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«INTERNATIONAL CONFERENCE ON SUSTAINABLE DEVELOPMENT, ARTIFICIAL INTELLIGENCE AND GLOBAL INNOVATION (SDAIGI–2026)»

London, Tashkent
July 13, 2026

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International Conference on Sustainable Development, Artificial Intelligence and Global Innovation (SDAIGI–2026) // materials international scientific-practical conference. London, United Kingdom. July 13, 2026.

Editorial board of the conference

This event aims to discuss contemporary research across various scientific fields, exchange new scientific ideas, and support scientific activities aligned with global innovative processes.

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CLINICAL AND LABORATORY SIGNIFICANCE OF ENDOTHELIAL DYSFUNCTION AND HEMOSTATIC ALTERATIONS IN THE EARLY STAGES OF RHEUMATOID ARTHRITIS

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***Аннотация**: Цель исследования — оценить эндотелиальную дисфункцию и изменения гемостаза у пациентов с очень ранним и ранним ревматоидным артритом. Обследованы 85 пациентов и 30 практически здоровых лиц. Определяли D-димер, антитромбин III, фактор фон Виллебранда, эндотелин-1 и sICAM-1. По мере увеличения длительности заболевания отмечались рост D-димера, фактора фон Виллебранда, эндотелина-1 и sICAM-1, а также снижение активности антитромбина III. Результаты свидетельствуют о раннем формировании эндотелиальной дисфункции и протромботического лабораторного профиля при ревматоидном артрите.*

***Ключевые слова:** ревматоидный артрит, эндотелиальная дисфункция, гемостаз, D-димер, эндотелин-1, sICAM-1, фактор фон Виллебранда.*

***Abstract**: Objective: to evaluate endothelial dysfunction and hemostatic alterations in very early and early rheumatoid arthritis. The study included 85 patients and 30 healthy controls. D-dimer, antithrombin III, von Willebrand factor, endothelin-1 and sICAM-1 were assessed. D-dimer increased from 210±60 ng/ml in controls to 420±110 and 680±160 ng/ml, while antithrombin III decreased from 102±6% to 94±7% and 88±8%. Endothelial markers also increased with disease duration. These findings indicate early endothelial dysfunction and a progressively unfavorable prothrombotic laboratory profile in rheumatoid arthritis.*

***Keywords:** rheumatoid arthritis, endothelial dysfunction, hemostasis, D-dimer, endothelin-1, sICAM-1, von Willebrand factor.*

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic systemic autoimmune disease characterized by persistent synovial inflammation, progressive joint damage and extra-articular manifestations. Increasing evidence indicates that RA is also associated with vascular dysfunction and abnormalities of hemostasis, contributing to excess cardiovascular and thrombotic burden (Aletaha et al., 2010; Smolen et al., 2023).

Endothelial dysfunction is an important component of systemic inflammation in RA. Chronic inflammatory signaling promotes endothelial activation, expression of adhesion molecules, platelet activation and a shift toward a prothrombotic state (Yang et al., 2016; Anyfanti et al., 2020).

Endothelin-1, soluble intercellular adhesion molecule-1 (sICAM-1) and von Willebrand factor (vWF) are markers of endothelial activation and injury. D-dimer and antithrombin III characterize coagulation activation and natural anticoagulant protection.

The objective of this study was to evaluate endothelial dysfunction and hemostatic parameters in patients with very early and early RA and to determine whether laboratory abnormalities become more pronounced with disease duration.

MATERIALS AND METHODS

This clinical and laboratory study was conducted at the multidisciplinary clinic of Tashkent State Medical University during 2022–2025. The study included 85 patients diagnosed with RA and 30 practically healthy individuals who formed the control group.

The diagnosis of RA was established according to accepted classification criteria. Inclusion criteria were age over 18 years, confirmed RA, moderate disease activity according to DAS28 and written informed consent. Exclusion criteria included severe somatic disease, acute infection, uncontrolled arterial

1. Endothelial dysfunction and hemostatic abnormalities are detectable in the early stages of rheumatoid arthritis.

2. D-dimer progressively increased from 210±60 ng/ml in controls to 420±110 ng/ml in very early RA and 680±160 ng/ml in early RA.

3. Antithrombin III activity decreased from 102±6% in controls to 94±7% and 88±8% in the respective patient groups.

4. vWF, endothelin-1 and sICAM-1 increased with disease duration, indicating progressive endothelial injury and inflammatory endothelial activation.

5. Combined assessment of these markers may help characterize an unfavorable vascular and prothrombotic laboratory profile; prospective validation is required before use as predictive markers of clinical thrombosis.

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Parameter	Control group, n=30	Very early RA, n=32	p1	Early RA, n=53	p2
D-dimer, ng/ml	210±60	420±110	<0.001	680±160	<0.001
Antithrombin III, %	102±6	94±7	<0.05	88±8	<0.05
Von Willebrand factor, %	98±9	126±14	<0.001	152±18	<0.001
Endothelin-1, pg/ml	0.86±0.21	1.42±0.33	<0.001	1.87±0.41	<0.001
sICAM-1, ng/ml	210±35	325±50	<0.001	412±70	<0.001

Note: RA, rheumatoid arthritis; sICAM-1, soluble intercellular adhesion molecule-1; p1, comparison between very early RA and control group; p2, comparison between early RA and control group. Data are presented as M±m.

DISCUSSION

The results demonstrate that endothelial dysfunction and hemostatic abnormalities are present from the early stages of RA. The increase in D-dimer is consistent with enhanced fibrin turnover and a procoagulant laboratory profile, whereas the progressive decrease in antithrombin III suggests weakening of natural anticoagulant protection.

The increase in vWF indicates endothelial injury and enhanced platelet adhesion potential (Vischer, 2006; Manz et al., 2022). Elevated endothelin-1 reflects vasoconstrictor imbalance, whereas increased sICAM-1 indicates inflammatory endothelial activation and leukocyte-endothelial interaction (Yang et al., 2016; Anyfanti et al., 2020).

Taken together, the simultaneous increase in D-dimer and endothelial markers together with the decrease in antithrombin III suggests that endothelial injury, coagulation activation and impairment of anticoagulant mechanisms are interconnected components of early RA. This interpretation is consistent with epidemiological evidence of increased venous thromboembolism risk in RA (Molander et al., 2021; Hu et al., 2021).

The principal limitation of the present study is its cross-sectional laboratory design. The findings demonstrate group differences but do not establish causality or predictive accuracy for future thrombosis. Prospective studies are required to validate the combined marker panel.

CONCLUSIONS

hypertension, current use of antiplatelet agents, anticoagulants or statins, and refusal to participate.

Patients with RA were divided into two groups according to disease duration: very early RA (n=32) and early RA (n=53). D-dimer, antithrombin III, vWF, endothelin-1 and sICAM-1 were evaluated.

Quantitative data are presented as M±m. Differences were considered statistically significant at $p<0.05$. In Table 1, p1 denotes comparison between very early RA and controls, whereas p2 denotes comparison between early RA and controls. No unsupported p3 values were introduced because the available material did not contain a calculated direct comparison between the two RA groups.

The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants.

RESULTS

D-dimer concentration was 210±60 ng/ml in the control group, 420±110 ng/ml in very early RA ($p1<0.001$), and 680±160 ng/ml in early RA ($p2<0.001$).

Antithrombin III activity decreased from 102±6% in controls to 94±7% in very early RA ($p1<0.05$) and 88±8% in early RA ($p2<0.05$).

vWF increased from 98±9% in controls to 126±14% and 152±18% in very early and early RA, respectively. Endothelin-1 increased from 0.86±0.21 pg/ml to 1.42±0.33 and 1.87±0.41 pg/ml. sICAM-1 increased from 210±35 ng/ml to 325±50 and 412±70 ng/ml. All endothelial marker differences versus controls were statistically significant at $p<0.001$.

The direction of the changes was consistent across the analyzed markers: coagulation activation and endothelial injury were detectable in very early RA and became more pronounced in early RA.

Table 1. Endothelial dysfunction and hemostatic parameters in patients with early stages of rheumatoid arthritis