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Histopathological and Biochemical Changes Induced by *Aspergillus niger* and Treated with *Ganoderma lucidum* Filtrate and Sodium Bicarbonate in Laboratory Mice

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ABSTRACT

Objective: *The objective of this study is to isolate Aspergillus niger and assess its synthesizing capacity of fumonisin B2 (FB2).* ***Method:*** *Identification of the fungal isolate translated as a phenotypic to molecular identification in which A. niger was confirmed from C1 and C2 isolates. FB2 production was determined in High-Performance Liquid Chromatography (HPLC). To assess the toxic effects of FB2 in male albino mice a toxicity study was carried out with A. niger filtrate containing FB2 and protective efficacy was evaluated by using Ganoderma lucidum filtrate and sodium bicarbonate against FB(2) induced toxicity.* ***Results:*** *Histopathology examination showed extensive histolopathological changes in the liver and kidney tissue of A. niger filtrate treated mice. Fumonisin B2 enhances liver enzymes (GOT, GPT, ALP) level (191.33, 56.33, and 183.00) U/L respectively significantly comparing with the control group (104.67, 35.33, and72.00) U/L respectively The kidney function analyses also showed significantly increased serum level of the urea and creatinine, (51.67) mg/dL (1.03) mg/dL respectively compared with control group values were (35.33) mg/dL (0.67) mg/dL respectively.* ***Novelty:*** *This study demonstrated the protective role of G. lucidum filtrate and sodium bicarbonate in preventing organ damage caused by mycotoxins, suggesting synergism between these two therapies.*

INTRODUCTION

Mycotoxin fungi have a prominent global impact on food safety and public health due to their burden of contamination on food and agricultural products [1]. Some representative species of the genus Aspergillus are known as potential contaminants across most agricultural commodities when accompanied by adequate environmental conditions [2]. These fungi are associated with mycotoxins, low molecular weight secondary metabolites which often resist destruction from food processing operations [3]. Consumption of foodstuff contaminated with these fungal produced toxins results in serious health damage in humans and animals, from acute poisoning to chronic organ toxicity as well as immunosuppression [4].

As one of the most frequently isolated fungal contaminants, Aspergillus niger is a species which produces harmful mycotoxins like ochratoxin A (OTA) and some others that are considered toxicologically relevant species [5]. Such as, this fungus is known to be a common high level producer of the mycotoxin fumonisin B2 (FB2), which has been associated with many serious toxic effects in humans and animals [6]. Exposure of A. niger filtrate containing FB2 is associates with severe systemic toxicity, mostly in kidney and liver [7]. These mycotoxins cause extensive cellular injury in the biological system, resulting in hepatocyte fatty degeneration, inflammatory cell infiltration and focal

necrotic lesions of liver tissues [8]. This tissue damage is biochemically reflected by a dramatic increase in serum liver enzymes together with strongly increased kidney function biomarkers as urea and creatinine levels [9]. To overcome the hazards of organ damage by mycotoxins, novel ecofriendly alternatives including bioprotective solutions have been a subject to study while outdated chemical approaches could reduce their nutritional value [10]. Ganoderma lucidum extract with its potent antioxidants, anti-inflammatory- and hepatoprotective characteristics were efficient to detoxify fungal toxins and repair the impaired cellular architecture [11]. Sodium bicarbonate has been widely used as a safe chemical control agent capable of modifying environmental conditions that limited fungal growth and mycotoxin development [12]. However, the full potential of combining these biological and chemical approaches against *A. niger* toxicity requires comprehensive evaluation. Therefore, this study was designed to investigate their combined protective efficacy of *G. lucidum* filtrate and sodium bicarbonate against *A. niger*-induced organ damage in laboratory mice.

RESEARCH METHOD

Experimental Design and Animal Treatment

To evaluate the toxicological effects of *Aspergillus niger* filtrate and the protective potential of *Ganoderma lucidum* filtrate and sodium bicarbonate, 30 adult male albino BALB/c mice (*Mus musculus*) were randomly allocated to six experimental groups (five per group) and monitored over a 21-day period, following the experimental procedures described by [5]. The control group received only food and water. Based on preliminary toxicity testing, a 10% concentration of *A. niger* filtrate(0.5mL/animal) was orally administered to the animals, whereas *G. lucidum* filtrate and sodium bicarbonate were administered at a concentration of 10%. Treatments were administered every 48 hours for 21 consecutive days. The distribution of experimental animals among the treatment groups is presented in Table 1.

Experimental Treatment Groups and Dosage Regimens

All treatments were administered orally every 48 hours at a dosage of 0.5 mL per 30 g body weight throughout the experimental period. The experimental groups were treated as follows: the negative control group received exclusively water as the vehicle baseline, whereas mice in the intoxicated group received *Aspergillus niger* culture filtrate orally throughout the study period. To evaluate its protective effect, *G. lucidum* filtrate was administered orally as a single treatment. The combined treatment group received *A. niger* fungal filtrate, followed 24 hours later by administration of *G. lucidum* filtrate. To assess the combined toxicological and chemical intervention, the animals received an initial dose of *A. niger* filtrate, which was succeeded by a therapeutic dose of sodium bicarbonate after a 24hour post-exposure interval. Finally, the protective efficacy was investigated by exposing the mice to *A. niger* filtrate, followed by oral administration of sodium bicarbonate after the mice had been systematically dosed with *G. lucidum* filtrate for a 24-hour stabilization period. At the end of this experiment, the animals were anesthetized using chloroform and scarified. A midline abdominal incision was then

performed for the collection of liver and kidney tissues. Blood samples were collected for biochemical analyses, and liver and kidney tissues were excised for histopathological examination [5]. This in vivo study aimed to evaluate the protective and synergistic effects of the biological and chemical treatments against *A. niger* filtrate toxicity. The experimental focus was placed on tracking changes in body mass, evaluating weight gain rates, and performing detailed histological and biochemical analyses of liver and kidney tissues.

Table 1. Experimental Design and Treatment Regimens of Laboratory Mice

No.	Treatments	Treatment Description
1	Control	Mice received water only
2	*A. niger* Filtrate	Mice were orally administered *A. niger* filtrate (0.5 mL/30 g body weight) every 48 h
3	*G. lucidum* Filtrate	Mice were orally administered *G. lucidum* filtrate (0.5 mL/30 g BW) every 48 h
4	*A. niger* + *G. lucidum*	Mice received *A. niger* fungal filtrate, followed by *G. lucidum* filtrate after 24 h (0.5 mL/30 g BW).
5	*A. niger* +Sodium Bicarbonate	Mice were given a dose of *A. niger* fungal filtrate, and then they received a dose of sodium bicarbonate (0.5 mL/30 g BW) after a 24 h period.
6	*A. niger*+ *G. lucidum* Filtrate+ Sodium Bicarbonate	Mice received *A. niger* filtrate, followed by *G. lucidum* filtrate and sodium bicarbonate at 24 h intervals.

Biochemical Examinations

The serum levels of GPT, GOT, ALP, blood urea and creatinine were measured using R Krey biochemical analyzer (Japan). Each blood sample was obtained directly from the heart of 3 mice anaesthetized by isoflurane and immediately put into gel tubes for serum separation. Serum obtained after centrifugation was utilized for biochemical analyses. GOT, GPT, and ALP activities were measured as an indication of liver function, while the serum urea and creatinine concentrations were also determined to assess kidney function.

Histopathological Examination

After blood collection, liver and kidney were dissected out, washed with normal saline to remove residual blood and fixed in 10% neutral buffered formalin for 24 hours. The tissues were processed for histological analyses according to standard protocols, including dehydration through graded ethanol series, clearing in xylene, paraffin embedding and sectioning into 5~7 μm thick sections. Histopathological changes were evaluated in H&E-stained sections of tissues monitored by imaging under a light microscope.

Statistical Analysis

In all analyses, statistical comparisons between experimental groups were conducted. Data were represented as Mean ± SD (mean ± standard deviation). Differences among treatment groups were assessed by one-way ANOVA; significant differences between treatment means were determined using the Least Significant Difference (LSD) test. A P value of ≤ 0.05 was considered statistically significant, and a 95% confidence interval (CI) was applied [13].

RESULTS AND DISCUSSION

Effect on body weight

Body weight levels of mice were recorded during the experiment period, and results are summarized in Table 2. Similarly, the body weight values of the control were not affected throughout the whole experiment with an overall means value of 34.3 g while Aspergillus niger filtrate group had mean lowest body weight (31.46 g) after ten days and ((30.20 g before sacrifice). The decrease were also related to fumonisin B2 toxicity and other fungal metabolites which negatively affect growth performance. Chen et al. saw the same trend (nurse practitioners vs family physicians). (2022) reported a decrease in body weight after fumonisin exposure. On the other hand, Ganoderma lucidum filtrate-treated mice exhibited a body weight gain from 31.80 g before dosing to 33.00 g at ten days and slightly elevated with respect to body mass (MB) in before sacrifice (33.40 g). This improvement may be attributed to the antioxidant and immunomodulatory properties of *G. lucidum* [14]. The combined treatment of *A. niger, G. lucidum* and sodium bicarbonate showed the highest body weight values, reaching 36 g after 10 days and 36.80 g before sacrifice. This increase may be related to protective effects of *G. lucidum* and sodium bicarbonate against fungal toxicity. According to the LSD test (P ≤ 0.05), the combined treatment group exhibited the highest mean body weight (35.46 g), whereas the *A. niger* filtrate group showed the lowest mean body weight (31.47 g).

Table 2. Changes in body weight (g) of mice during the experimental period

No	Treatments	Mean body weight (g) ± Standard Deviation			
		Before dosing	After ten days	Before sacrifice	Overall mean ±SD
1	Control	32.20±0.84	34.40±1.54	35.40±0.89	34.3±1.11
2	*A. niger* filtrate	33.00±1.58	31.20±0.84	30.20±0.44	31.46±1.42
3	*G. lucidum* filtrate	31.80±0.83	33.00±0.71	33.40±1.52	32.73±0.83
4	*G. lucidum* + *A. niger*	33.20±1.31	34.20±1.31	35.60±1.14	34.33±1.21
5	*A. niger*+ sodium bicarbonate	32.40±0.89	33.80±0.84	35.00±1.58	33.73±1.31
6	*G. lucidum* + *A. niger* + sodium bicarbonate	33.60±1.54	36.00±0.71	36.80±1.09	35.46±1.66
Mean of time points ± SD		32.70± 1.15	33.77±1.68	34.40±2.41	

Treatments	Mean body weight (g) ± Standard Deviation		
LSD (P ≤ 0.05)	1.3	1.08	1.5

Examining the Levels of Liver Function Enzymes in Albino Mouse Blood

The effect of *Aspergillus niger* filtrate on liver function was evaluated by measuring the serum levels of GPT, GOT, and ALP enzymes, in experimental mice (Table 3). The highest values were recorded in the *A. niger* group, reaching 56.3 IU/L for GPT, 191.3 IU/L for GOT, and 183.0 IU/L for ALP, compared with 35.3, 104.7, and 72.0 IU/L, respectively, in the control group. These elevations were statistically significant and may be attributed to the toxic effects of fumonisin B2 and other fungal metabolites, which can induce hepatic tissue damage and increase membrane permeability leading to the leakage of intracellular enzymes into the bloodstream. The mice treated with *G. lucidum* filtrate showed significantly lower enzyme levels compared to the *A. niger* group, recording GPT, GOT, and ALP values of 54.7, 186.3, and 109.0 IU/L, respectively. These findings may indicate an improvement in liver function, variations in GPT, GOT, and ALP activities are commonly considered indicators of the physiological and functional status of the liver [9]. The combined treatment of *A. niger* and *G. lucidum* reduced the enzyme levels to 50.7 IU/L, for GPT, 187.7 IU/L for GOT, and 124.3 IU/L for ALP. A greater reduction in enzyme levels was observed in the group treated with *A. niger* and sodium bicarbonate, particularly for GOT, which decreased to 152.0 IU/L. The lowest enzyme levels among the treated groups were recorded in the combined treatment of *A. niger*, *G. lucidum*, and sodium bicarbonate, reaching 35.7 IU/L, for GPT, 110.7 IU/L for GOT, and 87.7IU/L for ALP, indicating a protective effect against fungal toxicity. Similar observations were reported by Zhang et al., (2024) who demonstrated the protective effect of *G. lucidum* on liver function in experimental animals. According to the LSD test (P ≤ 0.05), significant differences were observed among the treatment groups for GPT, GOT, and ALP activities [15].

Table 3. Effects of different treatments on serum liver enzymes activities in mice

Totals	Treatment	GPT (IU/L)	GOT (IU/L)	ALP (IU/L)
1	control	35.3±7.76	104.7±13.5	72.0±13.2
2	*A. niger* filtrate	56.3±5.68	191.3±18.2	183.0±11.3
3	*G. lucidum* filtrate	54.7±8.96	186.3±15.8	109.0±12.4
4	*A. niger* + *G. lucidum*	50.7 ± 4.04	187.7±18.4	124.3±17.4
5	*A. niger*+ sodium bicarbonate	44.3±5.51	152.0±11.1	122.6±7.51
6	*A. niger* +*G. lucidum* + sodium bicarbonate	35.7±6.02	110.7±10.5	87.7±2.72
Average times ± standard deviation		46.2±9.31	155.4±21.3	116.4±29.3
LSD value		10.9	28.2	13.7
Mean values		34 > IU/L	46 > IU/L	240 > IU/L

Examining the Levels of Creatinine and Urea in Albino Mouse Blood

The effect of *Aspergillus niger* filtrate on kidney function was evaluated by measuring serum urea and creatinine levels in experimental mice (Table 4). The highest values were recorded in the *A. niger* group, reaching 51.7 mg/dl for urea and 1.03 mg/dl for creatinine, compared with 35.3 mg/dl and 0.67 mg/dl, respectively, in the control group. These elevations may be attributed to the nephrotoxic effects of fumonisin B2 and other fungal metabolites, which can induce renal tissue damage, impair glomerular filtration and increase serum urea and creatinine levels [16]. Mice treated with *Ganoderma lucidum* filtrate showed lower urea and creatinine levels than the *A. niger* group, recording 42.9 mg/dl and 0.83 mg/dl, respectively. Similar reductions were observed in the groups treated with *A. niger* and *G. lucidum* (44.9 mg/dl and 0.8 mg/dl), and *A. niger* and sodium bicarbonate (39.7 mg/dl and 0.77 mg/dl), indicating partial improvement in the renal function. The lowest urea level among the treated group was recorded in the combined treatment of *A. niger, G. lucidum*, and sodium bicarbonate, reaching 36.2 mg/dl for urea and 0.77 mg/dl. The lowest creatinine value (0.77 mg/dl) was observed in both combined treatment and *A. niger* and sodium bicarbonate group. These findings suggest that the combined treatment alleviated the nephrotoxic effects of fungal metabolites and improved renal function, possibly by reducing oxidative stress and preserving kidney tissue integrity [17], [18]. According to the LSD test ($P \leq 0.05$), significant differences were observed among the treatment groups for serum urea and creatinine levels.

Table 4. Effects of different treatments on serum urea and creatinine levels in mice

Groups	Treatments	Blood urea mg/dl	Creatinine mg/dl
1	control	35.3±2.08	0.67±0.05
2	*A. niger*	51.7±6.93	1.03±0.15
3	*G. lucidum*	42.9±4.31	0.83±0.05
4	*A. niger*+ *G. lucidum*	44.9±2.06	0.8±0.01
5	*A. niger*+ sodium bicarbonate	39.7±2.81	0.77±0.05
6	*A. niger*+ *G. lucidum* + sodium bicarbonate	36.2±6.52	0.77±0.05
Mean ±SD	standard deviation	41.78±6.83	0.82±0.13
LSD	value	8.31	0.14
Mean	values	28.7-59.5 mg/dl	0.6-1.2mg/dl

Histological Sections of Liver

Histopathological examination of the liver section from control group revealed normal hepatic architecture, as regularly arranged hepatocytes with normal hepatic cords and intact central veins and sinusoids (Fig 1). Histopathological alteration induced by Aspergillus niger group (Fig 2) was also very severe, including fatty degeneration hepatocytes, disruption of hepatic cords, the central veins and sinusoids were marked congested with inflammatory cell infiltration and focal necrosis. Such changes might to be correlated with the toxic effects of fumonisin B2 produce, which resulted in oxidative stress and subsequent hepatocyte damage [19]. The impact on liver architecture at

optimum treatment period (Fig 3): Mice treated with this mushroom demonstrated great amelioration of liver structure, margination with normal central veins but mild congestive and few degenerative changes maybe due to antioxidant and anti-inflammatory activities of its bioactive constituents [20]. Compared with the toxin treated group, the A. niger and G. lucidum group (Fig 4) displayed less inflammatory infiltration and hepatocellular degeneration. Fig 5: The A. niger and sodium bicarbonate group also yielded a moderate histological improvement, although mild congestion and residual degenerative alterations were still seen ([21]. The greatest histopathological recovery was recorded in the combined treatment group *A. niger, G. lucidum*, and sodium bicarbonate (Fig 6), where liver tissue appeared nearly normal with regulary arranged hepatocytes, minimal congestion, and markedly reduced inflammatory cell infiltration, suggesting a synergistic protective effect against fumonisin-induced hepatotoxicity [15].

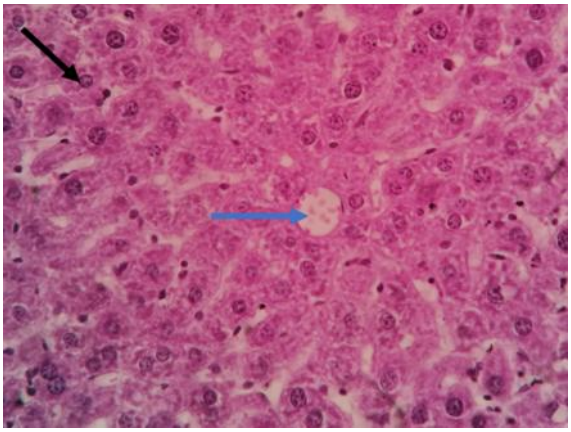

Figure 1. Histological section of the liver tissue from the control group showing normal hepatic architecture with regularly arranged hepatocytes (black arrow) surrounding the central vein (blue arrow), without evidence of cellular degeneration, necrosis, or inflammatory cell infiltration. H&E stain, ×400.

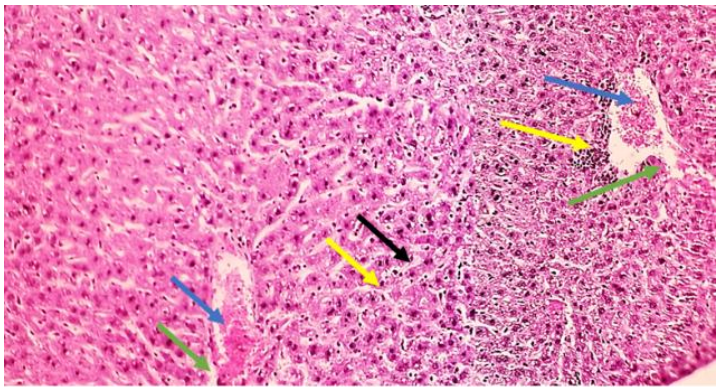

Figure 2. Histological section of the liver tissue from mice treated with *Aspergillus niger* filtrate showing fatty degeneration of hepatocytes (black arrow), congestion of the central vein and hepatic sinusoids (blue arrow), inflammatory cell infiltration around blood vessels (green arrow), and necrotic changes with pyknotic nuclei in hepatocytes (yellow arrow). H&E stain, ×400.

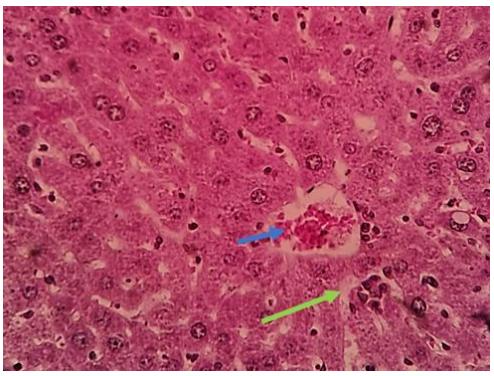

Figure 3. Histological section of the liver tissue from mice treated with *Ganoderma lucidum* filtrate showing improvement in hepatic architecture, with nearly normal hepatocytes (green arrow), mild vascular congestion (blue arrow), and only mild residual degenerative changes. H&E stain, ×400.

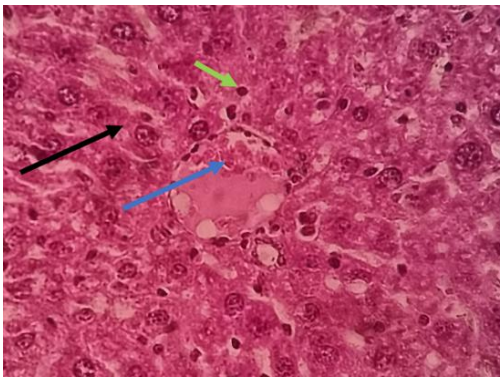

Figure 4. Histological section of the liver tissue from mice treated with *Aspergillus niger* and *Ganoderma lucidum* filtrate showing a marked improvement in hepatic architecture, with better organization of hepatic cords (black arrow), reduced cellular degeneration and inflammatory cell infiltration (green arrow), and only mild vascular congestion remaining (blue arrow). H&E stain, ×400.

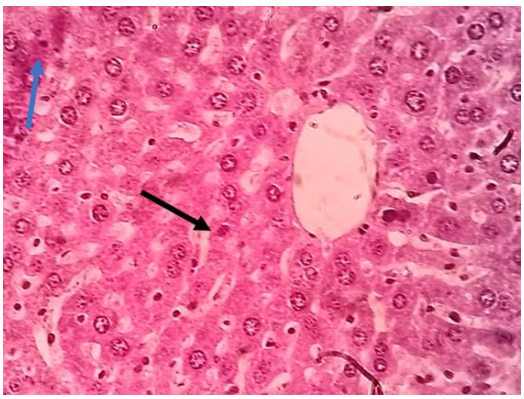

Figure 5. Histological section of the liver tissue from mice treated with *Aspergillus niger* filtrate and sodium bicarbonate showing moderate improvement in hepatic architecture, with better organization of hepatic cords (black arrow) and reduced degenerative changes, although mild vascular congestion (blue arrow) remained evident. H&E stain, ×400.

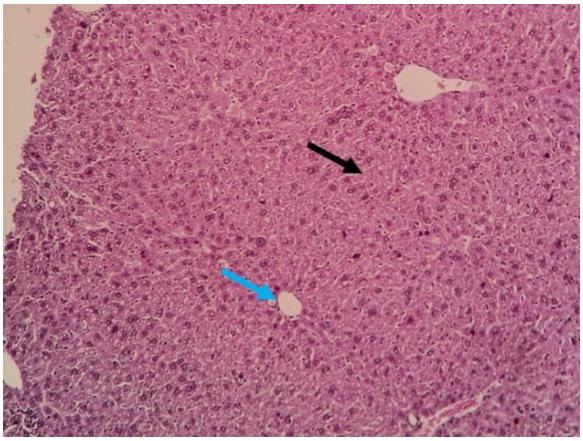

Figure 6. Histological section of the liver tissue from mice treated with *Aspergillus niger, Ganoderma lucidum* filtrate and bicarbonate, showing near complete restoration of normal hepatic architecture. The hepatic cords appear regularly arranged (black arrow), with a substantial reduction in congestion and inflammatory infiltration, while the central vein exhibits an almost normal appearance (blue arrow). H&E stain, ×400.

Histopathological Section of Kidney

Kidneys obtained from animals in the control group (Fig 7) showed a normal renal structure, well-preserved glomeruli and preserved renal tubules on histopathological sections. Histopathological examinations also revealed that there were no any significant changes in the renal sections from group I (Fig 8), however, severe histopathological changes such as tubular epithelial cell degeneration, vascular congestion, inflammatory cells infiltration and necrotic foci in renal tubules was observed in kidney sections isolated from Aspergillus niger group (Fig 8). Chronic lesions may be related to the nephrotoxic effects of fumonisins, which induce oxidative stress and cell damage in kidney tissues [17]. Sections of kidney tissues from Ganoderma lucidum filtrate (Fig 9) treated mice also showed a significant improvement of renal histology, with less degenerative changes and inflammatory infiltration than the untreated control group. In the same way that we obtained for the group A. niger and G. lucidum (Fig 10), also in relation to the granular changes, we noted a further restoration of renal architecture when compared with MCD. The A. niger and sodium bicarbonate group (Fig 11) showed moderate improvement, with less congestion and degenerative changes in the liver as previously reported ([22]. The greatest histopathological recovery was observed in the combined treatment group *A. niger, G. lucidum* and sodium bicarbonate (Fig 12), where kidney tissues appeared nearly normal with minimal pathological lesions. This protective effect may be related to the antioxidant and anti-inflammatory properties of *G. lucidum*, which contribute to preserving renal tissue integrity and reducing toxin-induced damage [23], [24].

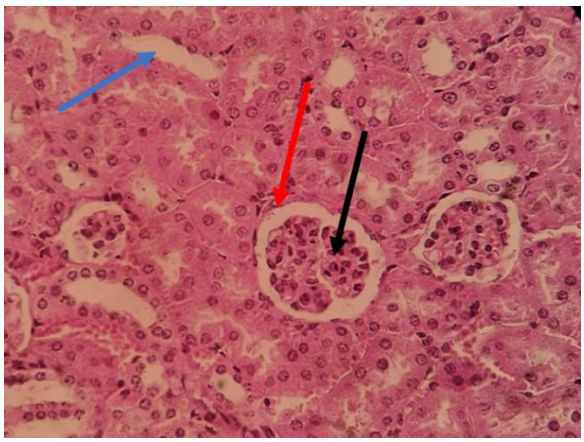

Figure 7. Histological section of the kidney tissue from the control group showing a normal structure of the glomeruli (black arrow) and urinary tubules (blue arrow), with an intact Bowman's capsule (red arrow) and no pathological changes or congestion in the blood vessels. H&E stain, ×400.

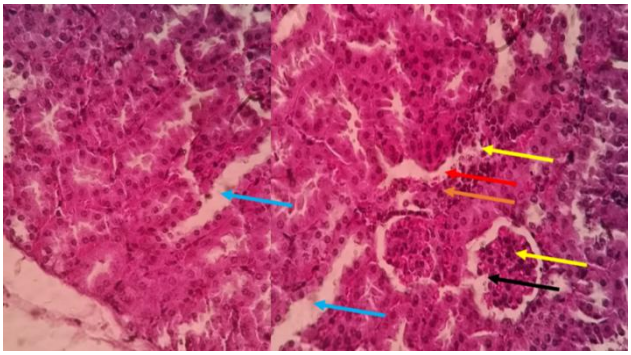

Figure 8. Histological sections of the kidney tissue from mice treated with *A. niger* filtrate showing degeneration and swelling of renal tubular epithelial cells (yellow arrows), with dilation of some renal tubules containing cellular debris (blue arrows), in addition to severe congestion of the glomeruli (black arrow), infiltration of inflammatory cells (orange arrow). H&E stain, ×400.

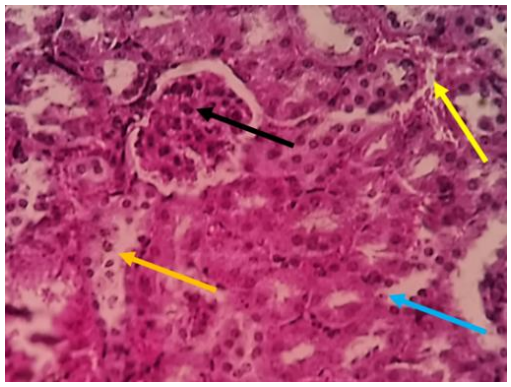

Figure 9. Histological sections of the kidney tissue from mice treated with *G. lucidum* filtrate showed a marked improvement in the histological structure, with most glomeruli (black arrow) and renal tubules appearing normal, with mild vascular congestion (blue arrow) and very minor degenerative changes in some renal tubules (yellow arrows). H&E stain, ×400.

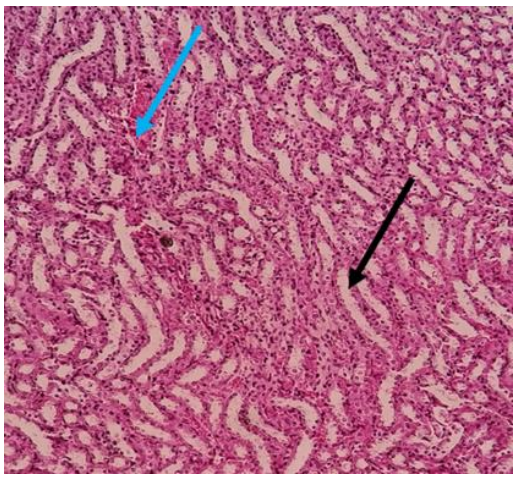

Figure 10. Histological section of the kidney tissue from mice treated with *A. niger* filtrate and *G. lucidum* filtrate showing improved renal histoarchitecture, near-normal renal tubules (black arrow), and mild vascular congestion (blue arrow). H&E stain, ×400.

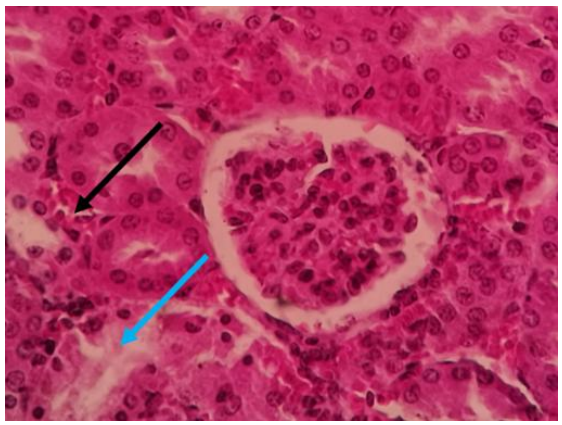

Figure 11. Histopathological sections of the kidney tissue from mice treated with *A. niger* filtrate and sodium bicarbonate showing mild vascular congestion (black arrow) and slight dilatation of some renal tubules with mild residual degenerative changes (blue arrow). H&E stain, ×400.

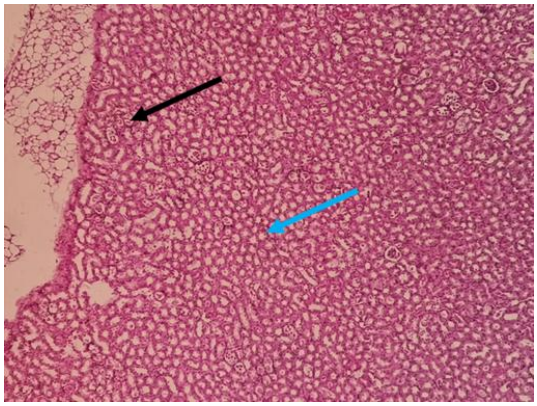

Figure 12. Histological section of the kidney tissue from mice treated with *A. niger* filtrate, *G. lucidum* filtrate and sodium bicarbonate showing largely intact renal tubules (black arrow) and near-normal renal histoarchitecture (blue arrow). H&E stain, ×400.

CONCLUSION

Fundamental Finding : Identification of the fungal isolate translated as a phenotypic to molecular identification in which *A. niger* was confirmed from C1 and C2 isolates. Histopathology examination showed extensive histolopathological changes in the liver and kidney tissue of *A. niger* filtrate treated mice. Fumonisin B2 enhances liver enzymes (GOT, GPT, ALP) level (191.33, 56.33, and 183.00) U/L respectively significantly comparing with the control group (104.67, 35.33, and 72.00) U/L respectively. **Implication :** This study demonstrated the protective role of *G. lucidum* filtrate and sodium bicarbonate in preventing organ damage caused by mycotoxins, suggesting synergism between these two therapies. **Limitation :** To evaluate the toxicological effects of *Aspergillus niger* filtrate and the protective potential of *Ganoderma lucidum* filtrate and sodium bicarbonate, 30 adult male albino BALB/c mice (*Mus musculus*) were randomly allocated to six experimental groups (five per group) and monitored over a 21-day period, following the experimental procedures described by Hussien and Saadon (2022). **Future Research :** However, the full potential of combining these biological and chemical approaches against *A. niger* toxicity requires comprehensive evaluation.

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