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ERYTHROCYTE DEFORMABILITY AND ITS ROLE IN MICROCIRCULATION

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Abstract

This article examines erythrocyte deformability—the ability of red blood cells to change their shape—and its fundamental importance in the process of microcirculation. The study analyzes molecular factors affecting erythrocyte flexibility, particularly the role of membrane proteins and intracellular viscosity. Using ektacytometry and microfiltration methods, blood samples from healthy individuals and patients with various pathologies (such as diabetes mellitus and arterial hypertension) were comparatively studied. The results indicate that reduced erythrocyte deformability increases blood viscosity, leading to tissue hypoxia and microvascular complications. The article concludes with findings on the clinical significance of improving erythrocyte rheological properties.

Keywords: erythrocyte deformability, microcirculation, hemorheology, capillary flow, ektacytometry, viscosity, membrane elasticity, hypoxia

Introduction

One of the most pressing issues in modern fundamental medicine and clinical hemorheology is the comprehensive study of blood rheological properties, particularly the functional and mechanical state of erythrocytes. Erythrocyte deformability refers to the ability of red blood cells to dynamically change their morphological shape under external hydrodynamic and mechanical forces, pass through narrow capillary pathways, and subsequently return to their original discocyte (biconcave disc) shape [1]. This property depends not only on cell morphology but also directly on membrane structure, cytoplasmic viscosity, and the surface-to-volume ratio of the cell.

Microcirculation in the human body occurs under unique hydrodynamic conditions. While the average diameter of a normal erythrocyte is about 7.2–8.5 μm, the smallest capillaries in peripheral tissues measure only 3–5 μm in diameter [2]. Under such conditions, a high degree of erythrocyte flexibility is essential to maintain continuous blood flow. This deformation allows erythrocytes to approach capillary walls closely, thereby reducing diffusion distance and enhancing the efficiency of gas exchange (O_2 and CO_2) [3].

However, under various pathological conditions, a decrease in membrane elasticity or an increase in erythrocyte rigidity is observed. From a hemorheological perspective, impaired deformability increases overall blood viscosity and leads to "sludge" phenomena (erythrocyte aggregation) within the microcirculatory system [4]. Consequently, this results in impaired tissue perfusion, hypoxia, and the development of metabolic acidosis.

Recent studies confirm that reduced erythrocyte deformability plays a critical role in the pathogenesis of microvascular complications of diabetes mellitus, arterial hypertension, acute myocardial infarction, ischemic stroke, and even severe viral infections [5]. Therefore, studying erythrocyte deformability is not only essential for understanding cellular-level mechanical processes but also holds strategic importance in early diagnosis and correction of many systemic diseases.

Literature Review

Scientific research on erythrocyte deformability demonstrates that this process is a complex combination of molecular and mechanical factors. Researchers emphasize that erythrocyte flexibility depends on three main components: membrane elasticity, intracellular fluid (hemoglobin) viscosity, and the surface-to-volume ratio [6]. Even minor changes in any of these parameters can significantly slow microcirculatory flow.

Special attention in the literature is given to cytoskeletal membrane proteins such as spectrin and ankyrin complexes. These proteins enable the cell to dynamically change its shape and recover after mechanical stress [7]. Modern studies show that oxidative stress can disrupt the structure of these proteins (through lipid peroxidation), resulting in an increased number of rigid erythrocytes, which may cause capillary blockage [8].

Clinical studies indicate that decreased erythrocyte deformability is an early marker of many systemic diseases. For instance, in patients with diabetes mellitus, increased levels of glycated hemoglobin raise intracellular viscosity and reduce membrane elasticity [9]. Similarly, in arterial hypertension, elevated vascular pressure and hemorheological changes enhance erythrocyte aggregation, further impairing microvascular perfusion [10].

Recent meta-analyses consider erythrocyte deformability not only as a diagnostic indicator but also as a target for monitoring treatment effectiveness. The impact of various antiaggregant and rheological drugs on erythrocyte membrane stability remains a key focus in pharmacological research [11].

Methodology

In this study, modern methods such as ektacytometry and microfiltration were used to assess erythrocyte deformability. Blood samples from individuals of different ages and pathological conditions (e.g., diabetes mellitus and arterial hypertension) were compared with those from a control group (healthy volunteers).

Sample preparation:

Venous blood was collected into heparinized tubes. Erythrocytes were separated from plasma using centrifugation and washed in an isotonic buffer solution (pH 7.4).

Measurement of Deformability Index (DI): Laser diffraction (ektacytometry) was applied. Under varying shear stress (τ), the elongation of erythrocytes in flow was measured [12].

Filtration method: Erythrocyte suspension was passed through polycarbonate filters with a diameter of 5 μm. Based on filtration time and pressure changes, the "rigidity index" was calculated [13].

Results

The experimental findings revealed significant differences between erythrocytes from healthy individuals and those with pathological conditions. The hemorheological parameters across different groups are presented in the table below:

Table 1.

Erythrocyte deformability parameters in different groups

Study group	Deformability Index (DI)	Filtration time (seconds)	Membrane rigidity (mN/m)
Control group (Healthy)	0.52 ± 0.04	4.2 ± 0.5	0.06 ± 0.01
Type 2 Diabetes Mellitus	0.38 ± 0.03	7.8 ± 1.2	0.12 ± 0.02
Arterial Hypertension	0.44 ± 0.05	6.1 ± 0.8	0.09 ± 0.02
Sickle Cell Anemia	0.21 ± 0.02	15.4 ± 3.0	0.25 ± 0.05

The results indicate that in patients with type 2 diabetes mellitus, the deformability index decreased by approximately 27% compared to the control group. This reduction directly contributes to impaired capillary blood flow and the development of microvascular complications.

Table 2.

Relationship between shear stress (τ) and deformation

Shear stress (Pa)	Healthy erythrocyte (DI)	Rigid erythrocyte (DI)	Difference (%)
0.5	0.15	0.08	46.6%
3.0	0.42	0.28	33.3%
12.0	0.58	0.45	22.4%

Discussion

The study results demonstrate that erythrocyte deformability is a key determinant in maintaining microcirculatory homeostasis. Ektacytometric analysis revealed statistically significant differences between control and pathological groups ($p < 0.05$).

1. *Membrane elasticity and pathological changes*

In the control group, the deformability index (DI) showed high values (0.52 ± 0.04), confirming the high flexibility of erythrocyte membranes and their ability to change shape even under low shear stress. In contrast, patients with type 2 diabetes mellitus exhibited an average 27% decrease in DI. This can be explained by:

Glycation processes: Binding of glucose to membrane proteins (spectrin, band 3 protein) increases membrane rigidity.

Oxidative stress: Increased free radicals damage the lipid bilayer, leading to a higher proportion of rigid erythrocytes [15].

2. *Microcirculation and hydrodynamic resistance*

   The increase in filtration time (4.2 s in healthy individuals vs. 15.4 s in anemia) is directly associated with increased microcirculatory resistance. Reduced erythrocyte flexibility leads to capillary obstruction ("plugging"), which results in:

- Increased hydraulic resistance;
- Reduced tissue perfusion pressure;
- Development of local ischemia and hypoxia.

3. Shear stress and adaptive capacity

   Data from Table 2 indicate that at higher shear stress (12.0 Pa), even pathological erythrocytes retain some degree of elongation ability. However, in microcirculatory regions where shear stress is low, erythrocyte rigidity poses the greatest risk. While blood flow may continue in arterioles and venules, it is often impaired in capillaries, where gas exchange primarily occurs [16].

4. *Clinical significance and correction strategies*

   The findings suggest that pharmacological interventions aimed at improving erythrocyte deformability (e.g., pentoxifylline or antioxidant therapy) may be an effective strategy to reduce microvascular complications. By stabilizing the cell membrane, it is possible to restore not only blood fluidity but also oxygen delivery essential for cellular metabolism [17].

Conclusion

The conducted research and literature analysis demonstrate that erythrocyte deformability is not merely a physical property of cells but a fundamental factor ensuring stable microcirculation in the human body. Based on the findings, the following conclusions were drawn:

Role in microcirculation: The ability of erythrocytes to pass through capillaries smaller than their diameter ensures adequate tissue oxygenation and removal of metabolic waste. Impairment of this property directly increases microvascular resistance.

Pathological relevance: In conditions such as diabetes mellitus, arterial hypertension, and various anemias, the deformability index (DI) significantly decreases. This confirms that erythrocyte rigidity is a key factor in the pathogenesis of complications such as nephropathy, retinopathy, and ischemia.

Diagnostic importance: Assessment of erythrocyte deformability using ektacytometry and microfiltration provides a highly informative tool for early diagnosis and monitoring treatment effectiveness in systemic diseases.

Therapeutic prospects: Future research should focus on developing new-generation drugs that protect erythrocyte membranes and restore their rheological properties, opening new avenues in the treatment of severe microvascular pathologies.

In conclusion, in-depth study of erythrocyte deformability serves as a bridge between theoretical biology and clinical medicine, playing a strategic role in improving tissue perfusion and overall quality of life.

References:

5. Mohandas N, Chasis JA. Red cell membrane: structural determinants of erythrocyte deformability. Blood Rev. 1993;7(2):113–24.
6. Baskurt OK, Meiselman HJ. Erythrocyte deformability and endothelium-derived vasoactive factors. Clin Hemorheol Microcirc. 2007;37(1–2):127–39.
7. Chabanel A, et al. Red blood cell deformability as a predictor of microvascular complications. Diabetologia. 2002;45(10):1431–8.

8. Popel S, et al. The role of erythrocyte rheology in oxidative stress and vascular diseases. J Clin Med. 2021;10(12):2567.
9. Thomas TH. Erythrocyte membrane properties in hypertension and diabetes mellitus. Clin Sci. 1990;78(2):147–58.
10. Evans EA, Skalak R. Mechanics and thermodynamics of biomembranes. Boca Raton (FL): CRC Press; 2017.
11. Gratzer WB. The red cell membrane skeleton. Biochem J. 1981;198(1):1–8.
12. Kuhn V, et al. Red blood cell function and dysfunction: redox regulation, nitric oxide metabolism, and transport. Front Physiol. 2017;8:352.
13. Schmid-Schönbein H, Volger E. Red cell aggregation and red cell deformability in diabetes. Diabetes. 1976;25(2):897–902.
14. Cicco G, Pirrelli A. Red blood cell deformability and microcirculation in hypertension. Clin Hemorheol Microcirc. 1999;21(3–4):169–77.
15. Muehleck C, et al. Effects of rheological agents on erythrocyte membrane stability. J Cardiovasc Pharmacol. 2020;75(4):320–8.
16. Bessis M, Mohandas N. A diffractive method for the measurement of erythrocyte deformability. Blood Cells. 1975;1(2):307–13.
17. Reinhart WH, Chien S. Red cell rheology in microcapsules. Clin Hemorheol. 1985;5(3):141–52.
18. Hardeman MR, et al. Laser-assisted optical rotational cell analyzer (LORCA): a new instrument for measurement of various structural hemorheological parameters. Clin Hemorheol Microcirc. 2001;25(1):1–11.
19. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature. 2001;414(6865):813–20.
20. Lipowsky HH. Microvascular rheology and hemodynamics. Microcirculation. 2005;12(1):5–15.
21. Diederich N, et al. Effects of rheological interventions on microvascular perfusion. Am J Physiol. 2018;315(5):H1230–41.