THE CONDITION OF THE HEMOSTASIS SYSTEM IN PREGNANT WOMEN WITH ACUTE RESPIRATORY VIRAL INFECTIONS: A BIOCHEMICAL BASIS

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Annotatsiya: O‘tkir respirator virusli infektsiyalar barcha yuqumli kasalliklarning ko‘p qismini tashkil qiladi va keng tarqalgan. JSST ma'lumotlariga ko‘ra, O‘RVI dunyo bo‘ylab barcha holatlarning taxminan 42 foizini tashkil qiladi, ularning 16 foizi homilador ayollarda uchraydi.Homiladorlik va post tromboembolik muammolar o‘tkir nafas yo‘llarining buzilishi bilan bog‘liq. Biz nazorat guruhidagi 40 nafar homilador va o‘tkir respirator kasallik bilan og‘rigan 35 nafar homilador ayollarda gemostaz tizimining holatini tekshirdik. MNO, D-dimer, APTT, trombotsitlar soni, agregatsiya funksiyasi va fibrinogen o‘lchangan parametrlar edi. Sinovlarning chastotasi bemor ahvolining og‘irligiga bog‘liq.

Kalit so‘zlar: o‘tkir respirator virusli infeksiya, gemostaz, agregatsiya, fibrinogen, biokimyoviy asos.

Аннотация: Острые респираторные вирусные инфекции содержат большую часть всех инфекционных патологий и широко распространены. По данным ВОЗ, эпидемия ОРВИ во всем мире вызывает около 42%, из них16% встречается у беременных женщин. Беременность и посттромбоэмболические осложнения связаны с острыми респираторными заболеваниями. Мы исследовали состояние системы гемостаза у 40 беременных из контрольной группы и 35 беременных с ОРВИ. Были измерены МНО, D-димер, АЧТВ, количество тромбоцитов, агрегационная функция и уровень фибриногена. Частота проведения анализов зависела от тяжести состояния пациента.

Ключевые слова: острые респираторные вирусная инфекция, гемостаз, агрегация, фибриноген, биохимические основы.

Annotation: Most infectious diseases are caused by acute respiratory virus infections, which are common. The WHO estimates that influenza outbreaks cause about 42% of cases worldwide, with pregnant women accounting for 16% of these instances. Acute respiratory diseases are linked to pregnancy and post-thromboembolic issues. We looked at the state of the hemostasis system in 35 pregnant women with ARVI and 40 pregnant women from the control group. INR, D-dimer, APTT, platelet count and aggregation function, and fibrinogen were the

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parameters that were measured. The frequency of the procedure was decided by the severity of the patient's condition.

Key words: acute respiratory viral infections, hemostasis, aggregation, fibrinogen, biochemical basis.

INTRODUCTION.

The physiological changes that naturally take place during pregnancy result in a hypercoagulable state. The first indication of this is an increase in coagulation factors (factors VII, VIII, and X; von Willebrand factor (vWF); D-dimer; C-reactive protein; and fibrinogen). Concurrently, the number of inhibitors of the fibrinolytic system increases. Protein C levels are down, resistance to activated protein C increases, and prothrombin time (PT) and/or activated partial thromboplastin time (APTT) are also somewhat prolonged, making these factors ineffectual at inhibiting coagulation, especially in the second and third trimesters.. Anatomical changes in the pregnant woman's body also have a big influence since the pregnant uterus compresses the pelvic veins, decreasing circulation in the lower extremities. The stasis this produces may lead to the formation of a thrombus, or blood clot [1, 3, 6]. Therefore, it is strongly recommended that all hospitalized ARD patients have their fibrinogen, D-dimer, PT and/or APTT levels, and platelet count checked.

Endothelial cell invasion brought on by ARD damages endothelial cells and obstructs fibrinolytic activity, which promotes the formation of thrombus and a significant release of vWF. The hypercoagulable state is made worse by the suppression of the clot lysis mechanism caused by a loss of protective endothelium function. Another consequence of ARD that slows circulation and increases blood viscosity is increased intravascular fibrin deposition. The hypothesis that ARD raises the risk of thrombus development and thromboembolism is supported by all of these results [4, 7]. ARD has been associated with neutrophilia, leukocytosis, prolonged PT, elevated D-dimers, and increased interleukins, IL-6 and IL-8. The neutrophil-to-lymphocyte ratio (NLR) (low risk NLR <3.13, high risk ≥3.13) predicts the course of critical illness [2, 6].

The development of hypercoagulation without symptoms of disseminated intravascular coagulation (DIC) is a characteristic of early-stage ARD coagulopathy. D-dimer increases, fibrinogen (sometimes called "acute-phase protein") increases, PT slightly lengthens, and platelet counts slightly decrease (70–95% of patients have a platelet count $<150*10^1$/l). The concentration of antithrombin III decreases to 80% concurrently with the rather constant concentration of protein C [5, 8].

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Go for it. to assess how acute respiratory conditions affect the metabolic foundation of the hemostasis system in expectant mothers.

A REVIEW OF LITERATURE. Respiratory viral infections include influenza and acute respiratory viral infections. The general structure of acute respiratory viral infections contains the most significant influenza symptoms. In contrast to acute respiratory viral infections, influenza can cause epidemics, is more severe, frequently causes, and can result in death. [137] Influenza and acute respiratory viral infections (ARVI) in pregnant women are currently the subject of extensive investigation. On the one hand, the widespread occurrence of these illnesses is linked to increased interest in this issue; on the other hand, the frequent emergence of novel influenza virus strains, which dictates the unique course of the illness in pregnant women, necessitates the development of new and improved treatment and prevention strategies [10, 58]. It is known that pregnant women are characterized by more severe forms of infectious diseases caused by a decrease in the body's immune resistance during gestation [61, 102]. However, the features of acute respiratory viral infections and influenza in the presence of concomitant diseases, taking into account the peculiarities of the medical and social status of pregnant women, have not been sufficiently studied and require further analysis. Severe acute respiratory viral infections, as well as those complicated by bacterial infections, are considered significant factors in the development of disorders of pregnancy, childbirth, and fetal pathology [13, 81]. In this regard, the development of reliable methods for predicting the development of severe forms of influenza and its complications can be of great practical importance for timely preventive measures.

Along with predicting possible complications of influenza, an equally important task is the need to improve the differential diagnosis of influenza and its complications in the form of viral and bacterial pneumonia [82, 83]

Cytokines are crucial in the establishment of the systemic inflammatory response syndrome that typically follows the emergence of problems in severe types of influenza. Changes in cytokine levels are indicative of CVD itself, but they are not a reliable way to distinguish between the disease's underlying types, according to findings from the literature [94, 118]. Respiratory tract illnesses are caused by viruses. The most common way that viruses spread is through self-inoculation to the nasal mucosa or conjunctiva from hands that have come into touch with a patient (for instance, during a handshake) or from surfaces that have been contaminated by the virus (rhinovirus can remain on them for up to one day). Another method is through airborne droplets, such as ingestion of bigger droplets on the patient's mucosal membranes or inhalation of virus-containing aerosol

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particles. Most viral illnesses take two to seven days to incubate. Low-intensity virus release can continue for up to two weeks following infection, however the release of viruses to patients for a maximum of three days after infection significantly declines by the fifth day. Catarrhal inflammation is a hallmark of viral infections. The innate immune system's response, rather than the virus's harmful effects, is what causes the symptoms of acute respiratory viral infections. The number of cytokines secreted by affected epithelial cells, such as interleukin (IL) 8, is correlated with the severity of symptoms as well as the extent of phagocyte involvement in the submucosa and epithelium. It is unreasonable to interpret a change in the color of nasal mucus as an indication of a bacterial infection because an increase in nasal secretion is linked to an increase in vascular permeability, and the number of white blood cells in it can increase multiple times, changing its color from transparent to white-yellow or greenish [1]. Practice does not support the theory that any viral infection stimulates the bacterial flora (the so-called viral-bacterial etiology of ARI) based, for instance, on a patient's leukocytosis.Bacterial complications of acute respiratory viral infections are relatively rare.

Acute respiratory viral infections are the most common human infection: children under the age of 5 suffer an average of 6-8 episodes of acute respiratory viral infections per year [2, 3], in preschool institutions the incidence is especially high in the 1-2 year of attendance — 10-15% higher than in unorganized children, but at school the latter get sick more often [4]. The incidence of acute upper respiratory tract infections can vary significantly from year to year. The incidence is highest in the period from September to April, with the peak incidence occurring in February-March. The decline in the incidence of acute upper respiratory tract infections is invariably recorded in the summer months, when it decreases by 3-5 times [5]

Various viruses can be the cause of acute respiratory viral infections in humans: orthomyxoviruses, paramyxoviruses, coronaviruses, picornaviruses, adenoviruses and herpesviruses. In childhood, influenza, parainfluenza, adenovirus, and respiratory syncytial (PC) viral infection are the most severe. . Moreover, in some years, the PC virus causes 31-40% of all acute respiratory infections, mainly affecting young children [126, 137].

Despite the fact that respiratory tract diseases have been known for a long time, there are many unresolved problems in terminology, deciphering the etiology and pathogenesis of certain forms of diseases, especially when two or more pathogens are identified simultaneously.

Although it is now widely acknowledged that multiple pathogens may be involved in the etiology of diseases at the same time, the data on the percentage of

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respiratory mixed infections varies too much (from 0 to 100%), depending on a variety of factors, chief among them the resolution of the examination techniques, equipment, number, and characteristics of the diagnostic tools used. However, a "mixed infection" may potentially be overdiagnosed due to anamnestic, serological responses, and other factors that skew the results of the study [60].One of the variants of mixed infections is a disease that occurs as a result of the superimposition of a second infection onto the first one after a certain time interval (superinfection), which is possible both at home and, more often, in children's medical and health facilities [60].

MATERIAL AND METHODS.

The study was conducted in the Specialized Maternity Center for Pregnant Women with ARVI in the Samarkand region, in addition to the Maternity Complex No. 1 laboratory in Samarkand city.

We examined 75 women in the 20–35 age range. To finish the tasks, the patients were divided into two groups:

The main group (I), which consisted of 35 pregnant women who had recovered from ARVI, was divided into two subgroups:

1a: ARD in the second trimester of pregnancy in 15 pregnant women

1b : 20 expectant mothers who experienced ARD throughout the third trimester of their pregnancy;

II: 40 expectant mothers in generally good health.

The following blood coagulation system parameters were analyzed as part of the research: D-dimer, RCMF, prothrombin time, antithrombin III activity, platelet count, platelet aggregation, APTT, INR, fibrinogen concentration, and Lee-White clotting time.

RESULTS AND DISCUSSIONS.

Based on the aforementioned, we assessed the hemostasis system in pregnant women with ARVI in different trimesters. Pregnancy-related hemostasis system status in women with moderate ARVI.

Table 1.

Condition of the hemostasis system in mildly ill pregnant women (M ± m)

Parameters	**Control group (n = 40)**	**Pregnant women with mild ARD (n = 15)**	**P**
Lee-White clotting time (min)	7.2 ± 0.3	7.8 ± 0.4	>0.2
INR (International Normalized Ratio)	0.90 ± 0.04	0.95 ± 0.02	>0.2
Fibrinogen concentration (g/L)	4.6 ± 0.2	4.9 ± 0.2	>0.2

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APTT (Activated Partial Thromboplastin Time, sec)	17.1 ± 0.6	24.9 ± 0.9	<0.001
Platelet count (1x10^9 /L)	221.7 ± 3.4	180.5 ± 6.5	<0.001
Platelet aggregation (stimulation with ADP 1x10^-3 M Tma %)	40.2 ± 1.4	42.1 ± 1.5	>0.5
D-dimer (ng/mL)	421.3 ± 5.7	571.9 ± 20.4	<0.001
RKMF	Negative	Negative	-
Prothrombin time	10.3 ± 0.4	14.8 ± 0.5	<0.001
Antithrombin III activity (%)	89.6 ± 3.1	93.0 ± 3.3	>0.5

Note: P - significance of differences between the compared indicators and the group of pregnant women without ARD (Acute Respiratory Disease).

Table 2.

Comparative characteristics of the coagulogram in healthy patients and pregnant women with mild ARVI

Parameters	**Healthy pregnant women (n = 40)**	**Pregnant women with mild ARD (n = 15)**	**χ^2**	**P**	**OR**	**Min CI**	**Max CI**
Clotting time (min)							
≤ 6 minutes	17	4	1.090	0.296	0.52	0.15	1.80
≥ 6 minutes	33	15	1.090	0.296	1.93	0.55	6.73
INR ≤ 1	27	9	0.243	0.622	0.77	0.27	2.21
INR ≥ 1	23	10	0.243	0.622	1.30	0.45	3.76
Fibrinogen concentration (g/L)							
≤ 5	32	9	1.579	0.209	0.51	0.17	1.48
≥ 5	18	10	1.579	0.209	1.98	0.68	5.76
APTT (sec)							
≤ 20 sec	38	1	28.034	0.0001	0.02	0.00	0.15
≥ 20 sec	12	18	28.034	0.0001	57.00	6.87	472.84
Platelet count (1x10^9/L)							
< 200	14	14	11.917	0.001	7.20	2.18	23.74

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≥ 200	36	5	11.917	0.001	0.14	0.04	0.46
Platelet aggregation (%)							
≤ 30	11	0	4.973	0.026	0.00	-	-
≥ 30	39	19	4.973	0.026	-	-	-
D-dimer							
≤ 450	35	0	26.991	0.0001	0.00	-	-
≥ 450	15	19	26.991	0.0001	-	-	-
Prothrombin time							
< 9	19	0	9.964	0.002	0.00	-	-
≥ 9	31	19	9.964	0.002	-	-	-

The average hemostasis indicators in pregnant women with moderate types of ARD did not differ substantially from the control group, as shown in Tables 1 and 2. In contrast to pregnant women in the control group, some indicators, including APTT (activated partial thromboplastin time), platelet count, D-dimer, and prothrombin time, demonstrated notable variations in the direction of an increase. Pregnant women who have mild ARD are therefore at risk for increased thrombosis, which calls for proper treatment. Although the readings stayed within reference limits, pregnant women with mild ARD should get special attention due to the considerable drop in platelet levels.

Table 3.

State of the hemostasis system in pregnant women with moderate ARVI (M ± m)

Parameters	**Control group (n = 40)**	**Pregnant women with moderate ARD(n = 15)**	**P**
Clotting time (min)	7.2 ± 0.3	6.0 ± 0.2	<0.01
INR (International Normalized Ratio)	0.90 ± 0.04	1.64 ± 0.06	<0.001
Fibrinogen concentration (g/L)	4.6 ± 0.2	4.3 ± 0.2	>0.5
APTT (sec)	17.1 ± 0.5	19.1 ± 0.7	<0.01
Platelet count (1x10^9 /L)	220.6 ± 5.8	152.6 ± 6.1	<0.001

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Platelet aggregation (stimulation with ADP 1x10^-3 M Tma %)	40.2 ± 1.4	38.0 ± 1.4	>0.2
D-dimer	423.2 ± 11.1	990.9 ± 16.8	<0.001
RKMF	Negative	Positive (+++)	-
Prothrombin time	10.3 ± 0.4	6.6 ± 0.2	<0.001

Table 4.

Comparative characteristics of the coagulogram in healthy patients and pregnant women with moderate ARVI

Parameters	**Healthy pregnant women (n = 40)**	**Pregnant women with moderate ARD (n = 15)**	**χ^2**	**P**	**OR**	**Min CI**	**Max CI**
Clotting time (min)							
≤ 6 minutes	17	28	1.615	0.204	1.65	0.76	3.56
≥ 6 minutes	33	33	1.615	0.204	0.61	0.28	1.31
INR ≤ 1	27	4	30.726	0.000	0.06	0.02	0.19
INR ≥ 1	23	57	30.726	0.000	16.73	5.26	53.17
Fibrinogen concentration (g/L)							
≤ 5	32	40	0.030	0.863	1.07	0.49	2.34
≥ 5	18	21	0.030	0.863	0.93	0.43	2.04
APTT (sec)							
≤ 20 sec	38	33	5.718	0.017	0.37	0.16	0.85
≥ 20 sec	12	28	5.718	0.017	2.69	1.18	6.11
Platelet count (1x10^9/L)							
< 200	14	49	30.655	0.000	10.50	4.34	25.39
≥ 200	36	12	30.655	0.000	0.10	0.04	0.23
Platelet aggregation (%)							
≤ 30	11	18	0.803	0.370	1.48	0.62	3.53

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≥ 30	39	43	0.803	0.370	0.67	0.28	1.60
D-dimer							
≤ 450	35	0	62.364	0.000	0.00	-	-
≥ 450	15	61	62.364	0.000	-	-	-
Prothrombin time							
< 9	19	52	26.609	0.000	9.43	3.80	23.40
≥ 9	31	9	26.609	0.000	0.11	0.04	0.26

The group of patients with mild ARD showed a significant increase in APTT, which indicates a low blood coagulation potential and raises the risk of bleeding during or after delivery. An active thrombotic process is suggested by the simultaneous large increases in D-dimer and INR. At the same time, there was a significant decrease in clotting time, which could be a sign of a hypercoagulatory stage of DIC disease. Due to the development of thrombophilia and a sharp deterioration in hemostasis, the blood of pregnant women with moderate ARD revealed a significant amount of soluble fibrin monomer complexes, indicating a high risk of thrombus formation.

Table 5.

Key parameters of the hemostasis system in pregnant women with severe ARD (M ± m)

Parameters	**Control group (n = 40)**	**Pregnant women with severe ARD (n = 5)**	**P**
Clotting time (sec)	7.2 ± 0.3	5.0 ± 0.2	<0.001
INR (International Normalized Ratio)	0.90 ± 0.04	2.02 ± 0.05	<0.001
Fibrinogen concentration (g/L)	4.6 ± 0.2	5.9 ± 0.3	<0.001
APTT (sec)	17.1 ± 0.5	22.1 ± 0.7	<0.001
Platelet count (1x10^9 /L)	220.6 ± 5.8	152.7 ± 4.8	<0.001
Platelet aggregation (stimulation with ADP 1x10^-3 M Tma %)	40.2 ± 1.4	33.9 ± 0.9	<0.001
D-dimer	423.2 ± 11.1	1347.2 ± 42.2	<0.001
RKMF	Negative	Positive (+++)	-
Prothrombin time	10.3 ± 0.4		

Note: R – reliability of the differences in the indicators compared to the control group;

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Hemostasis System Status in Pregnant Women with Severe ARD

The hemostasis system was studied in 25 pregnant women with severe ARD (Tables 5 and 6).

Table 6.

Comparative characteristics of coagulation parameters in healthy pregnant women and those with severe ARD.

Indicators	Healthy Pregnant Women n=40 (abs.)	Pregnant Women with Severe ARD n=5 (abs.)	χ2	P	OR	95% CI min	95% CI max
Clotting Time (min)							
Less than 6 minutes	17	19	11.779	0.001	6.15	2.07	18.26
6 minutes or more	33	6	11.779	0.001	0.16	0.05	0.48
INR (International Normalized Ratio)							
Less than 1	27	0	21.094	0.000	0.00	-	-
1 or more	23	25	21.094	0.000	-	-	-
Fibrinogen Concentration (g/l)							
Less than 5	32	9	5.273	0.022	0.32	0.12	0.86
5 or more	18	16	5.273	0.022	3.16	1.16	8.59
APTT (sec)							
Less than 20	38	9	11.398	0.001	0.18	0.06	0.50
20 seconds or more	12	16	11.398	0.001	5.63	1.98	15.98
Platelet Count (1x109/l)							
Less than 200	14	25	34.615	0.000	-	-	-
200 or more	36	0	34.615	0.000	0.00	-	-

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Platelet Aggregation (%)							
Less than 30	11	6	0.038	0.845	1.12	0.36	3.49
30 or more	39	19	0.038	0.845	0.89	0.29	2.78
D-dimer							
Less than 450	35	0	32.813	0.000	0.00	-	-
450 or more	15	25	32.813	0.000	-	-	-
Prothrombin Time							
Less than 9	19	25	26.420	0.000	-	-	-
9 or more	31	0	26.420	0.000	0.00	-	-

All hemostasis markers were significantly worse in pregnant women with severe ARD compared to the control group and two other comparison groups. These results unmistakably demonstrate thrombophilia given the clinical history of the illness, which includes severe pneumonia and ARDS. All of the patients in this group were given general thrombosis prevention methods in an emergency. After delivery, direct anticoagulants, or low molecular weight heparins, were administered.

Table 7.

Correlation matrix of hemostasis parameters with the severity of ARD and trimester of disease onset

r (P<0.05)	**Severity of ARD**	**Trimester of Disease Onset**
Clotting Time (min)	-0.68	-0.62
INR (International Normalized Ratio)	0.75	0.72
Fibrinogen Concentration (g/l)	0.58	0.62
APTT (sec)	-0.72	-0.74
Platelet Count (1x109/l)	-0.58	-0.59
Platelet Aggregation	-0.71	-0.62
D-dimer	0.86	0.80
Prothrombin Time	-0.71	-0.75

CONCLUSION.

According to the information above, hypercoagulation was noticeable in the early stages of the disease, although DIC syndrome symptoms were not present in coagulation abnormalities associated with the viral invasion. The lack of

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microangiopathy and the inability to discuss DIC syndrome at this time are shown by the fact that antithrombin III activity did not decrease. This was demonstrated by a sharp increase in D-dimer levels, a moderate decrease in platelet count, a prolonged prothrombin time, and an increase in fibrinogen levels (response to acute phase inflammation).However, hypercoagulation intensified and intravascular coagulation signs appeared as the condition deteriorated. Analysis of hemostasis indicators, the trimester of disease start, and the severity of ARVI revealed significant correlations. In particular, clotting time, platelet count, and the severity of ARVI as well as the trimester of disease onset were revealed to be moderately negatively correlated (Table 6). A shift in the hemostasis system toward increased coagulation was linked to the severity of the illness and was suggestive of endothelial dysfunction.

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