markers may function as economical supplementary tools for growth hormone risk classification, although necessitate disease-specific validation.

Keywords

Gestational Hypertension, Leukocytes, Neutrophil-to-Lymphocyte Ratio, Monocytes, Systemic Inflammation, Hypertensive Disorders of Pregnancy

1. Introduction

Hypertensive disorders of pregnancy (HDP) constitute a prevalent medical problem during gestation, impacting roughly 10–15% of pregnancies globally. They continue to be a major cause of maternal illness and death, as well as a major cause of problems during pregnancy, such as intrauterine growth restriction, premature delivery, placental abruption, and stillbirth. HDP is still a worldwide health problem, especially in low- and middle-income countries where access to good maternal care is limited. This is true even if antenatal care has improved and better diagnostic and monitoring techniques are now available [1].

Gestational hypertension (GH) is a rather common condition within the spectrum of HDP. Clinically, it is characterised by the recent emergence of increased blood pressure—systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg—occurring after 20 weeks of gestation in women who were previously normotensive. In contrast to preeclampsia, gestational hypertension (GH) does not present with substantial proteinuria, biochemical indicators of maternal organ impairment, or signs of uteroplacental problems. Nevertheless, it is not a benign disease, since 25–50% of women with gestational hypertension may advance to preeclampsia, particularly if hypertension manifests early in the third trimester [2].

Gestational hypertension is frequently seen as a diagnosis of exclusion, necessitating meticulous assessment to eliminate persistent hypertension, preeclampsia, or secondary aetiologies of hypertension. Some women may maintain stability with only mild hypertension and deliver without negative consequences, whereas others may experience severe hypertension, leading to maternal problems such as eclampsia, stroke, pulmonary oedema, and hepatic dysfunction. Gestational hypertension has been linked to diminished placental perfusion, resulting in negative consequences such as foetal growth restriction, low birth weight, and an elevated risk of neonatal intensive care [3] unit (NICU) admission.

The global burden of gestational hypertension (GH) is not fairly spread out. It is far more common in sub-Saharan Africa and South Asia. This difference is mostly due to structural problems including not being able to get good prenatal care, waiting too long to get maternal care, and not being able to find out about high blood pressure problems during pregnancy early on. In these areas, the risks of GH are even higher because of poor healthcare infrastructure, low socioeconomic status, and a lack of awareness. This leads to higher rates of maternal and neonatal morbidity and mortality than in high-income nations. On the other hand, in industrialised areas where routine antenatal care and advanced monitoring are easy to get, timely detection and intervention have led to better outcomes. This shows how important it is for everyone to have equal access to maternal healthcare around the world [4].

Managing GH necessitates a comprehensive strategy that harmonises maternal safety with foetal health. Current tactics stress close monitoring of both the mother and the foetus in the clinic and the lab, quick diagnosis of disease progression, and personalised treatment plans. This includes checking the mother's blood pressure regularly, looking for proteinuria and other signs of preeclampsia, checking how well her organs are working, and doing a series of ultrasounds to keep an eye on the baby's growth and the amount of amniotic fluid. The major goal is to keep the mother stable while finding the best time for delivery to lower the risks of prematurity on one hand and maternal problems on the other [5].

Preventive measures are also important for lowering the effects of GH and related problems. Comprehensive antenatal screening facilitates the early identification of women at elevated risk, while lifestyle modifications—such as sustaining a healthy weight, participating in regular physical activity, and adopting a balanced diet—enhance maternal cardiovascular health. The preventive use of low-dose aspirin, especially in women deemed high risk (e.g., those with a history of preeclampsia, chronic hypertension, or multiple pregnancies), has proven effective in reducing the progression from gestational hypertension (GH) to preeclampsia, thereby improving outcomes for both mothers and newborns. Moreover, calcium supplementation in populations with insufficient dietary calcium consumption has been demonstrated to contribute to prevention [6].

Historically, gestational hypertension (GH) was perceived as a relatively innocuous disease and only a potential precursor to preeclampsia; however, current data contests this perspective. Recent findings indicate that gestational hypertension (GH), irrespective of its progression to preeclampsia, correlates with negative pregnancy outcomes, such as preterm delivery, intrauterine growth restriction, and elevated caesarean section rates. Long-term follow-up studies indicate that women who have gestational hypertension (GH) during pregnancy may face an increased risk of developing chronic hypertension, ischaemic heart disease, and stroke in later life, suggesting that GH may act as an early indicator of future cardiovascular illness. These results show that GH has to be looked at again, not just as a short-term problem during pregnancy, but also as a condition that can have serious health effects on women in the long run. [7].

Pregnancy immunology is a very dynamic process that involves well controlled inflammatory and immunological changes that happen at different stages of pregnancy to help the mother tolerate the foetus and the pregnancy move forward. In the early stages of pregnancy, a regulated pro-inflammatory milieu is necessary for implantation, trophoblast invasion, and the formation of maternal-fetal circulation. During this period, innate immune cells such uterine natural killer (uNK) cells, macrophages, and dendritic cells become more active. They release cytokines and growth factors that help the placenta expand and the blood vessels change shape [4]. In contrast, mid-pregnancy is regarded as an immunologically quiet or anti-inflammatory phase. During this time, the mother's immune system changes to keep the semi-allogeneic foetus from causing harmful inflammatory reactions while yet being tolerant of it. Regulatory T cells (Tregs), changes in cytokine production that make the environment less inflammatory (for example, higher levels of IL-10 and TGF-β), and changes in the activity of antigen-presenting cells all help keep this equilibrium. This balance in the immune system is very important for keeping the foetus growing and lowering the chance of losing the pregnancy. During late gestation, the immune system reverts to a pro-inflammatory condition, essential for the commencement of labour and delivery. Pro-inflammatory cytokines including IL-1β, IL-6, and TNF-α, as well as chemokines that bring neutrophils and macrophages to the cervix and myometrium, help the cervix ripen, the membranes break, and the uterus contract. Consequently, pregnancy is characterised not by immunosuppression but by dynamic immune regulation, oscillating between pro-inflammatory and anti-inflammatory phases in response to physiological demands. These typical immunological alterations are evident in maternal haematologic profiles. During pregnancy, a physiological leukocytosis occurs, mostly due to neutrophilia, while lymphocyte numbers exhibit a relative decrease. This pattern shows that the innate immune system is more active during pregnancy and that the adaptive immune system is less active to protect against foetal rejection. Nevertheless, when these immunological adaptations are dysregulated, as observed in gestational hypertension (GH), the maternal immune response transitions from a balanced state to pronounced systemic inflammation. Women with gestational hypertension (GH) show changes in their leukocyte profiles, such as more active neutrophils, less effective regulatory T-cells, and T-helper cells that respond more to Th1 and Th17 cytokines than to Th2-mediated tolerance. This pro-inflammatory imbalance leads to endothelial dysfunction, oxidative stress, and aberrant placentation, all of which are key factors in the pathogenesis of GH. Moreover, elevated circulation concentrations of inflammatory mediators, including TNF-α, IL-6, and C-reactive protein, have been documented in GH, underscoring the significance of immunological dysregulation in its pathogenesis. In summary, our data demonstrate that GH is not solely a haemodynamic illness but also an immunologically mediated condition, wherein disturbances in the meticulously regulated immune equilibrium of pregnancy lead to maternal and foetal problems [8].

While the majority of material focusses on preeclampsia, there is a growing body of research documenting leukocyte disturbances in GH. Studies on large groups of people have shown that high white blood cell counts, a lot of neutrophils, and activated monocytes are all linked to the development of GH. Additionally, derived haematological indices, including the neutrophil-to-lymphocyte ratio (NLR), monocyte-to-HDL ratio (MHR), and systemic immune-inflammation index (SII), are under investigation as potential prognostic markers [9].

This review offers a thorough synthesis of leukocyte differences in GH, elucidating their mechanistic significance and diagnostic/clinical consequences, while prioritising the incorporation of recent information within a pathophysiological framework.

2. Physiological Leukocyte Modifications in Normal Gestation

2.1 Initial Leukocyte Dynamics

Pregnancy causes changes in the blood that show how the mother's immune system accepts the foetus and gets ready for birth. Leukocytosis is a consistently observed phenomenon, characterised by a steady increase in total white blood cell counts throughout gestation. It has been shown that pregnancy boosts the upper reference limit of WBC counts by about 36% compared to women who are not pregnant. This is mostly because neutrophils grow in size.

Neutrophilia indicates augmented innate immunity, potentially induced by placental-derived granulocyte colony-stimulating factor (G-CSF) and elevated cortisol levels. On the other hand, lymphocyte levels go down a little, which means that adaptive immunity is no longer working. This relative lymphopenia may be crucial for maternal–fetal tolerance [10].

2.2 Balance of the Immune System During Pregnancy

Pregnancy entails a dynamic equilibrium between pro-inflammatory and anti-inflammatory phases. In the first trimester, regulated inflammation is the main thing that happens, which helps trophoblasts invade. The second trimester is mostly anti-inflammatory, which helps the foetus thrive. The third trimester brings inflammation back to life, which leads to labour. So, changes in leukocyte levels throughout pregnancy reflect the changing immune system needs of pregnancy.

3. Changes in White Blood Cells in Gestational Hypertension

3.1 Total Counts of White Blood Cells

Numerous studies indicate that WBC levels are increased in women with GH. A substantial Chinese cohort research ($n > 10,000$) revealed that elevated maternal WBC counts during pregnancy were associated with heightened risks of gestational hypertension (GH), irrespective of confounding variables. Zhou et al. [6] similarly indicated that maternal leukocytosis correlated with the onset of HDP, encompassing GH. These results indicate that systemic leukocytosis signifies increased inflammation and endothelial dysfunction prior to manifest hypertension.

The temporal pattern is significant: elevations in WBC during early to mid-gestation seem to forecast subsequent GH, suggesting that leukocyte dysregulation may precede clinical illness presentation [10].

3.2 Neutrophils and Lymphocytes

GH is correlated with a more significant neutrophilia compared to normal pregnancy [11]. In hypertensive pregnancies, activated neutrophils secrete reactive oxygen species, proteases, and neutrophil extracellular traps (NETs), which lead to vascular injury and placental malperfusion [12].

Lymphopenia in GH indicates the reduction of adaptive immunity. Reduced regulatory T cell subsets exacerbate tolerance, hence intensifying systemic inflammation. Neutrophilia and lymphopenia together raise the NLR, which is a sign that is always linked to HDP.

3.3 Neutrophil-to-Lymphocyte Ratio (NLR)

NLR is an easily accessible index obtained from standard blood counts. Meta-analyses indicate that NLR is increased in HDP, encompassing women subsequently diagnosed with GH [13]. It has shown that both the NLR and the platelet-to-lymphocyte ratio (PLR) were much greater in people with preeclampsia and GH than in people without these conditions.

While NLR more effectively differentiates severity in preeclampsia, numerous studies suggest its predictive value for gestational hypertension, particularly when assessed in early gestation [14]. Clinically, NLR may function as a cost-effective supplementary biomarker.

3.4 Monocytes and Their Ratios

Monocytes are important for inflammation in blood vessels. It demonstrated that monocyte activation has a role in endothelial dysfunction during hypertensive pregnancies. Persistent monocyte changes have been observed postpartum in women with a history of HDP, suggesting long-term immunological memory.

The monocyte-to-HDL cholesterol ratio (MHR) and the neutrophil-to-HDL ratio (NHR) combine mechanisms for inflammation and lipid metabolism. In cohorts with hypertension, elevated MHR and NHR have been seen, indicating synergistic roles of dyslipidaemia and leukocyte activation in illness development [15].

3.5 Composite Systemic Inflammation Indices

Indices like SII (platelets × neutrophils/lymphocytes), SIRI (neutrophils × monocytes/lymphocytes), and PIV (neutrophils × monocytes × platelets/lymphocytes) provide us a better picture of systemic inflammation. Reports

indicate that increased SII is linked to preeclampsia and gestational hypertension (GH). Likewise, elevated SIRI levels were observed in women with HDP. Although encouraging, GH-specific validation remains constrained [16].

3.6 Eosinophils and Basophils

Eosinophil and basophil numbers exhibit variable alterations in GH, in contrast to neutrophils and monocytes. Systematic reviews indicate that these leukocyte subsets are not reliable indicators of HDP [17].

4. Mechanistic Pathways Connecting Leukocytes and GH

4.1 Malperfusion of the Placenta and Problems with the Endothelium

In gestational hypertension, dysfunctional remodelling of spiral arteries results in placental hypoperfusion and oxidative stress. This causes the production of inflammatory cytokines (IL-6, TNF-α) and anti-angiogenic factors (sFlt-1), which makes leukocytes stick to each other and damages blood vessels.

Additionally, Haemostatic Activation in HDP:

Hypertensive disorders of pregnancy (HDP), including gestational hypertension (GH), are becoming recognised as being closely associated with substantial haemostatic changes that signify the complex vascular and inflammatory environment of pregnancy. Pregnancy is a hypercoagulable state that reduces the risk of haemorrhage during parturition. In GH and other HDP, this balance is worsened, leading to pathological clotting and damage to the endothelium.

Biochemical data supports this alteration. In affected women, elevated levels of fibrinogen and D-dimer indicate a hypercoagulable state and increased fibrin turnover, signifying that the coagulation cascade remains active. At the same time, a longer prothrombin time (PT) and activated partial thromboplastin time (APTT) show that clotting factors are running low and that the haemostatic system is starting to adjust to this by using compensatory mechanisms. These irregularities highlight the dynamic interplay between coagulation activation and consumption coagulopathy. Additionally, increased circulating levels of tissue plasminogen activator (t-PA) have been seen, possibly signifying a compensatory augmentation of fibrinolysis to mitigate excessive thrombin generation and fibrin deposition [15].

This dysregulated haemostatic state has profound implications for the vascular health of both the placenta and the mother. Hypercoagulability, in conjunction with impaired fibrinolysis, facilitates the formation of placental microthrombi, resulting in localised ischaemia, inadequate uteroplacental perfusion, and ultimately, a deceleration of foetal growth and development. These coagulation defects exacerbate vascular dysfunction by elevating stress and injury to the endothelium at the maternal level. This is very essential because endothelial dysfunction is a big indicator of GH.

When these changes in haemostasis occurs at the same time as inflammation induced by white blood cells, they make blood vessels hurt more and more. When leukocytes are activated, they release pro-inflammatory cytokines and reactive oxygen species. These substances hurt the endothelium even more and make pro-coagulant activity greater by making more tissue factor and less nitric oxide available. This creates a synergistic link between inflammation and coagulation, where problems with the immune system make thrombotic tendencies worse, and problems with clotting then make inflammatory damage worse [16].

Consequently, GH can be conceptualised not only as a failure in blood pressure management but also as a syndrome marked by immune-coagulation interaction, wherein vascular dysfunction arises from the interplay of systemic inflammation and a hypercoagulable milieu. This expanded mechanistic framework clarifies the contribution of haemostatic imbalance to maternal complications, including stroke, thrombosis, and disseminated intravascular coagulation, along with adverse foetal outcomes, such as intrauterine growth restriction and preterm birth.

4.2 Activation of Neutrophils and NETosis

When neutrophils are activated, they release NETs, which are webs of DNA and protein that are outside of cells. NETs catch infections but also hurt the endothelium. NETs have been identified in hypertensive pregnancies, suggesting their involvement in vascular dysfunction [18].

4.3 Communication Between Monocytes and Endothelium

Routine haematological markers have emerged as potentially valuable for forecasting gestational hypertension (GH), especially when integrated with established clinical risk factors such as maternal age, parity, body mass index, familial history of hypertension, and previous obstetric difficulties. Because blood tests are cheap, don't need much effort, and can be done in places with few resources, they should be included in regular antenatal care to improve early detection approaches and risk classification frameworks.

Total white blood cell (WBC) counts among these indices signify the heightened immunological and inflammatory activity linked to GH. Elevated white blood cell counts, particularly neutrophilia, have been noted in women who later experience hypertensive disorders of pregnancy, suggesting that systemic inflammation precedes the appearance of clinical symptoms. The neutrophil-to-lymphocyte ratio (NLR), which combines both innate (neutrophils) and adaptive (lymphocytes) immune components into one number, has become popular as a sensitive way to detect subclinical inflammation. An elevated NLR is recognised as an indicator of gestational hypertension (GH) and its advancement to

preeclampsia, highlighting the balance between pro-inflammatory and regulatory immune responses throughout pregnancy.

Along with WBC and NLR, composite systemic inflammation indices such as the platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), and derived neutrophil-to-lymphocyte ratio (dNLR) give us further information that could help us guess what would happen. These indices integrate blood tests that demonstrate both inflammatory and haemostatic pathways, indicating how leukocyte activation, platelet reactivity, and endothelial dysfunction operate together. A high PLR may indicate that platelets are exacerbating GH's hypercoagulable condition, whereas the SII employs counts of neutrophils, lymphocytes, and platelets to provide a more comprehensive assessment of systemic inflammatory burden.

The accuracy of predictions is much improved when these blood markers are looked at along with other common clinical risk factors. This type of integration allows for the development of risk prediction models that categorise pregnant women into low, medium, and high-risk groups for GH. Finding women who are at risk early on could help maintain a closer eye on them, make changes to their lifestyle on time, or give them low-dose aspirin as a preventive intervention. This could lead to fewer unfavourable outcomes for mothers and newborns.

These blood tests are part of a typical complete blood count (CBC), which is a common test used in prenatal care. This means that they could be used in clinical practice without needing expensive or specialised lab equipment. This makes them especially beneficial in low- and middle-income countries, where it may be tougher to get advanced biochemical and imaging testing to predict GH.

4.4 Innate Immunity That Has Been Trained

Evidence indicates that monocytes display trained immunity—epigenetically modified responses—subsequent to hypertensive pregnancies. This may elucidate enduring cardiovascular risk subsequent to GH [19].

5. Implications for Clinical Practice

5.1 Assessing Risk and Screening

Routine haematological markers have emerged as potentially valuable for forecasting gestational hypertension (GH), especially when integrated with established clinical risk factors such as maternal age, parity, body mass index, family history of hypertension, and previous obstetric difficulties. Because blood tests are cheap, don't hurt, and can be done in places with few resources, they should be a part of normal prenatal care to improve early diagnosis and risk assessment [20].

The total white blood cell (WBC) counts among these indicators signify the heightened immunological and inflammatory activity linked to GH. Elevated white blood cell counts, particularly neutrophilia, have been noted in women who later experience hypertensive disorders of pregnancy, suggesting that systemic inflammation precedes the appearance of clinical symptoms. The neutrophil-to-lymphocyte ratio (NLR) combines innate (neutrophils) and adaptive (lymphocytes) immune components into one indicator. It has become popular as a sensitive way to detect subclinical inflammation. An elevated NLR is recognised as an indicator of gestational hypertension (GH) and its advancement to preeclampsia, highlighting the balance between pro-inflammatory and regulatory immune responses during pregnancy [21].

Along with WBC and NLR, composite systemic inflammation indicators such as the platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), and derived neutrophil-to-lymphocyte ratio (dNLR) offer additional data that may assist in outcome prediction. These indices incorporate blood tests that reveal both inflammatory and haemostatic pathways, illustrating the interplay of leukocyte activation, platelet reactivity, and endothelial dysfunction. A high PLR may indicate that platelets are contributing to GH's hypercoagulable state, whereas the SII employs counts of neutrophils, lymphocytes, and platelets to provide a more comprehensive assessment of systemic inflammatory burden [22].

When these haematological markers are analysed alongside traditional clinical risk factors, the precision of predictions is significantly improved. This type of integration enables the development of risk prediction models that categorise pregnant women into groups according to their risk for GH: low, medium, and high. Finding women who are at risk early on could help keep an eye on them more closely, make changes to their lifestyle on time, or give them low-dose aspirin as a prophylactic intervention. This could lead to fewer unfavourable outcomes for mothers and newborns [23].

These blood tests are part of a typical complete blood count (CBC) test, which is now often used in routine prenatal care. This means that they could be used in clinical practice without the need for expensive or specialised lab equipment. This makes them very helpful in low- and middle-income countries, where it may be tougher to receive advanced biochemical and imaging testing to predict GH.5.2 Differentiation from Preeclampsia. Leukocyte markers alone are insufficient to accurately differentiate gestational hypertension from preeclampsia. Combining haematological parameters with angiogenic markers (sFlt-1/PlGF ratio) enhances diagnostic precision [24].

5.2 Long-Term Risk for Mothers

Persistent leukocyte changes after giving birth show that GH increases the risk of long-term heart problems through chronic low-grade inflammation. Keeping an eye on inflammatory profiles could help with long-term prevention plans [25].

6. Suggestions and Directions for Further Research

1. GH-specific cohorts: Most research combine GH and preeclampsia. We need dedicated GH cohorts.

2. High-dimensional immune profiling: Flow cytometry and single-cell RNA-seq can identify leukocyte subsets.

3. Combining leukocyte indices with lipidomics and angiogenic biomarkers may help make early predictions more accurate.

4. Postpartum immune surveillance: Examining leukocyte persistence following GH may elucidate cardiovascular risk pathways.

7. Conclusion

Gestational hypertension is linked to substantial leukocyte changes, including increased WBC counts, neutrophilia with lymphopenia (↑NLR), monocyte activation, and raised systemic inflammatory indices. These findings indicate an intensified pro-inflammatory environment that leads to endothelial dysfunction and hypertension. Leukocyte-based indices show potential as supplementary biomarkers; nevertheless, specialised study on GH is necessary for validation and clinical use. In the end, combining leukocyte indicators with angiogenic and metabolic indices may make it easier to predict, diagnose, and manage long-term.

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