COVID-19 Effectiveness on Platelet Function in Women

Rasha A.Abidalmutalib Aljabawi[1], Shahad dakhil khalaf[2], Walaa Ali Mahdi[3]
[1]Department of Clinical laboratory Sciences,College of Pharmacy,University of Kerbala,Iq
[2]Department of Clinical laboratory Sciences, College Pharmacy,University of Kerbala,Iq
[2]Department of Clinical laboratory Sciences, College Pharmacy,University of Kerbala,Iq

Coressponding author email address : rasha.aziz@uokerbala.edu.iq

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Abstract

For several years, platelets were identified to be the core blood constituent that facilitates hemostasis and blood clots. In the latest decades, understood that platelets also achieve reflective immune purposes throughout infection by numerous pathogens. Now realize that platelets being moreover interceded a response to numerous other RNA viruses like flu and that numerous viral infections, counting severe Covid19, can impact platelet total. Thrombocytopenia and augmented clotting have been individually connected with amplified death .Laboratory features of Covid19 infection didn't differ according to pregnancy situation. A tendency of incomplete lymphocyte count was detected in the pregnant group.

Keywords

COVID-19, immunity, platelets, Covid19, thrombosis, coronavirus in pregnant and non-pregnant women

Conflict of interest

The authors declare that there is no conflict of interest

الخلاصة

تعتبر الصفائح الدموية لسنوات عديده على أنها المكون الأساسي للدم الذي يسهل الإرقاء والجلطات الدموية. عرفت في السنوات الأخيرة مفهوم أن الصفائح الدموية تحقق أيضًا أهدافًا مناعية خلال الإصابة بمسببات الأمراض العديدة. أن الصفائح الدموية قد توسطت أيضًا في الاستجابة للعديد من فيروسات الحمض النووي الريبي الأخرى مثل الأنفلونزا والعديد من الالتهابات الفيروسية .تؤثر الاصابه ب Covid19 الشديدة على عدد الصفائح الدموية. ارتبطت قلة الصفيحات وزيادة التخثر بشكل فردي بزيادة الوفيات. لم تختلف السمات المختبرية لعدوى Covid19 باختلاف حالة الحمل. تم التوصل الى وجود ميل للزياده في عدد الخلايا الليمفاوية لدى النساء الحوامل

1.1Platelet:

Platelets (P) are delivered by megakaryocytes that need genomic DNA [1] however hold megakaryocyte-determined courier RNA (mRNA) and the translational apparatus required for protein production. [2] whenever departure from the bone marrow, platelets flow for about ten days. According to their essential capacity to end blood loss subsequent cells damage and vascular harm [3]. The primary binding of Platelets at locations of vascular injury is facilitated by glycoprotein Ib/V/IX, a mainly extraordinary receptor composite stated in platelets. Von Willebrand factor (VWF) is the important ligand for a single part of this compound, glycoprotein Ib, also the absence of the element causes disorders trendy essential hemostasis and clotting [4]. Other than glycoprotein Ib, a few collagen receptors with a tying role are prearranged on the platelet outward area, relatively a glycoprotein VI and glycoprotein Ia, individuals from the immunoglobulin family [5]. Whenever adhesion of platelets to the extracellular matrix, the maintenance interaction involves a fast reaction to both autocrine and paracrine go-betweens, counting (ADP), thrombin, epinephrine, and thromboxane A2. These intermediaries intensify and support the early platelet action, and will motivate flowing platelets from the blood streaming to achieve a hemostatic pad. Furthermost agonists that trigger platelets are mediated via G-protein–coupled receptors [6].

1.1.2. Platelet Count (PC) in women

The PC distribution in pregnant ladies and non-pregnant women is shown in the Curve diagrams below [7]:

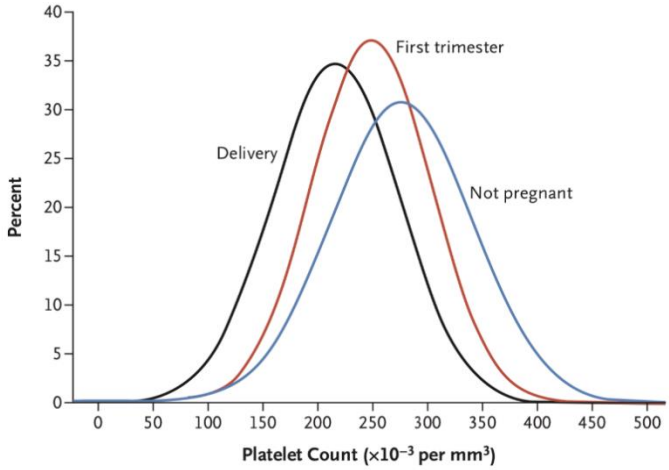

Figure 1. Platelet Count Distribution.

Shown are the dispersion of the normal platelet counts of the nonpregnant women and the amount of the average platelet counts during the primary trimester (mean pregnancy, 8.7 weeks) and at the time of birth in the women who had unchallenged pregnancies. The distributions of the normal platelet count for all three curves were symmetrical, with a minor skew toward elevated platelet counts that were compatible across all three curves. The average and median platelet count account for each of the three measurement curves differed by minor than 3%.

1.2. COVID-19

Humans who have been septic via coronavirus by the way are one of the viruses accountable for the public flu. It is spreadable viral contamination, which is extended via breath and eating of flu drops as a consequence sneezing, coughing, and attached contaminated surfaces are essential causes of disease. The Covid structure is included thousands of nucleotides. It has 4 types of proteins, Spike (S) protein, Nucleocapsid (N) protein, Envelope (E), Membrane (M) protein, protein, and a few non-structural proteins (NSP) (Figure 2).[8]

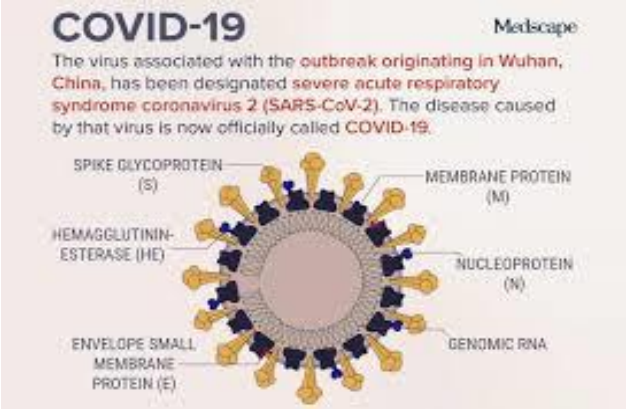

Figure 2. The covid19 surface proteins, spike, cover, and membrane, are surrounded by bilayer lipid. The individual-stranded positive since viral RNA is connected with the nucleocapsid protein[9].

Happening 2019, a new coronavirus (SARS-CoV-2) epidemic happened in a China city called Wuhan. As the head event of pulmonary events was labeled, SARS-CoV-2 infection Covid-19 swiftly extent global, incidence confirmed pandemic contamination on 11 March 2020 via the WHO. Subsequently, 116000000 infections and 2.5 million losses have been stated worldwide until now [9]. Iraq reported its first confirmed cases of SARS-CoV-2 infections on 22 February 2020 in Najaf. [10] The number of cases in Iraq continued to grow exponentially. Now, there are over 708,951 cases and about 13,483 deaths [11].

1.2.3 SARS-CoV-1 plus SARS-CoV-2

The Severe Acute Respiratory Syndrome Coronavirus 1 (SARS-CoV-1) and 2 (SARS-CoV-2). RNA viruses lead to the epidemic that occurred in 2002–2004 called the SARS epidemic and the current COVID-19 pandemic, correspondingly, bind angiotensin-converting enzyme 2 (ACE2), an inherent protein-membrane physiologically responsible by enzymatic activity that the stimulation of the renin-angiotensin-aldosterone system. ACE2 is activated largely, totaling in lung cells, endothelial cells, cardiac tissues, and the renal system. SARS-CoV-2 has approximately a ten-greater attraction for ACE2 compared to SARS-CoV-1 [8].

1.2.4. Pathogenesis COVID-19

The procedure of viral entering and duplication and RNA stuffing in the cell is outlined (Figure 3). The Covid (S) protein links to ACE2 receptors that are systematized on the superficial of several cells, as well as persons in the lungs permitting the virus to enter. The Covid S protein is visible to attack by proteases (proteolytic cleavages) such as trypsin and furin, in double places, located between the S1 and S2 classified as (S1/S2 site). Afterward, the cleavage of the S2 area is occurred (S20 site) to discharge the combination peptide. All latest events will trigger the stimulation of the layer combination process. Considering antibodies that find maintenance on molecular pointing that able to use the component gen (aa sequence) of the adherent unit that originates in(ACE2). In this manner, this procedure could deliver therapy to inhibit the viral passage. Ordinarily, the cell swallows the infection by an action named endocytosis. When passing into the cytosolic, this has been recommended probably that Covid-19 utilizes an interesting 3-stage technique for membrane merging, which includes receptor-binding then get an ensuring alteration in (S) glycoprotein go after cathepsin L proteolysis via proteases enzyme then stimulation of layer merging process in endosomes [12]. At this stage, the endosome exposed to release the virus to the systolic, and uncapping of the viral nucleocapsid (N) is initiated through proteasome, which is normally able to hydrolyze endogenous proteins, however, they also can break down exogenous proteins, for example, Coronavirus nucleocapsid protein [13]. An alternative double-step process has been suggested [14] and for this position, the virus tries to a receptor on the target host cells out layer via its S1 subdivision, and the S protein is sliced through proteases [15], the merging process occurs at acidic pH between viral and hosts target layer via S2 subdivide. Eventually, the viral hereditary products of a retiring abandoned RNA are completely delivered hooked on the cytoplasm. Here occurs the duplication with transcript techniques that are facilitated by the term replication/transcription complex (RTC). This complicated is programmed in the viral genome and is produced from (NSP). Such a complex is supposed to produce two folded assemblies in the cytoplasm of the contaminated cell [16]. Next, the progressive RNA genome is interpreted to produce replicas proteins from open interpretation structure 1a/b (ORF 1a/b) (see Figure 2). Previous proteins utilize the genome as a model to create whole-body negative-sense RNAs, which are hence considered as models in producing 2 S. Underlying viral proteins types including, M, S, and E are produced in the cytoplasm and situations insides of the endoplasmic reticulum (ER) (Figure 3), and transport to the endoplasmic reticulum-Golgi intermediate compartment (ERGIC) [17][18].

Furthermore, in the cytoplasm nucleocapsids are produced from the encapsulation of propagated genomes by N protein, and accordingly, they integrate inside the ERGIC layer to self-accumulate toward new virions. Lastly, these virions are sent out from injured cells by transmission to the cell membrane via vesicles and discharged through a process named exocytosis, which can contaminate different cells. Meanwhile, the pressure of viral production on the (ER), in the end, prompts cell death. Regardless, the mechanism of activity for novel COVID-19 is at this point unclear [17].

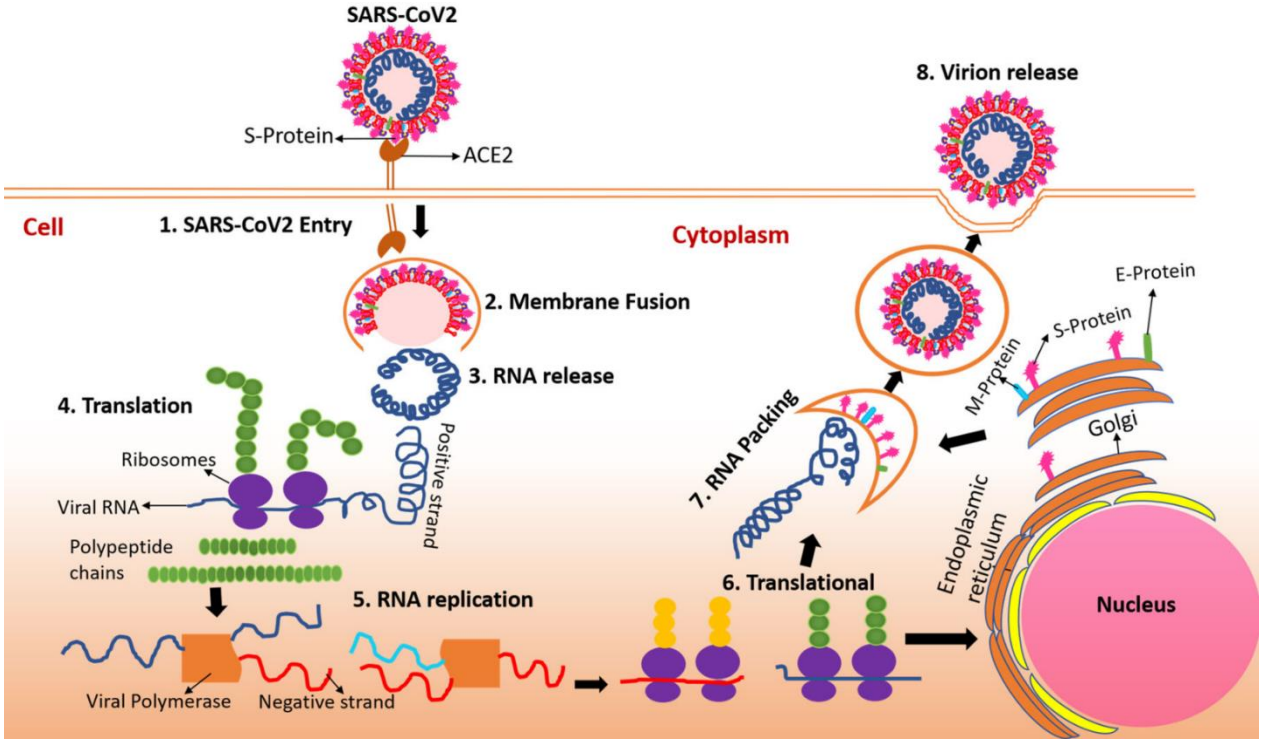

Figure 3. The schematic diagram of the mechanism of COVID-19 entry and viral replication and viral RNA packing in the human cell [19].

1.2.5 transition and symptoms of Covid-19

The change happens through respiratory fomites in an ill person and during the time of asymptomatic incubation time, which is assessed somewhere in the range of 2 and 10 days [19]. Most SARS-CoV-2-contaminated patients promote mild manifestations like dry cough, sore throat, and pyrexia, as a rule settling spontaneously [20]. However, more serious manifestations, for example, pneumonia and marked fever in addition to fatal complications as well as organ insufficiency, septic trauma, pulmonary edema, and intense respiratory distress condition [21].

1.2.6 COVID-19 Disease with hypercoagulability

SARS-CoV-2 is a new RNA covid and the contributing microorganism for the continuous covid illness 2019 pandemic. While SARS-CoV-2 disease is related to the progress of acute respiratory distress syndrome.The late record additionally defines people who have multi-organ insufficiency and thrombosis, as well as myocardial infarction (MI) and ischemic stroke.[22] People suffering from risk factors such as overweight, diabetes, and high blood pressure are more at risk for the improvement of thrombotic risk of 20 – 30 percent of analytically sick COVID-19 patients suffering from thrombosis throughout Covid infection[23-24]. Even though thrombotic obstacles are widespread during SARS-CoV-2 disease infection.

Although their traditional part is in blood clots and hemostasis, platelets act as a mediatory key to inflammatory and immune action[25]. Comparable to inherent immunological cells, platelets validate a wide collection of receptors, which include Toll-like receptors, C-type lectin receptors, and nucleotide-attach and oligomerization region–like receptors[26]. All different receptors are recognized to assistance identifying viral pathogens like HIV-1, and influenza [27-28]. When platelets are exposed to the attacking pathogens, they make immune reactions parenthetically via the liberation of cytokines with antimicrobial peptides and immediately by association with neutrophils, lymphocytes, as well as monocytes to enhance the immune reaction[29]. Due to platelet activation regularly happening when platelets react to attacking microorganisms, inflammatory and infectious diseases are regularly related to a prothrombotic reaction, named immune-thrombosis. This may motivate unfavorable immunological and hemostatic action, thus participating in disadvantageous clinical results like vascular thrombosis, structure insufficiency, and death. [30] However, regardless of whether platelets cause the pathophysiology of Covid 19 infection, which includes thrombosis, is unidentified.

In the forthcoming experimental study for (Manne et al.)[31]. the evaluation of platelet gene manifestation and active reactions in intensely sick patients with Covid-19, correlated with coordinated healthful contributors. RNA- sequencing (RNA-seq) examination from platelets remote from Covid 19 infected patients indicated variations in platelet gene expression, involving modifications in immune trails. However, platelet ultrastructure was unaffected, they were overexcited through SARS-CoV-2 disease, as demonstrated by improved surface P-selectin expression, motivation, and considerable production of flowing platelet-white blood cell combinations. Platelets from Covid-19 cases also show augmented accumulation, adhesion, and dispersing. These extreme reactive responses were ridden moderately by improved production and releasing of thromboxane A2. Obtained both, the data give the new indication that platelet gene expression is changed and efficient reactions are considerably augmented throughout SARS-CoV-2 disease. Here, assume that these alterations may participate in thrombotic episodes in Covid-19 patients.

1.2.7 The effect of COVID-19 on a pregnant and non-pregnant woman

At the point When Covid-19 and other CoV cause infection in pregnant women, it raises the danger of adversarial obstetrical and newborn impacts and caused serious respiratory disease [31]. The last pieces of information from different revisions of flu and other respiratory communicable illnesses have indicated an amplified danger of maternal obstetrical difficulties when we likened to nonpregnant according to physiological differences happening throughout the pregnancy period [32]. This relationship has furthermore been earlier illustrated to happen when pregnant women came to be infected by both pathogenic (SARS-CoV 2 or MERS-CoV) [33].

Pregnant women suffering from Covid -19 show clinical management more threat by persisting and obscuring the infection and cooperation with the therapy [34]. Investigators are staying in question concerning the conduction of the novel and last covid 19 infections from a pregnant to her fetus, a procedure called vertical transmission [35-36].

There are limited published events of Covid 19 infections happening throughout pregnancy and because of the probability of mother–fetal conventional transmission, there is a discussion that the fetus may be under the effect of complications of congenital Covid-19. Due to the sudden extent of Covid epidemics all through the world, extensive knowledge of the spread of the virus from pregnant women to fetuses in utero similar to other developing viral infections such as the Zika virus and Ebola virus [37-38], can risk the health and survival of infected pregnant women and fetus are significant for the effective administration of the infection and therapy.

Experience of COVID-19 infections throughout the pregnancy period.

Pregnancy intensifications the danger of challenging obstruction and newborn findings from several respiratory viral diseases. Many immunological and physical alterations that happen as a standard factor of pregnancy, have systemic outcomes that intensifier the chance of difficulties from respiratory infections. Differences in the protective respiratory and circulatory system, concerning rising cardiac pulses, stroke volume, O2 depletion, and reduced pulmonary capacity, also the development of immunologic modifications, which permit a mother to undergo an antigenically different fetus, upsurge the dangers for pregnant women to increase the severity of respiratory illness [39]. Consequences statistics from many studies of flu have confirmed a greater risk of maternal morbidity with mortality when described concerning nonpregnant women [40].

2.1. Study design

A meta-analysis and standardized examination were aimed to evaluate the consequence of Covid contagion (SARS-CoV-II, MERS-CoV, and SARS-CoV-I) in women and its influence on platelet, so we assess the study on non-pregnant women, pregnant ladies, and newborns of women was infected by the coronavirus.

3. 1. Clinical Analysis and Blood Parameters

The power of influence of the diseases was recognized, and predictable blood factors were observed, ranging from moderate to severe infection of Covid-19 (Table 1). Lymphocyte calculations, alanine aminotransferase, and aspartate aminotransferase statistics were comparable between groups of diseased patients. In comparison, the lactate dehydrogenase and C-reactive protein inflammation indicators, which were more sophisticated than the predictable standard variety in groups, were considerably amplified in patients with severe Covid-19 in evaluation with non-severe cases.

Table 1. Medical Investigation and Blood Factors [41]

Index No. (median IQR)	Patients with COVID-19		P-value
	Non-Severe	Severe	
female	32	18	
SARS-CoV-2 E gene (ct), detection, (n)	27 (31–24)	29.5 (34.25–24)	1.96×10−1
Age (n), y†	48 (62–37)†	64.5 (72.75–41.25)†	1.72×10−2†
Weight (n), kg	75 (87–62)	82 (90–65.5)	3.09×10−1
Duration of hospitalization (n), d†	14 (16–13)†	20 (28.75–14)†	1.22×10−4†
Platelet value at admission (n), ×106/mL†	192 (252–144)†	121 (179.5–80.75)†	6.91×10−5†
Lymphocyte value at admission (n), ×106/Ml	1.08 (1.49–0.64)	1.09 (1.8–0.525)	8.04×10−1
Platelet-Lymphocyte ratio at admission (n)†	168.4 (282.8–127.1)	121.7 (242.9–66.84)†	2.20×10−3†
ALT value at admission (n), U/L	35 (45.7–18.5)	35.35 (47.83–24.03)	3.32×10−1
AST value at admission (n), U/L	45 (55.4–26.5)	40.6 (56.43–24.63)	8.04×10−1
LDH value at admission (n), U/L†	506 (657–280)†	745.5 (946–468)†	8.0×10−4†
D-Dimers at admission (n), mg/L†	0.56 (1.04–0.37)†	0.845 (1.138–0.477)†	2.80×10−2†
CRP at admission (n), mg/L†	5.24 (9.67–2.14)†	23.26 (36.54–12.72)†	5.10×10−11†

† indicates P values smaller than 0.05

The calculated platelet statistics and platelet to lymphocyte proportions were calculated for the two teams. Platelet totals were in the minor values in both groups with Covid-19, in contrast to the predictable range in the healthy group. Furthermore, we detected an uncertain, nonetheless statistically important decrease in platelet totals between the two groups. The platelet to lymphocyte proportion was lesser in severe patients associated with non-severe patients.

Table 2 shows the research laboratory features containing the standard values measured by the Mega lab. WBC, Hb, platelets, and hepatic enzymes amounts were in the standard values in both teams. Lymphocytopenia was detected in the pregnant and control groups, correspondingly ($p = 0.44$). The relation of lymphocyte - WBC count was significantly determined in the pregnant group related to the controls. CRP levels were raised in both groups deprived of significant variance between them. Venous blood gas examination showed regular and similar PH levels between the groups. Nevertheless, pCO2 was pointedly lower, and disreputable excess was significantly higher among pregnant women compared to controls.

Table 2. Research laboratory exploration of groups between pregnant and non-pregnant[42]

	Normal range during the third trimester	**Pregnant (*n* = 11)**	**Normal range**	**Nonpregnant (*n* = 25)**	***p*-value**
Blood count					
White blood cell count (K/µL)	5.6–16.9	9.29 (6.80–12.00)	4.0–10.8	6.23 (4.64–7.58)	0.001
Neutrophil count (K/µL)	3.9–13.1	6.72 (4.43–9.21)	1.8–7.7	3.93 (2.80–5.34)	0.11
Neutrophil (%)	40–74	79.20 (72.00–91.55)	40–74	64.20 (55.77–75.15)	0.001
Lymphocyte count (K/µL)	1.0–3.6	1.13 (0.64–1.68)	1.0–4.8	1.49 (0.92–1.93)	0.11
Lymphocyte (%)	20–40	13.60 (4.50–19.37)	20–40	26.50 (15.70–29.90)	0.003
Monocytes (K/µL)	0.1–1.4	0.59 (0.42–0.66)	0–0.6	0.48 (0.38–0.60)	0.36
Monocytes (%)	4.7–12.5	6.1 (3.80–8.90)	4.7–12.5	7.60 (6.10–101.95)	0.03
Eosonophils (K/µL)	0–0.6	0.02 (0.001–0.05)	0–0.6	0.01 (0.005–0.05)	0.97
Eosonophils (%)	0–7	0.20 (0.001–0.50)	0–7.0	0.20 (0.10–1.00)	0.46
Hemoglobin (mg/dL)	9.5–15.0	12.06 (11.00–12.80)	11.7–15.7	12.55 (11.50–14.07)	0.29
Platelets (K/µL)	146–429	213 (130–223)	130–440	187 (152–239)	0.59
Chemistry					
Creatinine (mg/dL)	0.4–0.9	0.48 (0.40–0.52)	0.5–0.9	0.66 (0.55–0.83)	0.001
Urea (mg/dL)	15–45	13 (10–15)	15–45	24 (18–30)	0.001
AST (IU/L)	4–32	25 (21–37)	7–40	22 (19–37)	0.58
ALT (IU/L)	2–25	12 (8–16)	7–45	21 (14–30)	0.01

Table 3: Summary of the reported cases of coagulation in pregnancy.

		Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7	Case 8	Case 9	Case 10
Study numeral		48 (Canada)	48	67	67	50	118	NUH/UHL	155	101	81
Grouping of coagulopathy		DIC in pregnancy score 27	DIC in pregnancy score 27	Authors stated DIC	Authors stated coagulopathy	The authors state mild coagulopathy. DIC in pregnancy score 27	Authors stated coagulopathy	Authors stated DIC	Authors stated DIC	DIC in pregnancy score 27	Authors stated DIC
Maternal result		Recovered	Recovered	Died	Remains in Hospital	Remains on ICU	Died	Died	Recovered (after termination of pregnancy)	Remains in hospital	Recovered
Haematological indices	Platelets (minimum and maximum if multiple values reported)	82	54	122–188	122–170	114	40–119	57	33–94	required ~10 injections of platelets)	57
	APTT (normal range)	41 (18.5–29.9)	60 (28.0–41.9)			35.1	PTT 30.1–30.6	49.3 (24–33)	PTT 44.6–27.7	PTT 36	
	Prothrombin Time					20.2	10.6–10.9	23.9	12.7	16	
	INR	1.0	1.1			1.7	0.94–0.97	1.8			
	Fibrinogen (g/L) *Normal 2.48–5.06 (3rd trimester)*	2.2	0.8					1.1	Mg/dL < 60–275		
	D Dimer (mg/L) *normal 0.13–1.7*	25.79	> 20				6.5	19.06	> 33.89		
	Minimum ISTH Pregnancy DIC Score with available values	27	27			27		N/A (postpartum)	26	27	
	Minimum DIC score (ISTH)	4	5				2	6	6		

Platelets may achieve many roles in performance with neutrophils, RBC, and endothelium that participate in the occurrence of coagulation. At the onset of contagion, platelets may dynamically develop necrosis, which can come to be misregulated as the infection develops. Furthermore, platelets can react to injured endothelium, and there is an alteration in inflammatory cytokines, and antibodies, which all will participate in the intensification of their thrombotic tenacity. Performance thrombocytopenia and micro thrombosis are impossible to determine synchronously, possibly pointing to inflammatory cytokine indicating that necrosis throughout Covid-19 may be active. So, NETosis in CoV may be produced by epithelial and endothelial cells pretentious via the virus, through triggered platelets, and by inflammatory cytokines. Together, extreme NETosis is intricate in the expansion of the "cytokine storm" and thrombosis, which are the core pointers of the severe development of Covid-19[44]. Thrombocytopenia under 100x1000 /μL is observed equally a minimum standard for attending the hospital in the (ICD) by the American Thoracic Society (ATS) [45]. Focusing on the statement that platelet calculations are reduced in patients with difficulties complications. Consequently, they planned thrombocytopenia as a mild and commercial biomarker to observe the severity of the disease [46].

Agreeing to researchers, Covid-19 pregnant women are possibly showing an amplified threat of thrombosis because of inflammation, related to the virus infection, connected with the functional intensification in thrombin and prothrombin in pregnancy. With regard, the investigation focus that anticoagulation treatment and a hematological examination to distinguish the occurrence of circulated intravascular coagulation must be measured in experimental practice [47].

Method than supplying antiplatelet drugs. (SARS -CoV2) track into endothelium that precise ACE2 and the decrease of endothelium validity may maintain the staffing of flowing platelets to positions of infection, platelets stimulation, and degranulation, demonstrating that the reserve of endothelial cell triggering can stimulate disease consequence. Megakaryocytes in the pulmonary tolerate an immune formula separate from the megakaryocytes recognized in the bone marrow. Though this persists hypothetical, megakaryocytes in the pulmonary area, a superior position that potency stimulates cellular infection by respiratory viruses, may be valuable to covid-19 infection and may transference viral RNA or segments of the viral as pro-platelets are formed. Also, this virus will infect megakaryocytes, which affects megakaryocyte transmission. Therefore, platelets are activated by infected megakaryocytes and their accumulations in the inflamed atmosphere.

In table 2 the clinical demonstration of covid19 infection throughout the pregnancy period appears comparable to another group, as earlier described. However, respiratory irregularities are the maximum general offering signs in both groups, as contrasting to fever, which has been earlier defined as the most usually offering indication. Merely 27percent of the pregnant females in the present study had a fever.

Laboratory abnormalities, which describe covid19 infection have been recognized. Lymphocytopenia was described to be the maximum results presenting in 83.2 percent of 1099 diseased patients, tracked by thrombocytopenia in 36.2% of patients, and leukopenia in 33.7%. Moreover, 60% of the patients had raised CRP [48]. leukopenia remained in 24.1% of pregnant women, and higher CRP was recognized in 44% of pregnant ladies. The group with higher rates of infections had more visible laboratory irregularities connected with those with non-severe infections [49].

According to the earlier publication, the equal proportion of lymphocytopenia (44%) in the group of pregnant women, remained comparable to the level recognized in the other group. Nevertheless, similarly, the lymphocyte – WBC account was significantly diminished in the group of pregnant women related to the standard group. No additional laboratory alterations about CRP intensities, platelet amounts, and level of PH were measured between groups except minor grades of pCO2 in the group of pregnant women, maximum likely secondary to the similar hyperventilation. Pregnant women with covid19 disease took a minor acceptance degree compared to other groups. A lower percentage of hospitalization among pregnant ladies with viral infection has recently been described. Table 3 hemostatic and thromboembolic instabilities described of pregnant ladies with viral disease. The total harm of thromboembolic problems in pregnant women regardless of such viral infection is 0.1% [50].

Measures of the occurrence of DIC in pregnant ladies varied range from 0.03 to 0.35% [51]. The previous results propose that the threat of hemostatic and thromboembolic difficulties is greater in pregnant ladies with viral infection related to pregnant women without SARS - CoV2 disease.

4.1. Conclusion:

Platelets collected from people suffering from (SARS -CoV 2) were energetic. They were alerted to discharge inflammatory elements, gathered, and adhered to collagen outward further proficiently when arising from patients with such viruses. Equally exceedingly, overactive platelets might participate in the disease mechanism by the release of inflammatory intermediaries and blood clots. In assuming, this review has shown that platelets (man and woman) are hyper-responsive in SARS -CoV 2 and may participate in the sign of systemic inflammatory consequence and thrombotic measures recognized in this infection. The barrier of platelet response lanes may augment the consequences of this disease. Also, this review concludes that laboratory features of viral infection don't diverge between two groups related to the pregnancy, although a tendency for condensed lymphocyte number was detected in the group of pregnant women. These data must be important to the health care team and patients too. Further analysis is required to approve the outcomes.

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