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Review

The Postoperative Redox Status of Patients with Diabetes Mellitus: a Mini Review

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Abstract

The human organism is exposed to variable endogenous and exogenous factors that result in the induction of free radicals. Surgeries are associated with increased inflammation and production of free radicals through various mechanisms, including ischemia, hemolysis, or endogenous antioxidant depletion. Importantly, a more oxidized redox status could lead to the condition known as "oxidative stress," which is related to alterations in insulin signaling and may lead to insulin resistance and diabetes mellitus. Therefore, patients with diabetes who undergo surgery could be more vulnerable compared with healthy individuals. Thus, this review focuses on the currently available literature regarding the link between the redox status and diabetes complications after surgery and the methods used to assess the redox status of patients, which is the first step to designing an appropriate treatment strategy. To this end, we queried PubMed for novel works published up to May 2020.

Keywords

diabetes, free radicals, oxidative stress, reactive oxygen species, surgery

INTRODUCTION

Diabetes mellitus (DM) constitutes a major worldwide health issue, affecting the quality of life of a considerable proportion of the global population. According to the World Health Organization, the number of patients with DM had reached 422 million by 2014, highlighting a significant increase from the 108 million affected individuals back in 1980. Although DM was considered a disease mainly observed in the Western countries, its prevalence in middle-income countries is increasing.[1] DM is related to various vascular complications, including atherosclerosis, diabetic neuropathy, and retinopathy, which could lead to further complications such as blindness, cardiovascular (CVD), and renal diseases.[2-4] Interestingly, approximately 1.5 million people died in 2012 of causes directly related to DM, while an additional 2.2 million deaths occurred due to DM-associated CVDs.[1] DM also has a marked economic impact; indeed, studies[5] that have analyzed the economic consequences of this disease have indicated that the absolute global economic burden would increase from 1.3 in 2015 to 2.1 trillion USD in 2030.

There is increasing evidence regarding the association between postoperative hyperglycemia and increased free radical, inflammatory mediator, and free fatty acid overproduction, which could lead to cellular damage, vascular and immune dysfunction, and further increase in the cellular redox status. Indeed, chronic systemic inflammation and increased oxidative stress affect the pathogenesis of obesity and type 2 DM.[6] Specifically, the link between oxidative stress and insulin resistance has drawn marked attention, with the body of evidence regarding the connection of increased redox status with dysregulation of insulin signaling being continually on the rise. As it seems that

this vicious circle is postoperatively induced in patients with DM, factors enhancing disease progression and those related to its complications should be extensively analyzed to reveal the most effective treatment intervention for each patient. This study aimed to review the postoperative redox status of patients with DM and propose methods reported in the literature for the immediate evaluation of this condition (See **Fig. 1**).

Free radicals and oxidative stress

Free radicals are products of the organisms' normal metabolism. They are divided into two main categories, reactive oxygen species (ROS), such as superoxide anion radical ($O_2^{\bullet -}$), hydroxyl radical ($OH^{\bullet}$), and peroxyl radical ($RO_2^{\bullet}$), and reactive nitrogen species (RNS), such as nitric oxide and the peroxynitrite radical ($ONOO^{\bullet}$).[7] Free radicals are mainly derived from the mitochondria, endoplasmic reticulum, NADPH oxidases, and peroxisomes.[8] Increased intracellular presence of free radicals could lead to oxidative stress. This condition is caused when the intracellular levels of free radicals overwhelm the anti-oxidant defences of the cells.[9] Increased redox status in the cell could result in irreversible damage in the intracellular constituents, including proteins, lipids, and nucleic acids, causing severe modifications in the structure and function of these molecules. This pathological condition is associated with adiposity and insulin resistance in patients with DM, indicating that it is an evolutionary factor and not just a consequence of disease development. Especially, free radical generation is induced under stress conditions, such as DM, leading to oxidative stress.[10]

Free radicals and cell signaling

In the last 2 decades, it has become apparent that free radicals at intermediate levels could serve as second messengers in cell signaling. Specifically, they oxidize the thiol groups of cysteine residues and change the function of proteins. Especially, mitogen-activated protein kinase (MAPK) signaling is affected by the redox status, regulating various cellular processes, including cell differentiation, apoptosis, and proliferation.[11] Endogenous free radicals could also activate the apoptosis signal-regulated kinase 1 (ASK1) and induce p38 MAPK signaling in several human cell types. Moreover, free radicals and especially ROS can regulate phosphoinositide-3-kinase (PI3K), serving as a second messenger for the stimulation of growth factors and cytokines. ROS can further maintain the activation of PI3K as they can inhibit PTEN, which is the negative regulator of PI3K.[11,12] To the best of our knowledge, PI3K is considered the key mechanism in insulin signaling and elicits several insulin effects on glucose and lipid metabolism. Considering the critical role of PI3K in regulating glucose metabolism, it is obvious that alterations in expression or regulation of this enzyme would play a crucial role in the development of insulin resistance and DM.

Free radicals and macrophages

Free radicals are mandatory for the induction of M1 macrophages. Macrophages are key molecules in wound healing and orchestrate the switch from the inflammatory to the proliferative phase.[13] Free radicals contribute to the activation of nuclear factor-κB (NF-κB) and p39 MAPK

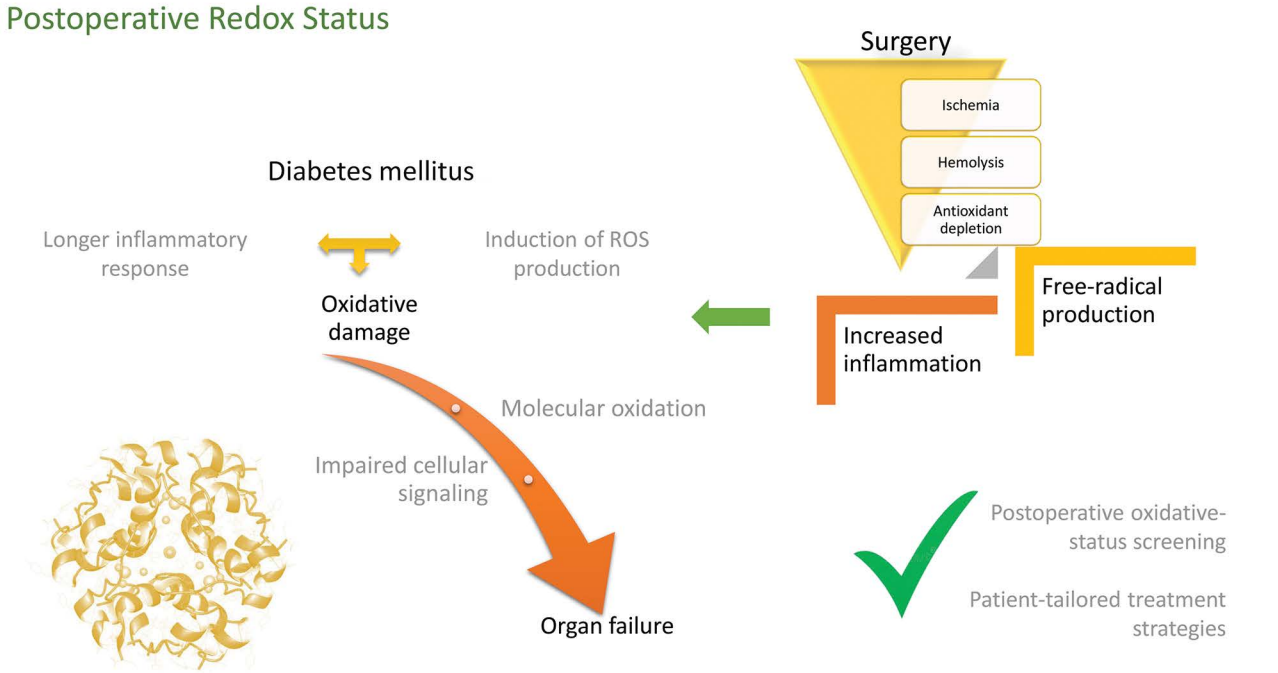

Figure 1. Graphical abstract

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signaling that promotes pro-inflammatory gene expression in macrophages. These molecules further exacerbate inflammation by mediating tissue injury (especially after surgery) and by secreting pro-inflammatory cytokines and proteases, inducing the production of ROS in the tissues. Hyperglycemia contributes to chronic inflammation after injury, including surgical trauma, through activation of THP-1 derived macrophages, increases the expression of pro-inflammatory cytokines and chemokines by macrophages, and decreases their ability of phagocytosis, which are required for the resolution of inflammation. Moreover, hyperglycemia has been linked to poor wound healing and increased advanced glycation end products, which further activate NF-κB and induce TNF-a expression.[14] The aforementioned lead to the maintenance of the vicious circle of the increased redox status caused by chronic inflammation in patients with DM after an operation, which would subsequently lead to further complications of insulin sensitivity state and signaling.

Surgical stress and anesthesia

Normally, the plasma glucose levels are maintained at 60–100 mg/dl (3.3–5.5 mmol/l). However, the balance between glucose production and utilization is hindered after surgery and anesthesia. Surgeries are considered major activators of the inflammatory response.[15] Even in cases where there are only small scar contractures, an increase in the levels of C-reactive protein is usually observed, leading to increased inflammatory stress.[16] Nevertheless, despite being an homeostatic mechanism, the inflammatory response could be overactivated and maintained after surgery, especially in patients with hyperglycemia, resulting in organ failure and increased mortality.[17]

It has been reported that postoperative hyperglycemia occur in 20%–40% and 80% of patients after general and cardiac surgery, respectively.[18,19] Interestingly, another study reported that 12%–30% of patients who develop postoperative hyperglycemia are not diabetic, but many of those who newly develop hyperglycemia after surgery could develop DM after 1 year.[20] Similarly, a recent study revealed increased oxidative damage in patients who underwent cardiac surgery highlighting the risk of further development of the disease in patients with DM who undergo such surgery.[21]

After a surgery, counter regulatory hormones, such as epinephrine, cortisol, and growth hormones, are secreted and cause excessive release of inflammatory cytokines (e.g., tumor necrosis factor-α [TNF-α], interleukin [IL]-6, IL-10) which increase insulin resistance by interfering with insulin signaling.[22] Specifically, TNF-α could prevent insulin-mediated activation of PI3K, which is involved in tissue glucose uptake, by activating c-Jun NH_2-terminal kinase that phosphorylates insulin receptor substrate-1. In addition, there is evidence that TNF-α affects the translocation of the glucose transporter GLUT-4, causing decrease in cell glucose uptake.[23] Moreover, cortisol stimulates protein catabolism and promotes gluconeogenesis, while epinephrine stimulates glucagon secretion and inhibits insulin release by pancreatic β-cells.[24] The aforementioned entirely affect the state of insulin action, leading to increased insulin resistance, beginning from postoperative day 1 and lasting for 9–21 days.[25]

Biomarkers for in vivo assessment of the redox status

It appears that the assessment of the redox status of each patient with diabetes is mandatory to evaluate his/her condition, develop the appropriate treatment strategy, and prevent any further complications. Several biomarkers have been proposed for this purpose and can be applied to plasma, erythrocyte, or tissue lysate samples in in vivo settings. These biomarkers are divided in three major categories: those evaluating the oxidative status of biomolecules, their antioxidant capacity, and the oxidative potential.[26]

The first category includes protein carbonyls, as a protein oxidation biomarker. Accordingly, human serum albumin (HAS, the most abundant protein in plasma) dimmers can be assessed to evaluate the levels of protein oxidation and modifications in the function of this protein.[27] Moreover, thiobarbituric acid reactive substances (TBARS) are widely reported in the literature as markers of lipid peroxidation.[28,29] Likewise, malondialdehyde (lipid peroxidation by-product) levels can be measured with high performance liquid chromatography with diode-array detector (HPLC-DAD) and represent a more specific and credible evaluation of lipid oxidation compared to TBARS.[30]

In the second category are included biomarkers that assess the oxidative potential, such as H_2O_2, $NO^{\bullet}$, catalase (main enzyme for H_2O_2 detoxification), glutathione peroxidase (GPX), and peroxiredoxin activity.

Further, among the biomarkers of antioxidant capacity evaluation are the ratio of reduced (GSH) to oxidized (GSSG) glutathione (GSH/GSSG), the levels of GPX, NADPH oxidase, thioredoxin, thioredoxin reductase, superoxide dismutase, glutathione S-transferase, NAD^+ kinase, reduced HSA, and total antioxidant capacity.[26,28,29,31]

Except for the aforementioned markers, most of whom are measured spectrophotometrically, there are also markers to evaluate the expression of several enzymes or proteins, such as Nrf2, through western blot analysis.[32] The expression of Nrf2 target genes can also be assessed by real time polymerase chain reaction in blood and tissues.[33]

Finally, a novel, fast, and accurate method of evaluating the redox status in plasma samples has been proposed, in which a diagnostic system assesses the balance between oxidant and reductant molecules and the antioxidant capacity in a specimen.[7,34] It should be noted that the application of these markers has been proposed for the prediction of complications following pediatric cardiac surgery.[35]

Treatment and future perspectives

As the redox status of patients with diabetes can be more oxidized postoperatively, which could lead to disastrous consequences, a strategy to obtain the best outcomes in this patient population is needed. It has been reported that moderate exercise is effective for improving the redox homeostasis of patients with DM by increasing endogenous antioxidant defences and improving redox signaling.[36] Moreover, during exercise, glucose uptake occurs in an exercise intensity and duration-dependent manner, independent of insulin. Indeed, increased GLUT4 trafficking has been reported in such cases due to changes in Ca^{2+} concentration, cell energy status, remodeling of the actin cytoskeleton via GTPase Rac1, and nitric oxide mediation.[37-39] Further, exercise was found to increase parameters of insulin sensitivity.[40,41] Nevertheless, it should be noted that during recovery from surgery, patients might have limited mobility. Therefore, it is recommended for the patients to exercise to the degree that this is possible to improve the postoperative functional status and reduce the complication and mortality rates.[42]

Antioxidant administration could be another effective means of improving glucose homeostasis and insulin sensitivity.[43] Indeed, ascorbic acid and tocopherol administration could successfully increase insulin sensitivity in peripheral tissues.[44,45] To the same end, flavonoid and α-lipoic acid have been proposed and presented similar positive results.[46] However, a question arises regarding their beneficial use combined with exercise for patients with DM. Although antioxidants have been proven to be essential tools in the management of oxidative-stress related diabetic pathologies, their efficacy remains controversial.[47] Actually, Ristow et al. examined the effects of vitamin C and E supplementation on exercise-induced insulin sensitivity and stated that exercise had positive effects on insulin sensitivity parameters only in the absence of antioxidants.[48]

Finally, preserving a healthy weight is also crucial for treating insulin resistance. Restricting fat, especially natural fat, can combat systemic inflammation and decrease the occurrence of DM.[49] Novel approaches need to be created to raise the accuracy of measuring visceral fat, as the body mass index can be wrongly influenced by several parameters.[49]

CONCLUSIONS

Patients with DM may be burdened by more complicated wound healing, increased redox status, and longer chronic inflammatory response after surgery compared to healthy individuals. Undoubtedly, oxidative stress constitutes a fundamental issue in patients with DM and could result in oxidation of molecules, organ failure, and impaired cellular signaling. The limited antioxidant reserves due to the existence of inflammation in the recovery period and the induction of ROS production could maintain this condition and increase the vulnerability of these patients to oxidative damage. Therefore, postoperative screening of the oxidative status of these patients is mandatory, and a clear patient-tailored treatment strategy should be developed to minimize the potentially detrimental consequences of an increased oxidative status.

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Competing interests

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Послеоперационный окислительно-восстановительный статус пациентов с сахарным диабетом: мини-обзор

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Резюме

Организм человека подвергается воздействию различных эндогенных и экзогенных факторов, которые приводят к индукции свободных радикалов. Хирургическое вмешательство ассоциируется с усилением воспаления и выработкой свободных радикалов через множество механизмов, включая ишемию, гемолиз или эндогенное истощение антиоксидантов. Что еще более важно, окислительно-восстановительный статус с более высокой степенью окисления может вызвать состояние, называемое «окислительный стресс», которое связано с изменениями в передаче сигналов инсулина и может привести к инсулинорезистентности и диабету. Следовательно, пациенты с диабетом, перенёсшие операцию, могут быть более восприимчивыми, чем здоровые люди. Поэтому в данном обзоре основное внимание уделяется имеющейся литературе о взаимосвязи между окислительно-восстановительным статусом и послеоперационными осложнениями диабета и методам оценки окислительно-восстановительного статуса пациентов, что является первым шагом к разработке соответствующей стратегии лечения. С этой целью мы искали новые разработки в PubMed, опубликованные до мая 2020 года.

Ключевые слова

диабет, свободные радикалы, окислительный стресс, активные формы кислорода, хирургия