

# Thrombocytopenia in Pregnancy: Insights from Automated Platelet Analysis

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## Abstract

## Original Research Article

Thrombocytopenia is a frequent hematological abnormality during pregnancy, resulting from physiological adaptations or pathological conditions affecting platelet production or destruction, and its accurate evaluation is essential for optimal maternal and fetal management. This prospective descriptive study, conducted at the Mohammed VI University Hospital of Marrakech, included 100 hospitalized pregnant or postpartum women with hematological abnormalities, among whom 49 presented with thrombocytopenia. The aim was to assess the etiological profile of gestational thrombocytopenia and the contribution of advanced automated platelet parameters. Complete blood counts were performed using the Sysmex XN-3100 analyzer, combining impedance and fluorescence platelet counting (PLT-F) as well as immature platelet fraction (IPF) measurement. Thrombocytopenia was predominantly observed in the third trimester and mainly associated with HELLP syndrome and severe preeclampsia. Fluorescence platelet counts were consistently higher than impedance counts, leading to reclassification of platelet levels in several patients and reducing unnecessary diagnostic investigations. The IPF was significantly elevated in thrombocytopenic patients (mean 12.25%), particularly in HELLP syndrome, acute fatty liver of pregnancy, and immune thrombocytopenic purpura, reflecting increased bone marrow platelet production and compensatory thrombopoietic activity. Peripheral blood smear examination provided complementary morphological information, including schistocytes, anisopoikilocytosis, and macroplatelets, supporting etiological orientation. Overall, the integration of fluorescence platelet counting and IPF into routine obstetric practice enhances diagnostic accuracy and supports more appropriate clinical decision-making in the management of thrombocytopenia during pregnancy.

**Keywords:** Thrombocytopenia – Pregnancy – Gestational thrombocytopenia – HELLP syndrome – Acute fatty liver of pregnancy – Fluorescence platelet count – Immature platelet fraction.

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## 1. INTRODUCTION

Pregnancy is accompanied by significant hematological changes, among which thrombocytopenia is a common abnormality, linked either to physiological mechanisms such as hemodilution or to pathological conditions that alter platelet production or destruction [1].

The complete blood count using platelet impedance remains the main test for screening and monitoring these abnormalities. Manual platelet count constitutes the classical gold standard replaced since 2000s by flow cytometry based on CD41 and/or CD61 detection. Some studies show that fluorescence platelet detection by specific RNA fluorochrome (PLT-F-Sysmex) is equivalent to the gold standard method. The integration of advanced hematological parameters, in

particular immature platelet fraction (IPF); obtained both in reticulocyte channel in many systems and PLT-F channel in Sysmex channel; provides additional information on the dynamics of thrombopoiesis, allowing for a better distinction between central and peripheral thrombocytopenia and a more accurate assessment of bone marrow response [2]. The use of these tools in pregnant women contributes to a more refined stratification of bleeding risk, individualized monitoring, and a reduction in invasive investigations, improving the diagnostic and prognostic management of thrombocytopenia in an obstetric context [3].

## 2. MATERIAL AND METHODS

This is a prospective, descriptive study conducted to analyze thrombocytopenia during pregnancy by evaluating platelet abnormalities in blood

counts and the contribution of new automated hematological parameters to the management of pregnant patients. The study was carried out at the hematology laboratory of Errazi Hospital, in collaboration with the gynecology-obstetrics department and the gynecological-obstetric intensive care unit of the Mother and Child Hospital at the Mohammed VI University Hospital Center.

A total of 100 hospitalized patients aged 18 years and older presenting with hematological abnormalities were included. Among them, 49 patients with thrombocytopenia constituted the main study population. In this study, thrombocytopenia was defined as a platelet count <120 G/L. Hematological analyses were performed using an automated hematology analyzer (Sysmex XN-3100).

### 3. RESULTS

A total of 100 patients were included in our study on hematological disorders in the gynecology-obstetrics and gynecology-obstetrics intensive care unit at the mother and Child Hospital of the Mohammed VI University Hospital in Marrakech. Data analysis revealed 49 cases of thrombocytopenia (34.69% isolated and 65.3% associated with other abnormalities), while three cases of thrombocytosis were associated with anemia.

Epidemiological analysis showed that the average age of thrombopenic patients was 30.7 years, with the most affected age group being between 25 and 35 years (40.82%). Thrombocytopenia was detected mainly in the third trimester (87.79%).

Regarding medical history, 20.4% of thrombopenic patients had a history of medical or surgical conditions, mainly obstetric (8.16%) and endocrine (6.12%), and 28.57% had a history of abortion. The majority of thrombopenic patients were multiparous (59.18%) and only 40.82% were primiparous. Among those who gave birth, 80.56% had a cesarean section and 19.44% had a vaginal delivery.

Clinically, the majority of hospitalizations for thrombocytopenia were for HELLP syndrome and severe preeclampsia (61.22%), followed by gestational thrombocytopenia (20.4%), acute fatty liver of pregnancy (AFLP) (12.24%), and immune thrombocytopenic purpura (ITP) (6.12%).

Hemorrhagic signs were observed in a limited number of patients: purpuric spots (8.16%), gingival bleeding (12.24%), and hematuria (2.04%).

Impedance platelet count (IPC) showed an average of 71,000/mm<sup>3</sup>, with 30.61% of patients <50,000/mm<sup>3</sup>, 40.82% between 50,000 and 100,000/mm<sup>3</sup>, and 28.57% between 100,000 and 150,000/mm<sup>3</sup>. According to etiology, the average count was 58,000/mm<sup>3</sup> for HELLP, 85,000/mm<sup>3</sup> for gestational thrombocytopenia, 39,000/mm<sup>3</sup> for AFLP, and 22,000/mm<sup>3</sup> for ITP.

Fluorescence counting (FLC) revealed higher values, with an overall average of 91,000/mm<sup>3</sup> and extremes ranging from 18,000 to 216,000/mm<sup>3</sup>. By etiology, the mean FLC was 77,000/mm<sup>3</sup> for HELLP, 105,000/mm<sup>3</sup> for gestational thrombocytopenia, 48,000/mm<sup>3</sup> for AFLP, and 51,000/mm<sup>3</sup> for ITP.

Analysis of associated erythrocyte and leukocyte indices showed that 46.9% of thrombopenic patients had concomitant anemia (mean Hb 9.78 g/dL) and 24.48% had neutrophilic hyperleukocytosis (mean 12,748/mm<sup>3</sup>).

Peripheral blood smears revealed the presence of schistocytes (24.4%), anisopoikilocytosis and erythrocyte poikilocytosis (8.16%), polymorphic hyperlymphocytosis (2.04%), and macroplatelets or giant platelets (2.04%).

The mean immature platelet fraction (IPF) was 12.25% in thrombopenic patients, with 71.4% having an IPF >7.4%, and varied according to etiology: HELLP 14%, gestational thrombocytopenia 11.34%, AFLP 16%, ITP 15.04%. Non-thrombopenic patients had a mean IPF of 5.7%.

<b>Etiology of thrombocytopenia</b>	<b>Mean Impedance platelet count (IPC)</b>	<b>Mean Fluorescence counting (FLC)</b>	<b>mean immature platelet fraction (IPF)</b>
HELLP syndrome	58,000/mm <sup>3</sup>	77,000/mm <sup>3</sup>	14%
gestational thrombocytopenia	85,000/mm <sup>3</sup>	105,000/mm <sup>3</sup>	11.34%
fatty liver of pregnancy	39,000/mm <sup>3</sup>	48,000/mm <sup>3</sup>	16%
immune thrombocytopenic purpura	22,000/mm <sup>3</sup>	51,000/mm <sup>3</sup>	15.04%.

Treatment was stratified according to the underlying etiology and clinical presentation. In hypertensive disorders of pregnancy, including HELLP syndrome and severe preeclampsia, alpha-methyldopa was the most frequently used antihypertensive agent (61.2%). Calcium channel blockers (NICARDIPINE) was administered either intravenously (34.6%) or orally

(55.1%). Antihypertensive management consisted of monotherapy in 10% of cases and combination therapy in 90%. Magnesium sulfate was used in 60% of patients with HELLP syndrome for seizure prophylaxis.

In immune thrombocytopenic purpura (ITP), corticosteroid therapy was systematically used in all

patients. One patient with gestational thrombocytopenia received corticosteroids due to severe thrombocytopenia. Blood products were administered to 9 patients (18.3%).

The majority of deliveries were by cesarean section (80.5%) for obstetric or maternal reasons, and 19.4% delivered vaginally.

The maternal prognosis was generally favorable, with complications found in 32.6% of patients (eclampsia 8.16%, acute renal failure 28.5%, retroplacental hematoma 4.08%, subcapsular hematoma of the liver 2.04%) and no maternal deaths.

The fetal prognosis revealed a 10.2% neonatal mortality rate, 23% with prematurity, and 5 newborns hospitalized for respiratory distress.

#### 4. DISCUSSION

Pregnancy is a unique physiological state marked by significant hormonal changes, leading to alterations in numerous biological parameters, including an increase in total blood volume of approximately 1.5 liters, intended to meet the demands of the new vascular bed and compensate for blood loss associated with childbirth [4]. Large cross-sectional studies conducted in healthy pregnant women, excluding those with hypertension, have shown a gradual decrease in platelet count during pregnancy, which is particularly marked in the third trimester. This phenomenon corresponds to gestational thrombocytopenia, the pathophysiology of which is based on both pregnancy-related hemodilution and increased platelet consumption in the placenta [5]. This thrombocytopenia is generally moderate, with platelet concentrations most often above  $75 \times 10^9/L$  [6]. The platelet count increases after delivery in response to and as compensation for the increased platelet consumption during the delivery process [4].

In this context, the etiologies of thrombocytopenia occurring during pregnancy are multiple and can be subdivided into two main categories: obstetric and non-obstetric causes of thrombocytopenia. Their occurrence is related to gestational age, and four main diagnostic groups should be systematically considered due to their frequency: gestational thrombocytopenia, pregnancy-specific hypertensive and metabolic disorders (preeclampsia, HELLP syndrome, acute fatty liver of pregnancy), immune thrombocytopenic purpura (ITP), and thrombotic microangiopathies. Other, rarer causes may also be for thrombocytopenia during pregnancy and therefore require additional etiological investigations [7].

The etiological analysis carried out in our study showed that preeclampsia and HELLP syndrome were the most common causes of gestational thrombocytopenia, accounting for 61.2% of cases. Gestational thrombocytopenia ranked second with a rate of 20.4%, followed by acute fatty liver of pregnancy in

12.24% of cases. These results differed from those reported in the literature, particularly in Kouhen's study of 43 patients, in which gestational thrombocytopenia was the predominant etiology [8].

Thrombocytopenia may be discovered in the presence of hemorrhagic symptoms suggestive of a primary cutaneous-mucosal hemostasis disorder. A retrospective study published by Webert *et al* involving 119 pregnancies complicated by thrombocytopenia showed that the majority of pregnancies (76 cases, or 65.5%) progressed without hemorrhagic complications [9]. Mild clinical manifestations, such as ecchymosis or purpura, were observed in 12.9% of patients, while moderate symptoms (epistaxis, post-traumatic or mucosal bleeding) affected 21 patients. Severe forms remained rare, occurring in four patients, including one hematoma, two cases of hematuria, and one gastrointestinal bleed [9]. These results were consistent with those of our series of 49 pregnancies, in which the majority of patients (77.55%) were asymptomatic, while mild, moderate, and severe hemorrhagic manifestations were found in 8.16%, 12.24%, and 2.04% of cases, respectively.

Beyond clinical data, the analysis of biological parameters, particularly platelet count using the impedance method, is a determining factor in the evaluation of thrombocytopenia during pregnancy. In our series, the distribution according to the severity of thrombocytopenia differed significantly from that reported in the literature. Compared to Boehlen's study [7], which found 1% of severe thrombocytopenia ( $< 50,000/mm^3$ ), 20% moderate thrombocytopenia ( $50,000-100,000/mm^3$ ) and 79% moderate thrombocytopenia ( $100,000-150,000/mm^3$ ), our study showed significantly higher proportions of severe (30.61%) and moderate (40.82%) forms, with a lower proportion of moderate forms (28.75%). This could be explained by the severity of thrombocytopenia in our patients.

In addition to platelet count, peripheral blood smear examination provides key morphological information for understanding the mechanisms of thrombocytopenia. In our series, the presence of schistocytes (12 cases, 24.4%) suggested intravascular fragmentation of red blood cells, possibly linked to thrombotic microangiopathies such as HELLP syndrome or acute fatty liver of pregnancy [10]. Anisopoikilocytosis and erythrocyte poikilocytosis (4 cases, 8.16%) reflected alterations in the size and shape of red blood cells, which may be indicative of hemolytic anemia [11]. Reactive polymorphic lymphocytosis (1 case, 2.04%) could indicate an immune or inflammatory response, while the presence of macroplatelets and giant platelets (1 case, 2.04%), reflected an increased thrombopoietic response to thrombocytopenia, which could be associated with hemolytic syndrome or other conditions causing platelet destruction or reduction.

These findings underscored the morphological diversity and confirmed the utility of peripheral blood smears in diagnosing and managing gestational thrombocytopenia [12].

The introduction of flow cytometry applied to platelet counting, using specific RNA fluorochrome labeling, has enabled highly precise measurements to be performed, particularly for [13,14,15].

This precise determination of platelet count is particularly crucial in pregnant women in order to optimize care. In our series, fluorescence platelet counting revealed significantly higher values than those obtained by impedance, allowing several patients to be reclassified as having gestational thrombocytopenia and thus avoiding unnecessary investigations [16].

The immature platelet fraction (IPF) reflects the proportion of platelets recently released by megakaryocytes and is a reliable marker of thrombopoietic activity [17,18]. Similar to reticulocytes, these immature platelets are larger and rich in cytoplasmic RNA, with their proportion increasing in response to stimulated bone marrow production and decreasing as platelets age [19,20]. In clinical practice, a cut-off value of approximately 7% on the Sysmex PLT-F channel is commonly used to distinguish peripheral platelet destruction from impaired platelet production [28]. IPF therefore may provide an early indicator of platelet regeneration, which is useful for guiding treatment and limiting unnecessary invasive procedures or platelet transfusions and their associated risks [21].

In our series, IPF values varied according to etiology: high in HELLP syndrome (14%) and acute fatty liver of pregnancy (16%), moderate in gestational thrombocytopenia (11.34%) and immune thrombocytopenic purpura (15%), reflecting bone marrow responses adapted to the specific compensatory needs of each pathology [22,23,23]. These results were consistent with the literature, where IPF can be used to predict bone marrow regeneration after chemotherapy or to evaluate therapeutic efficacy [19]. Thus, IPF allows the dynamics of platelet production to be assessed, with a marked increase suggesting a compensatory bone marrow response, while a moderate increase reflects physiological adaptations or specific pathological reactions.

The management of thrombocytopenia during pregnancy depends on both the obstetric context and the etiology. Regarding the mode of delivery, vaginal delivery is reported to be predominant in the studies by Kouhen [8] and Webert [9], with rates of 81% and 82%, respectively. However, in our series, only 19.4% of patients delivered vaginally, reflecting a more frequent use of cesarean section. This difference could be explained by the predominance of HELLP syndrome as the main cause of thrombocytopenia in our population, a

condition often associated with severe forms requiring rapid fetal extraction. The choice of delivery method therefore appears to be closely linked to the severity of thrombocytopenia and the maternal-fetal context.

The therapeutic management of thrombocytopenia varied according to its etiology. In gestational thrombocytopenia, therapeutic abstention was generally sufficient, as reported in Kouhen's study, in which none of the 30 patients required specific treatment [8]. In our series, among the 10 patients with gestational thrombocytopenia, only one received a bolus of corticosteroids (20 mg/24 h intravenous betamethasone) due to severe thrombocytopenia with a platelet count below 50 G/L. This difference compared with the data in the literature could be explained by the more severe biological profiles observed in our population.

In cases of preeclampsia and HELLP syndrome, antihypertensive treatment was an essential part of management. In the study conducted at the Fez University Hospital [24], dual therapy combining a calcium channel blocker (nicardipine) with a central antihypertensive agent (alpha-methyldopa) was administered to 55.8% of patients, while 39.55% received monotherapy. In our series, dual therapy was used in 90% of patients, while monotherapy with alpha-methyldopa was used in only 10% of cases, which may reflect the greater severity of the forms of preeclampsia observed. Furthermore, contrary to this study, in which no patients received magnesium sulfate, this anticonvulsant treatment was administered to 60% of patients in our series, again underlining the severity of the clinical presentations.

The management of acute fatty liver of pregnancy (AFLP) was based on early delivery, recognized as a key factor in improving maternal and fetal prognosis. In our series, all patients with AFLP benefited from emergency fetal extraction [25].

Finally, for immune thrombocytopenic purpura, the therapeutic management differed from that reported in Webert's series [9], where 45% of patients received treatment, including corticosteroids, intravenous immunoglobulins (IVIg) alone or in combination. In our series, the three patients with ITP were treated exclusively with corticosteroids, mainly due to the unavailability of intravenous immunoglobulins.

No maternal deaths were reported in our series, reflecting an overall favorable maternal prognosis. However, several serious complications were observed. In Sibai's study of 437 women with HELLP syndrome, 60% of patients delivered by cesarean section, 10% had eclampsia, 5% had renal failure, and 5% had retroplacental hematoma [26]. Our results are consistent with these data for the incidence of eclampsia and renal failure, while differences are observed for hemorrhagic

complications, such as retroplacental hematoma and subcapsular hematoma of the liver. Furthermore, when comparing perinatal mortality, our series found a rate of 10%, similar to the average of 13.5% reported by Haddad, confirming a concordance with published data on fetal prognosis in HELLP syndrome [27].

## 5. CONCLUSION

In conclusion, thrombocytopenia is a common hematological abnormality during pregnancy, particularly in pathological contexts such as HELLP syndrome, preeclampsia, or acute fatty liver of pregnancy. Automated blood counts, enhanced by advanced parameters such as the immature platelet fraction (IPF), provide an accurate assessment of platelet production and help differentiate between central and peripheral thrombocytopenia. Although IPF provides information on thrombopoiesis, its usefulness alone remains limited and must be interpreted in the clinical context and in combination with other parameters. The integration of these data into obstetric practice allows for targeted surveillance, anticipation of hemorrhagic complications, and more informed therapeutic decision-making, optimizing maternal and fetal safety. These results underscore the importance of a combined approach, combining conventional measurements and innovative parameters, for the effective management of patients with platelet abnormalities during pregnancy.

## REFERENCES

1. Yahyaoui G, Bensedik N, Benjelloun S, Tlamçani I. Variations physiologiques de l'hémogramme au cours de la grossesse. *REVUE DE SYNTHÈSE*. 2013 ;2.
2. Tessier-Marteau A, Geneviève F, Godon A, Macchi L, Zandecki M. [Automated hematology analysers and spurious counts. Part 1. Platelets]. *Ann Biol Clin (Paris)*. 2010 ;68(4) :393-407.
3. Hématologie physiologique de la grossesse - EM consulte [Internet]. [Cité 18 janv 2026]. Disponible sur: <https://www.em-consulte.com/article/694234/hematologie-physiologique-de-la-grossesse>
4. Chandra S, Tripathi AK, Mishra S, Amzarul M, Vaish AK. Physiological changes in hematological parameters during pregnancy. *Indian J Hematol Blood Transfus*. sept 2012;28(3):144-6.
5. Shehata N, Burrows R, Kelton JG. Gestational thrombocytopenia. *Clin Obstet Gynecol*. Juin 1999 ;42(2):327-34.
6. Masson E. EM-Consulte. [cité 18 janv 2026]. Thrombopénie et grossesse. Disponible sur : <https://www.em-consulte.com/article/739017/thrombopenie-et-grossesse>
7. Boehlen F, Hohlfeld P, Extermann P, Perneger TV, de Moerloose P. Platelet count at term pregnancy: a reappraisal of the threshold. *Obstet Gynecol*. Janv 2000 ;95(1):29-33.
8. Thrombopénie et grossesse : résultats d'une étude prospective à propos de 43 cas - EM consulte [Internet]. [cité 18 janv 2026]. Disponible sur: <https://www.em-consulte.com/article/233633/thrombopenie-et-grossesse-resultats-dune-etude-pro>
9. Webert KE, Mittal R, Sigouin C, Heddle NM, Kelton JG. A retrospective 11-year analysis of obstetric patients with idiopathic thrombocytopenic purpura. *Blood*. 15 déc 2003 ;102(13):4306-11.
10. Coppo P, Veyradier A. Microangiopathies thrombotiques: physiopathologie, diagnostic et traitement. *Réanimation*. nov 2005;14(7):594-603.
11. Bandaru SS, Killeen RB, Gupta V. Poikilocytosis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cité 18 janv 2026]. Disponible sur: <http://www.ncbi.nlm.nih.gov/books/NBK562141/>
12. Lynch EC. Peripheral Blood Smear. In: Walker HK, Hall WD, Hurst JW, éditeurs. *Clinical Methods: The History, Physical, and Laboratory Examinations* [Internet]. 3rd éd. Boston : Butterworths ; 1990 [cité 18 janv 2026]. Disponible sur: <http://www.ncbi.nlm.nih.gov/books/NBK263/>
13. Schoorl M, Schoorl M, Oomes J, van Pelt J. New fluorescent method (PLT-F) on Sysmex XN2000 hematology analyzer achieved higher accuracy in low platelet counting. *Am J Clin Pathol*. oct 2013;140(4):495-9.
14. Briggs C, Longair I, Kumar P, Singh D, Machin SJ. Performance evaluation of the Sysmex haematology XN modular system. *J Clin Pathol*. nov 2012;65(11):1024-30.
15. Seo JY, Lee ST, Kim SH. Performance evaluation of the new hematology analyzer Sysmex XN-series. *Int J Lab Hematol*. avr 2015;37(2):155-64.
16. Wang Z, Jin X, Wang S, You Q, Wang J, Xu D. Selection of Automated Platelet Counting Methods Based on Mean Platelet Volume (MPV). *Clin Lab*. 1 mai 2023;69(5).
17. van der Linden N, Klinkenberg LJJ, Meex SJR, Beckers EAM, de Wit NCJ, Prinzen L. Immature platelet fraction measured on the Sysmex XN hemocytometer predicts thrombopoietic recovery after autologous stem cell transplantation. *Eur J Haematol*. août 2014;93(2):150-6.
18. Ferreira FLB, Colella MP, Medina SS, Costa-Lima C, Fiusa MML, Costa LNG, *et al.*, Evaluation of the immature platelet fraction contribute to the differential diagnosis of hereditary, immune and other acquired thrombocytopenias. *Sci Rep*. 13 juin 2017;7(1):3355.
19. Briggs C, Kunka S, Hart D, Oguni S, Machin SJ. Assessment of an immature platelet fraction (IPF) in peripheral thrombocytopenia. *Br J Haematol*. juill 2004;126(1):93-9.
20. Richards EM, Baglin TP. Quantitation of reticulated platelets: methodology and clinical application. *Br J Haematol*. oct 1995;91(2):445-51.

21. Goel G, Semwal S, Khare A, Joshi D, Amerneni CK, Pakhare A, *et al.*, Immature Platelet Fraction: Its Clinical Utility in Thrombocytopenia Patients. *J Lab Physicians*. sept 2021;13(3):214-8.
22. Everett TR, Garner SF, Lees CC, Goodall AH. Immature platelet fraction analysis demonstrates a difference in thrombopoiesis between normotensive and preeclamptic pregnancies. *Thromb Haemost*. juin 2014;111(6):1177-9.
23. Naz A, Mukry SN, Shaikh MR, Bukhari AR, Shamsi TS. Importance of immature platelet fraction as predictor of immune thrombocytopenic purpura. *Pak J Med Sci*. 2016;32(3):575-9.
24. Mamouni N, Bougern H, Derkaoui A, Bendahou K, Fakir S, Bouchikhi C, *et al.*, Le HELLP syndrome: à propos de 61 cas et revue de la littérature. *Pan Afr Med J*. 20 févr 2012; 11:30.
25. Campillo B, Bernuau J, Witz MO, Lorphelin JM, Degott C, Rueff B, *et al.*, Ultrasonography in acute fatty liver of pregnancy. *Ann Intern Med*. sept 1986;105(3):383-4.
26. Sibai BM, Ramadan MK, Usta I, Salama M, Mercer BM, Friedman SA. Maternal morbidity and mortality in 442 pregnancies with hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome). *Am J Obstet Gynecol*. oct 1993;169(4):1000-6.
27. Haddad B, Barton JR, Livingston JC, Chahine R, Sibai BM. Risk factors for adverse maternal outcomes among women with HELLP (hemolysis, elevated liver enzymes, and low platelet count) syndrome. *Am J Obstet Gynecol*. août 2000 ;183(2):444-8.
28. Ko YJ, *et al.*, Reference interval for immature platelet fraction on Sysmex XN hematology analyzer: a comparison study with Sysmex XE-2100. *Clin Chem Lab Med*. 2015;53(7):1091–1097. doi:10.1515/cclm-2014-0839. PMID:25460288