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Original Research Article

Correlation Between Malondialdehyde and Neutrophil/Macrophage Status After Tooth Extraction in Diabetic Rats

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ABSTRACT

Diabetes mellitus (DM) is a chronic metabolic disorder characterized by persistent hyperglycemia, which increases reactive oxygen species (ROS) production, leading to lipid peroxidation, inflammatory imbalance, and delayed wound healing. Malondialdehyde (MDA) is a biomarker of oxidative stress, while neutrophils and macrophages play essential roles in the inflammatory and resolution phases of socket healing. This study aimed to analyze oxidative stress and inflammatory cell responses following tooth extraction in diabetic rats. A post-test-only control group experimental design was conducted using 20 male *Rattus norvegicus* (200–250 g). Diabetes was induced by a single intraperitoneal injection of streptozotocin (50 mg/kg body weight). On the third day after lower incisor extraction, blood samples were collected to measure MDA levels using the thiobarbituric acid reactive substances (TBARS) method, and mandibular tissues were stained with hematoxylin–eosin for microscopic evaluation of neutrophil and macrophage counts. Data were analyzed using the independent samples t-test, with $p < 0.05$ considered statistically significant. The results showed that diabetic rats had significantly higher glucose and MDA levels compared to controls ($p < 0.05$). Neutrophil counts were significantly increased, whereas macrophage counts were significantly decreased following tooth extraction ($p < 0.05$). These findings indicate that hyperglycemia enhances oxidative stress and disrupts the balance of inflammatory cells, thereby contributing to impaired post-extraction wound healing under diabetic conditions.

Keywords: Complication of diabetes; Inflammatory; Wound healing; Dental treatment.

Introduction

Chronic hyperglycemia caused by decreased insulin sensitivity, impaired insulin secretion, or a combination of both is a characteristic of diabetes mellitus (DM), a chronic metabolic disease .[1,2] Clinically, there are several categories of DM: Type 1 DM, Type 2 DM, gestational diabetes, and other specific forms. According to data from the International Diabetes Federation (IDF), approximately 463 million individuals aged 20–79 years were living with diabetes in 2019, representing a global prevalence of 9.3%.[3]
One of the systemic consequences of diabetes is delayed wound healing, including after tooth extraction procedures. Hyperglycemic conditions promote excessive production of reactive oxygen species (ROS) in periodontal tissues, which further aggravates local insulin resistance and tissue dysfunction.[4,5] At the cellular level, metabolic dysregulation in diabetes enhances mitochondrial superoxide generation in endothelial cells of both the macrovascular and microvascular systems. Excessive superoxide formation subsequently activates several key biochemical pathways implicated in diabetic complications, including increased polyol pathway flux, accelerated formation of advanced glycation end-products (AGEs), increased expression of AGE receptors and their ligands, stimulation of protein kinase C (PKC) isoforms, and heightened activity of the hexosamine pathway.[6] Moreover, superoxide directly suppresses important anti-atherosclerotic enzymes, such as endothelial nitric oxide synthase (eNOS) and prostacyclin synthase. Through these interconnected mechanisms, intracellular ROS stimulate pro-inflammatory signaling cascades and induce persistent epigenetic modifications that sustain inflammatory gene expression. Elevated oxidative stress also intensifies lipid peroxidation of cellular membranes, generating malondialdehyde (MDA), a stable end-product widely recognized as a biomarker of oxidative damage.[2]
Reactive oxygen species function not only as damaging molecules but also as signaling mediators in inflammatory processes. Excessive ROS production by polymorphonuclear neutrophils (PMNs) at sites of inflammation contributes to endothelial dysfunction and subsequent tissue injury.[7] Under diabetic conditions, sustained hyperglycemia alters intracellular glucose metabolism, thereby disrupting each stage of the inflammatory phase and prolonging tissue repair. ROS play a significant role in this dysregulated healing response.[8] Clinically, delayed post-extraction healing has been reported in approximately 12.5% of patients with diabetic complications.[9]
This study aims to investigate the relationship between malondialdehyde levels and neutrophil and macrophage counts after tooth extraction in diabetic rats. Neutrophils are the primary leukocytes in the early phase of healing and form extracellular traps (NETs) by releasing decondensed chromatin coated with cytotoxic proteins. Neutrophil counts positively correlate with the severity of inflammation.[10] Neutrophils and macrophages have interconnected mechanisms that influence the healing process. Macrophages function

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in host defense, regulation of inflammation, removal of apoptotic cells, and tissue restoration.[11] Although several studies have investigated oxidative stress and inflammatory cell responses in diabetic wound healing, the association between malondialdehyde levels and neutrophil and macrophage counts following tooth extraction in diabetic models remains unclear. Clarifying this relationship is important for a better understanding of the mechanistic link between oxidative stress and inflammatory cell dynamics during post-extraction healing under diabetic conditions. Such understanding may provide a scientific basis for the development of targeted therapeutic strategies to improve oral wound healing in diabetic patients.

Materials and Methods

Chemicals

All chemicals were purchased from commercial distributors. The following is a list of the chemicals used: Streptozotocin (STZ) (Bioworld Scytek, Logan, United States), Thiobarbituric acid (TBA) (Sigma Aldrich, Registered and Commercial Office Jakarta, Indonesia), Trichloroacetic acid (TCA) (Sigma Aldrich, Registered and Commercial Office, Jakarta, Indonesia), Hydrochloric Acid (Merck, Jakarta, Indonesia), Tetramethoxypropane (TMP) (Merck, Jakarta, Indonesia), Formalin (10% Neutral Buffered Formalin) (Merck, Jakarta, Indonesia), Phosphate buffer (Merck, Jakarta, Indonesia), Ethanol (Merck, Jakarta, Indonesia), Xylene (Merck, Jakarta, Indonesia), Hematoxylin (Merck, Jakarta, Indonesia) and Eosin (Merck, Jakarta, Indonesia).

Ethical Approval

Ethical approval was obtained from the Health Research Ethical Clearance Committee of the Faculty of Dental Medicine, Universitas Airlangga (No. 0063/HRECC.FODM/II/2024). Information regarding the committee is available on the official university website (https://fkg.unair.ac.id/en/#). The entire experimental protocol complied with institutional standards for the care and handling of laboratory animals.

Preparation of Experimental Animals

Twenty male Wistar rats (*Rattus norvegicus*), aged 2–3 months and weighing 200–250 g, were included in this experiment. Male animals were intentionally selected to minimize hormonal fluctuations related to the estrous cycle, which could potentially affect glucose regulation and inflammatory parameters. Throughout the study period, the rats were maintained under controlled laboratory conditions with unrestricted access to standard feed and water. Their general health conditions were observed and recorded daily.

The animals were randomly allocated into two equal groups (n = 10 each): a non-diabetic control group (Non-DM) and a diabetic group (DM). Experimental diabetes was induced in the DM group through a single intraperitoneal injection of streptozotocin (STZ) (50 mg/kg body weight). Blood glucose concentrations were assessed 28 hours after STZ injection using a glucometer. Rats demonstrating blood glucose levels ≥ 250 mg/dL were classified as diabetic and retained in the DM group for subsequent procedures.

For the tooth extraction procedure, general anesthesia was achieved using ketamine (50–80 mg/kg) in combination with xylazine (20 mg/kg). The lower left incisor was carefully removed using dental extraction forceps. All animals were euthanized on the third day following extraction to obtain tissue samples for biochemical assessment and histopathological evaluation.

Table 1: Results of the Shapiro–Wilk Normality Test for Study Variables

Parameter	Shapiro-Wilk			
	FBG	MDA	Neutrophile	Macrophage
Non-DM	0.600	0.743	0.657	0.376
DM	0.470	0.889	0.134	0.479

Sample Collection

Blood samples for malondialdehyde (MDA) analysis were collected from the cardiac chamber of euthanized rats, with approximately 2 mL obtained from each animal. MDA concentrations were measured using the thiobarbituric acid reactive substances (TBARS) assay, employing thiobarbituric acid (TBA) and trichloroacetic acid (TCA) as reagents to quantify lipid peroxidation products.[12]

On day 3 following the intervention, the animals were anesthetized with a ketamine–xylazine combination prior to tissue harvesting. The left mandible was carefully dissected and trimmed to isolate the extraction socket area. The specimens were subsequently fixed in 10% neutral buffered formalin and subjected to decalcification before histological preparation. The processed tissues were embedded, sectioned, and stained with hematoxylin and eosin (H&E) for the evaluation of inflammatory cell infiltration, specifically neutrophils and macrophages.

Microscopic examination was performed using a light microscope. Cell counts were conducted in five distinct fields of view per slide at 400× magnification under binocular observation.

Statistical Analysis

Glucose levels, MDA levels, and neutrophil and macrophage counts are presented as mean ± standard deviation (SD). Data normality and homogeneity of variance were assessed prior to analysis using Shapiro–Wilk (p-value>0.05). The independent sample t-test was used to compare the diabetic and non-diabetic groups, and Levene's test was applied to evaluate the equality of variances. A p-value < 0.05 was considered statistically significant.

Results and Discussion

The normality of the study variables was evaluated using the Shapiro–Wilk test, as presented in Table 1. The results indicated that all variables met the assumption of normality, (p-value>0.05). Therefore, parametric statistical methods were considered appropriate for further analysis. Accordingly, the independent samples t-test was employed to compare the diabetic and non-diabetic groups (p-value < 0.05). The differences between the non-diabetic and diabetic groups were analyzed using the independent samples t-test, as presented in Table 2. Prior to the analysis, Levene's test indicated homogeneity of variances for all variables (p > 0.05), confirming that the assumption of equal variances was met. Fasting blood glucose (FBG) levels were substantially higher in the diabetic group (456.700 ± 44.681) compared to the non-diabetic group (118.400 ± 24.649). Levene's test (table 2) showed p = 0.140, indicating that the assumption of equal variances was met. The independent t-test (table 2) revealed a highly significant difference between groups (p < 0.001), confirming successful induction of hyperglycemia in the diabetic rats. MDA levels were also elevated in the diabetic group (20.514 ± 3.351) compared to the non-diabetic group (14.606 ± 2.905). The homogeneity of variance assumption was satisfied, as indicated by Levene's test (p = 0.692). Statistical analysis showed a significant difference between groups (p = 0.001), suggesting increased oxidative stress in diabetic animals following tooth extraction. Neutrophil counts were markedly higher in diabetic rats (15.721 ± 0.543) than in non-diabetic rats (5.700 ± 0.485). Levene's test confirmed equal variances between groups (p = 1.000). The independent samples t-test demonstrated a highly significant difference (p < 0.001), indicating enhanced inflammatory cell infiltration under diabetic conditions. In contrast, macrophage counts were lower in the diabetic group (5.427 ± 1.135) compared to the non-diabetic group (11.000 ± 2.906). The assumption of homogeneity of variances was met (Levene's p = 0.344). The difference between groups was statistically significant (p < 0.001), reflecting a reduced macrophage response in diabetic rats during the healing phase. The present study demonstrates that diabetic conditions significantly increase MDA levels and neutrophil infiltration while reducing macrophage counts following tooth extraction. These findings indicate a clear shift in oxidative and inflammatory responses under hyperglycemic conditions. Previous studies have reported that diabetes impairs post-extraction healing and enhances oxidative damage in oral tissues,[13,14] thereby supporting the current findings.

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Table 2: Results of the Independent t-test for Study Variables

Parameter	Non-DM (Mean ± SD)	DM (Mean ± SD)	Levene's p-value	p-value (2-tailed)
FBG	118.400 ± 24.649	456.700 ± 44.681	0.140	<0.001
MDA	14.606 ± 2.905	20.514 ± 3.351	0.692	0.001
Neutrophil	5.700 ± 0.485	15.721 ± 0.543	1.000	<0.001
Macrophage	11.000 ± 2.906	5.427 ± 1.135	0.344	<0.001

The elevated MDA levels observed in diabetic rats reflect enhanced lipid peroxidation due to excessive reactive oxygen species (ROS) production. Hyperglycemia is known to promote ROS generation through several metabolic pathways, including protein kinase C activation, mitochondrial dysfunction, and advanced glycation end-product (AGE) formation.[20] These mechanisms increase intracellular oxidative stress, leading to membrane lipid damage and the formation of secondary products such as MDA,[2] Consistent with earlier reports, increased oxidative stress under diabetic conditions contributes to delayed tissue repair and prolonged inflammation.[14,13]

A notable finding of this study is the marked elevation of neutrophil counts in diabetic rats. Neutrophils are critical for early bacterial clearance; however, persistent neutrophil infiltration can exacerbate tissue injury due to excessive ROS release and proteolytic enzyme activity.[17] In hyperglycemic environments, neutrophils exhibit an enhanced oxidative burst and prolonged survival, which may explain the significantly higher neutrophil levels observed in this study.[17] This prolonged inflammatory phase is commonly reported in diabetic wound models.

Conversely, macrophage counts were significantly reduced in diabetic animals. Macrophages normally regulate the transition from the inflammatory to the proliferative phase of healing by clearing apoptotic neutrophils and secreting growth factors. Impaired macrophage recruitment and dysfunction under hyperglycemic conditions have been documented in diabetic wound studies.[18,19] Hyperglycemia and oxidative stress interfere with macrophage polarization, thereby delaying the transition toward the reparative phenotype required for tissue remodeling.[18] Reduced macrophage function has also been associated with impaired efferocytosis and the accumulation of inflammatory mediators in diabetic wounds.[21,23]

Conclusion

This study demonstrates that hyperglycemia significantly increases MDA levels and neutrophil counts while reducing macrophage counts following tooth extraction. These findings indicate that diabetes enhances oxidative stress and disrupts the balance of inflammatory cell responses during the early phase of healing. The observed imbalance between excessive neutrophil infiltration and reduced macrophage presence suggests impaired resolution of inflammation under diabetic conditions.

Conflict of Interest

The authors declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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