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Oxidative Stress and Red Blood Cell Membrane Damage in Diabetes Mellitus

Ivan Mutebi

Department of Pharmacognosy Kampala International University Uganda
Email: ivan.mutebi@studwc.kiu.ac.ug

ABSTRACT

Diabetes mellitus is a chronic metabolic disorder characterized by persistent hyperglycaemia and disturbances in carbohydrate, lipid, and protein metabolism. Beyond its established effects on vascular endothelium, nerves, kidneys, and the retina, diabetes causes substantial structural and functional changes in red blood cells. Red blood cells are particularly vulnerable to oxidative injury because they continuously transport oxygen, contain high concentrations of iron-rich haemoglobin, and possess membranes enriched in oxidation-sensitive polyunsaturated fatty acids. Chronic hyperglycaemia increases reactive oxygen species formation through glucose autoxidation, protein glycation, activation of the polyol and protein kinase C pathways, advanced glycation end-product formation, and impaired antioxidant defence. In diabetic red blood cells, excessive reactive oxygen species promote lipid peroxidation, membrane protein oxidation, spectrin–cytoskeleton disruption, phospholipid asymmetry, ion imbalance, and depletion of endogenous antioxidants. These changes reduce membrane fluidity and cellular deformability while increasing osmotic fragility, aggregation, phosphatidylserine exposure, microparticle release, eryptosis, and premature clearance from the circulation. Damaged erythrocytes may consequently impair microvascular blood flow and tissue oxygen delivery and may contribute to endothelial dysfunction, inflammation, thrombosis, anaemia, and diabetic vascular complications. This review examines the biochemical origins of oxidative stress in diabetes, the mechanisms through which oxidative injury damages the red blood cell membrane, the resulting rheological and clinical consequences, relevant laboratory biomarkers, and current therapeutic perspectives. Although antioxidant interventions are biologically plausible, effective protection of red blood cells depends primarily on sustained glycaemic control, management of cardiovascular risk factors, and therapies that reduce the metabolic sources of oxidative stress.

Keywords: oxidative stress; diabetes mellitus; red blood cells; membrane lipid peroxidation; erythrocyte deformability

INTRODUCTION

Diabetes mellitus encompasses a group of metabolic diseases in which insulin deficiency, insulin resistance, or both cause chronic elevation of blood glucose [1]. Persistent hyperglycaemia damages multiple organs and contributes to retinopathy, nephropathy, neuropathy, coronary artery disease, stroke, and peripheral vascular disease. Oxidative stress is widely regarded as an important mechanistic link between hyperglycaemia and these complications [2]. It develops when the production of reactive oxygen and nitrogen species exceeds the capacity of enzymatic and non-enzymatic antioxidant systems to neutralize them. Red blood cells, or erythrocytes, are increasingly recognized as active participants in diabetic vascular pathology rather than passive carriers of oxygen [3]. A normal erythrocyte is a flexible, biconcave cell capable of repeatedly deforming as it passes through capillaries narrower than its resting diameter. This mechanical behaviour depends on the integrity of the lipid bilayer, membrane proteins, spectrin-based cytoskeleton, intracellular adenosine triphosphate, ion gradients, and antioxidant systems [4]. Erythrocytes are highly susceptible to oxidative stress because they circulate for

approximately 120 days, are continuously exposed to oxygen, contain haemoglobin capable of generating oxidants, and lack nuclei and mitochondria [5]. They cannot synthesize new proteins or replace severely damaged cellular components. Their membranes also contain polyunsaturated fatty acids that are readily oxidized. Studies in diabetes have documented reduced erythrocyte deformability, increased oxidative modification, abnormal morphology, greater haemolytic susceptibility, and enhanced erythrocyte–endothelial interaction [6].

Red Blood Cell Membrane Structure and Antioxidant Protection

The erythrocyte membrane consists of a phospholipid-cholesterol bilayer connected to an underlying protein cytoskeleton. Integral proteins such as band 3 and glycophorins support ion transport, cellular recognition, and interactions with the cytoskeleton [7]. Spectrin, ankyrin, actin, protein 4.1, and protein 4.2 maintain membrane stability, elasticity, and the characteristic biconcave shape. Membrane phospholipids are asymmetrically distributed. Phosphatidylcholine and sphingomyelin occur mainly on the outer membrane surface, whereas phosphatidylserine and phosphatidylethanolamine are concentrated in the inner leaflet [8]. This organization is maintained by ATP-dependent enzymes, particularly flippases. Loss of asymmetry and externalization of phosphatidylserine signal macrophages to remove erythrocytes and also provide a negatively charged surface that can support coagulation. Despite their vulnerability, erythrocytes possess strong antioxidant defences. Superoxide dismutase converts superoxide radicals to hydrogen peroxide, which is subsequently removed by catalase and glutathione peroxidase [9]. Reduced glutathione neutralizes peroxides and protects protein sulfhydryl groups, while the pentose phosphate pathway supplies nicotinamide adenine dinucleotide phosphate needed to regenerate reduced glutathione. Vitamins C and E, uric acid, and other circulating antioxidants provide additional protection. In diabetes, these defences may become depleted, inhibited, or insufficient relative to oxidant production [10].

Sources of Oxidative Stress in Diabetes

Hyperglycaemia promotes oxidative stress through several interconnected pathways. First, glucose can undergo autoxidation, producing superoxide, hydrogen peroxide, and reactive carbonyl intermediates [11]. Second, non-enzymatic glycation modifies haemoglobin, membrane proteins, and plasma proteins. Early glycation products undergo further oxidation to produce advanced glycation end-products, a process often described as glycoxidation. Advanced glycation end-products can cross-link proteins, alter membrane organization, and interact with their cellular receptor, RAGE, to stimulate inflammation and oxidant-generating enzymes [12]. Glycation of erythrocyte membrane proteins, including spectrin, ankyrin, and band 3, weakens membrane–cytoskeletal interactions and contributes to reduced flexibility. Glycated haemoglobin may also be more susceptible to oxidation and may release haem or iron, which catalyses highly reactive hydroxyl radical formation through Fenton chemistry [13-17]. Hyperglycaemia also increases glucose entry into the polyol pathway. Aldose reductase converts glucose to sorbitol while consuming NADPH. Because NADPH is required for glutathione regeneration, excessive polyol pathway activity can weaken the erythrocyte antioxidant system. Protein kinase C activation, increased advanced glycation, hexosamine pathway activity, and altered cellular redox signalling further amplify oxidative stress in diabetic tissues [14,18-25]. These pathways are closely interrelated and may sustain oxidant production even after glucose concentrations improve, contributing to the concept of metabolic memory.

Lipid Peroxidation and Membrane Instability

Lipid peroxidation is a principal mechanism of oxidative erythrocyte membrane damage. Hydroxyl radicals and other oxidants remove hydrogen atoms from polyunsaturated fatty acids, producing lipid radicals [15,26-30]. These radicals react with oxygen and initiate chain reactions that generate lipid hydroperoxides and reactive aldehydes such as malondialdehyde and 4-hydroxynonenal. The resulting products alter membrane fluidity, permeability, thickness, and lipid–protein interactions. Oxidized phospholipids can disturb the normal cholesterol-to-phospholipid balance and create membrane regions that are rigid or mechanically unstable. Reactive aldehydes also form covalent bonds with membrane proteins, causing cross-linking and loss of function [31-34]. Research has reported altered lipid composition and increased oxidative damage in erythrocyte membranes from individuals with diabetes. These effects make erythrocytes less capable of changing shape during capillary passage. Oxidized membranes are also more permeable to calcium and other ions, contributing to cellular dehydration, volume changes, potassium loss, and increased fragility [35-40].

Oxidation of Membrane Proteins and the Cytoskeleton

Oxidative stress damages erythrocyte proteins through carbonyl formation, sulfhydryl oxidation, nitration, glycation, fragmentation, and cross-linking [18, 41-44]. Spectrin oxidation is particularly important because spectrin forms the flexible network that supports the lipid bilayer. Oxidative cross-links between spectrin molecules increase cytoskeletal stiffness and reduce membrane elasticity. Band 3 is another major target. Oxidative modification and haemichrome binding encourage band 3 clustering. Naturally occurring antibodies recognize

these clusters and promote complement deposition and macrophage-mediated erythrocyte clearance [19, 45-48]. Oxidation can also impair membrane-associated enzymes and transporters, including calcium pumps and sodium–potassium ATPase. Declining intracellular ATP aggravates these abnormalities. ATP is required to maintain membrane phospholipid asymmetry, ion gradients, cytoskeletal interactions, and normal cell shape [20, 48-50]. Diabetes-related metabolic stress may therefore establish a cycle in which oxidative injury impairs membrane function, ion imbalance increases cellular stress, and the damaged erythrocyte becomes increasingly susceptible to additional oxidation [21,51-53].

Reduced Deformability, Aggregation, and Microvascular Dysfunction

Reduced erythrocyte deformability is one of the most clinically relevant consequences of diabetic membrane damage [22]. Healthy erythrocytes elongate under shear stress and pass through small vessels with minimal resistance. Oxidized and glycated erythrocytes become rigid, spherical, or irregular and require greater force to traverse the microcirculation. A contemporary clinical study found that impaired red blood cell deformability is associated with diabetes and may occur relatively early in the disease process [23]. Reduced deformability increases blood viscosity, delays capillary transit, and may decrease tissue oxygenation. These effects are especially significant in tissues already vulnerable to diabetic microangiopathy, including the retina, kidney, peripheral nerves, myocardium, and lower limbs [24]. Diabetes may also increase erythrocyte aggregation. Glycation and oxidation alter membrane charge, glycocalyx properties, and interactions with plasma proteins such as fibrinogen. Aggregated erythrocytes increase low-shear blood viscosity and may obstruct microvascular flow. Damaged erythrocytes can additionally adhere more strongly to endothelial cells, stimulating endothelial activation and impairing nitric oxide availability [25]. Erythrocytes normally participate in nitric oxide metabolism and may release ATP in response to low oxygen tension, thereby promoting vasodilation. Membrane rigidity and oxidative stress can disrupt these functions. Consequently, diabetic erythrocyte dysfunction may combine with endothelial injury to create a self-reinforcing state of vasoconstriction, inflammation, poor perfusion, and tissue hypoxia [26].

Phosphatidylserine Exposure, Eryptosis, and Microparticle Formation

Severe oxidative stress can initiate eryptosis, a form of programmed erythrocyte death [27]. It is characterized by calcium entry, activation of potassium channels, cell shrinkage, membrane scrambling, and phosphatidylserine exposure. Experimental glycation of erythrocytes increases reactive oxygen species, phosphatidylserine externalization, haemolytic susceptibility, and uptake of erythrocytes by endothelial cells [28]. Phosphatidylserine-exposing erythrocytes are rapidly recognized by macrophages in the spleen and liver, shortening their circulatory lifespan. If erythrocyte removal exceeds bone marrow replacement, anaemia may develop or worsen. Anaemia in diabetes is multifactorial and may also involve kidney disease, inflammation, nutritional deficiency, reduced erythropoietin production, and medication effects. Nevertheless, oxidative membrane damage and premature erythrocyte clearance may provide an additional contribution [29]. Oxidatively stressed erythrocytes also shed membrane-derived microparticles. These vesicles can carry phosphatidylserine, oxidized lipids, haemoglobin, and haem. Their procoagulant surface may enhance thrombin generation, while extracellular haemoglobin can consume nitric oxide and promote vascular oxidative stress [30]. Thus, erythrocyte membrane injury may influence thrombosis and endothelial dysfunction beyond the lifespan of the damaged cell itself.

Biomarkers and Assessment

Erythrocyte oxidative injury may be evaluated using biochemical and biophysical methods. Common markers include malondialdehyde, thiobarbituric acid-reactive substances, lipid hydroperoxides, protein carbonyls, oxidized glutathione, reduced glutathione, catalase, superoxide dismutase, glutathione peroxidase, methaemoglobin, and antioxidant vitamins [31]. Membrane damage can be assessed through osmotic fragility, mechanical fragility, phosphatidylserine exposure, membrane lipid composition, electron microscopy, atomic force microscopy, microparticle measurement, and analysis of spectrin or band 3 oxidation. Ektacytometry, filtration methods, and microfluidic systems can quantify erythrocyte deformability [32]. However, many oxidative biomarkers lack standardized reference intervals and are influenced by diet, age, smoking, kidney function, medications, sample storage, and analytical methodology. They therefore remain primarily research tools rather than routine diagnostic tests. HbA1c remains essential for evaluating average glycaemia, although altered erythrocyte lifespan can affect its interpretation [33]. Premature erythrocyte destruction may produce a falsely low HbA1c, whereas prolonged survival or iron deficiency may raise it independently of glucose exposure.

CONCLUSION

Oxidative stress is a central mechanism connecting chronic hyperglycaemia with red blood cell membrane injury in diabetes mellitus. Excess reactive species generated through glucose autoxidation, glycation, haemoglobin oxidation, and altered metabolic pathways attack membrane lipids, proteins, antioxidant systems, and the spectrin

cytoskeleton. The resulting erythrocytes are rigid, fragile, adhesive, procoagulant, and susceptible to eryptosis and premature clearance. These alterations may impair capillary perfusion and oxygen delivery while intensifying endothelial dysfunction, inflammation, thrombosis, anaemia, and diabetic microvascular complications. Red blood cell abnormalities should therefore be viewed as both indicators of systemic metabolic stress and possible contributors to disease progression. Although erythrocyte oxidative biomarkers and deformability measurements have research and prognostic potential, methodological standardization and longitudinal clinical studies are needed before routine implementation. Comprehensive diabetes management remains the foundation of prevention. Future research should determine whether targeted protection of erythrocyte membranes can improve microvascular blood flow and reduce clinically meaningful diabetic complications.

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