Journal of Neonatal Surgery
ISSN(Online): 2226-0439
Vol. 14, Issue 26s (2025)
https://www.jneonatalsurg.com

OPEN ACCESS

Comparative Evaluation of DNA Damage in SARS-CoV-2 Infected Patients with and Without Type 2 Diabetes Using Comet Assay

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Cite this paper as: Osamah Imad Ibrahem, Asma Sumiea Karomi, (2025) Comparative Evaluation of DNA Damage in SARS-CoV-2 Infected Patients with and Without Type 2 Diabetes Using Comet Assay, *Journal of Neonatal Surgery*, 14 (26s), 169-173

ABSTRACT

SARS-CoV-2, the virus that causes COVID-19, is a virus with a widespread systemic effect, not just on the respiratory system. Its most prominent effects include the ability to induce DNA damage as a result of oxidative stress and chronic inflammation. These effects are more severe in individuals with metabolic diseases such as type 2 diabetes mellitus (T2DM), where cellular damage is exacerbated.

The study included the analysis of 22 control and 72 blood samples from COVID-19 patients confirmed using RT-PCR, including 43 patients with type 2 diabetes and 29 non-diabetic patients. Samples were collected from Kirkuk General Hospital and Azadi Teaching Hospital in Iraq. The Comet assay was performed to measure DNA damage, and the results were analyzed using TriTek Comet Score v2.0.0.38 software, where damage was expressed as the percentage of DNA in tail. Statistical analyses were performed using a t-test to verify the significance of differences between groups.

The results of the Comet test showed that the diabetic group exhibited the highest percentage of DNA damage (38.03 ± 6.40%), followed by the non-diabetic COVID-19 group (3.06 ± 1.73%), and then the control group (1.71 ± 1.12%). The differences between the diabetic group and the other two groups were highly statistically significant ($p < 0.001$), while no significant differences were recorded between the non-diabetic group and the control group ($p > 0.05$).

The results indicate that type 2 diabetes significantly contributes to the increased impact of COVID-19 on DNA damage, likely due to increased systemic inflammation and oxidative stress. These findings underscore the importance of glycemic control and early intervention to reduce genetic complications resulting from the virus

Keywords: *COVID-19, Comet test, DNA damage, type 2 diabetes, genomic instability, oxidative stress*.

1. INTRODUCTION

Since its emergence in 2019, SARS-CoV-2 has become recognized as a multi-organ pathogen with significant systemic effects . While pulmonary complications were the focus of much early research, recent studies highlight the virus's ability to cause oxidative stress, mitochondrial dysfunction, and DNA damage, particularly in vulnerable populations (Delgado-Roche & Mesta, 2020) (Albarzanji *et al.*, 2024) .

Type 2 diabetes is a known risk factor for worsening COVID-19 symptoms due to chronic inflammation resulting from hyperglycemia and impaired immune function (Bornstein *et al.*, 2020) (Raza & Hamad , 2016). In these patients, elevated levels of reactive oxygen species (ROS) lead to genomic instability by breaking DNA strands and impeding repair mechanisms (Ceriello & Motz, 2004).

The comet assay, a sensitive technique for detecting DNA strand breaks in single cells, is widely used to measure genotoxicity in viral infections and chronic diseases (Zaman *et al.*, 2020) (Singh *et al.*, 1988). Previous studies suggest a symbiotic relationship between viral infection and metabolic disease in accelerating DNA damage (Sharma *et al.*, 2021).

This study aimed to examine and compare DNA damage levels using the comet assay in COVID-19 patients with and without diabetes, as well as healthy controls, to understand the impact of comorbid metabolic diseases on viral-induced genome instability.

2. METHODOLOGY

Study design and participants

The study included 72 participants divided into three main groups:

Group 1: 43 COVID-19 patients with type 2 diabetes

Group 2: 29 COVID-19 patients without diabetes

Group 3 (control): 22 healthy individuals without COVID-19 or any chronic diseases

SARS-CoV-2 infection was confirmed in the first and second groups using RT-PCR. All samples were collected from patients admitted to Kirkuk General Hospital and Azadi Teaching Hospital in Iraq during the study period.

Comet Test Procedure

Venous blood samples were collected, and the alkaline Comet test was performed. Cells were suspended in agarose gel, mounted on slides, and processed through electrophoresis, washing, and staining.

The resulting DNA migration patterns (the "tail" pattern) were analyzed using TriTek Comet Score v2.0.0.38 software, which calculated the percentage of DNA in the tail, a direct indicator of the degree of genetic damage in the cell.

Statistical Analysis

Means and standard deviations were calculated for the results of each group. An independent t-test was performed to compare the results between the three groups. Differences were considered statistically significant when the p-value was <0.05.

3. RESULTS

Table 1**. DNA Damage Analysis - DNA in Tail**.

Group	DNA in Tail (Mean ± SD)	p-value vs Control	p-value vs Non-Diabetic
Control	1.710 ± 1.122	N/A	N/A
SARS-CoV-2 Non-Diabetic	3.059 ± 1.730	> 0.05	N/A
SARS-CoV-2 with Diabetes	38.031 ± 6.395	< 0.001	< 0.001

The control group and the non-diabetic group were not statistically significant ($p > 0.05$). The diabetic group and the other groups were statistically significant ($p < 0.001$).

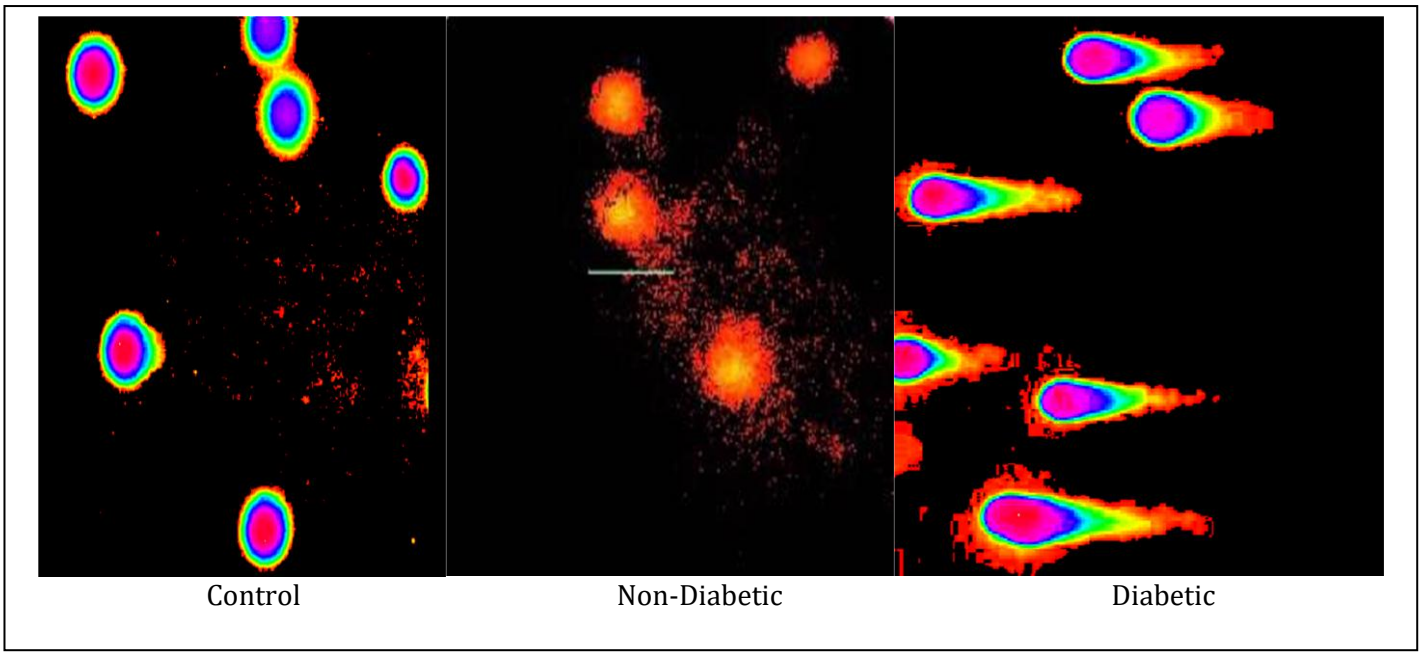

Figure 1**. shows the percentage of damage DNA in tail of the control, non-diabetic and diabetic groups using Tri Tek Comet score 2.0.0.38 software.**

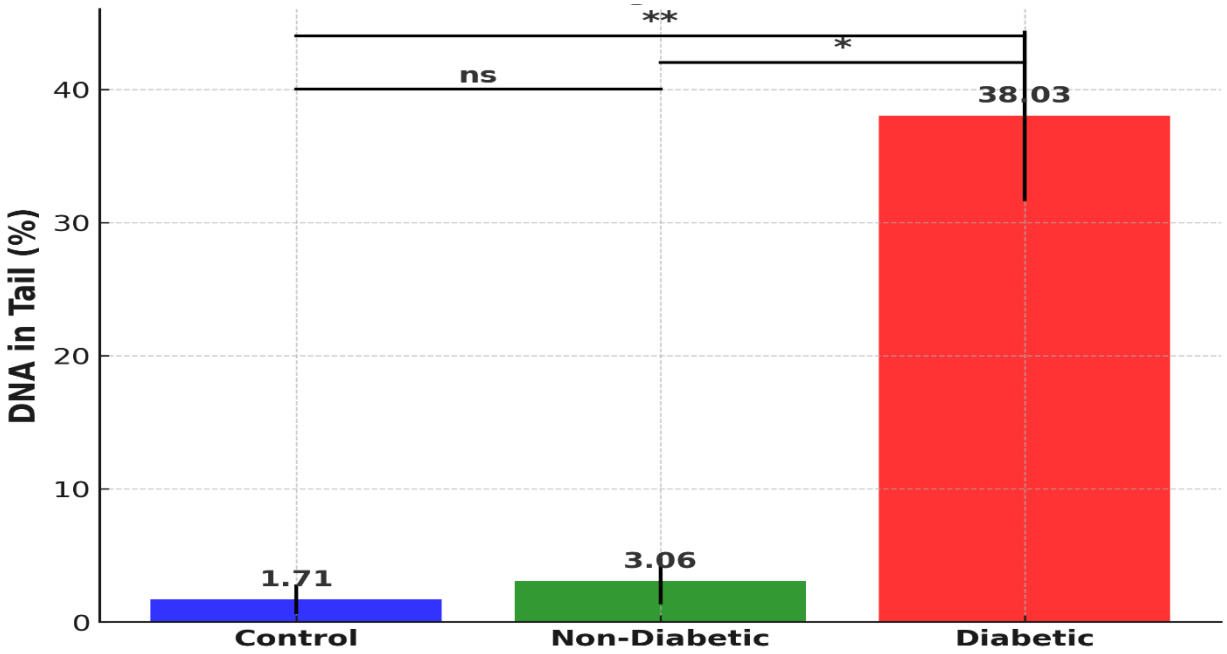

Figure 2 **. DNA Damage in Sars-Cov-2 Patients .**

"ns" means no statistical significance. "*" means moderate significance.

"**" means moderate significance.

4. DISCUSSION

Based on the data shown in Figures (1) and (2) and Table (1) , SARS-CoV-2 patients with diabetes were found to have the highest level of DNA damage in the tail, with an average damage of 38.03 ± 6.40, compared with non-diabetic COVID-19 patients (3.06 ± 1.73) and the control group (1.71 ± 1.12).

The p-value between the control group and the non-diabetic group (p = > 0.05), indicating no statistically significant

difference between them. The p-value between the control group and the diabetic group (p = < 0.001), indicating a strong statistical difference and a significant increase in DNA damage.

Studies suggest that SARS-CoV-2 directly causes DNA damage in immune cells, and is associated with increased oxidative stress and an exaggerated inflammatory response, leading to DNA fragmentation and tail damage when examining blood cells (Popova *et al.*, 2024) (Ameen *et al.*, 2021) .

The study found that DNA damage levels were higher in patients with chronic diseases, especially diabetes, with the average tail damage being 35.2 ± 5.8, which is close to the result obtained in our current study (38.03 ± 6.40) (Popova *et al.*, 2024).

A study published in Springer - Mammalian Genome found that diabetic patients with COVID-19 had a significant increase in DNA damage compared to non-diabetic patients, with an average of 36.5 ± 6.2, a result consistent with the result obtained in our current study (38.03 ± 6.40) (Moghbeli & Atashi, 2024).

Patients with poorly controlled diabetes have elevated levels of oxidative stress and systemic inflammation, leading to increased DNA breakage (Zhu *et al.*, 2024).

Increased levels of pro-inflammatory cytokines such as IL-6 and TNF-α in diabetic patients lead to greater oxidative stress, which reduces the ability of cells to repair DNA (Domingues, 2024) (Hadeedy *et al.*, 2019) .

5. CONCLUSIONS

Diabetic patients with COVID-19 had the highest DNA damage in the tail, suggesting that diabetes increases DNA damage.

The non-diabetic group did not show a statistically significant difference compared to the control group, suggesting that the effect of the virus alone may not be sufficient to cause significant DNA damage without other factors such as metabolic diseases.

The results of our study are consistent with previous studies, as the DNA damage in diabetic patients was in a similar range to what other studies have found

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