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Synergistic Biochemical Effects of Pesticide Exposure and Helicobacter pylori Infection in Egyptian Farmers

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ABSTRACT

Background: Pesticide exposure and Helicobacter pylori infection are two common environmental risk factors. Egyptian farmers often face the dual challenge of both. However, the synergistic effects of these two factors remain poorly understood. **Objective**: To investigate effects of pesticide exposure and Helicobacter pylori infection on biochemical parameters. Subjects and **Methods**: 86 farmers chronically exposed to pesticides along with 64 matched unexposed controls were enrolled, the two groups were subdivided based on Helicobacter pylori infection into 4 subgroups: group A (unexposed/uninfected, n=32), group B (unexposed/infected n=32), group C (exposed/uninfected, n=33), and group D (exposed/infected (n=53). Biochemical and oxidative markers were measured, as well as exposure and infection markers. Statistical analyses: The statistics were performed with the SPSS (version 27) program. **Results**: Malondialdehyde levels showed a significant step-by-step increase in groups B, C, and D compared to group A. Glutathione-s-transferase activity decreased in exposed groups (C and D) compared to non-exposed controls (A). The activities of Butyrylcholinesterase showed significant reduction in the exposed groups (C and D) compared to the non-exposed groups (A and B). Creatinine was significantly higher in the C and D exposed groups compared to the A group, and the level of calcium, magnesium, and iron was also gradually decreased from control to the exposed/infected group. However, prevalence of Helicobacter pylori and liver enzymes are comparable between groups. **Conclusion**: Pesticide exposure and Helicobacter pylori infection synergistically disturbed the studied biomarkers in Egyptian farmers, revealing amplified biochemical impacts from the combined exposures versus individually.

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INTRODUCTION

Agricultural workers worldwide face an array of occupational hazards that include both chemical and biological exposures [1]. Pesticides are agrochemicals widely used especially in developing countries for pest control and prevention [2]. However, there are several health problems associated with chronic occupational pesticide exposure [3]. Exposure to pesticides over extended periods, even in small quantities, holds the capacity to cause alterations in various biochemical factors [4].

The main occupational exposure routes are skin absorption during pesticide preparation, inhalation during spraying, and ingestion through contaminated water and food. various factors affect pesticide toxicity, including pesticide type, duration, and exposure frequency as well as personal protective equipment [5]. Occupational exposure to pesticides, even at low doses, can lead to changes in biochemical markers in the body [6], Pesticide use is a major contributor to mortality and morbidity in agricultural workers, due to both acute and chronic toxicities. Long-term exposure to low levels of pesticides can also lead to a variety of health problems, including metabolic dysfunction, carcinogenesis, immunological disorders, neurotoxicity, and reproductive effects [7].

Helicobacter pylori (H. pylori) is a gram-negative, microaerophilic, and spiral-shaped bacterium that infects the gastric mucosa of humans during early stages of life. It establishes colonies within the mucosal gel layer and leads to persistent inflammation in the stomach. H. pylori is a common bacterium that can cause chronic infections in humans, and it is found in people of all generations and in all parts of the world [8,9]. Helicobacter pylori infects more than half of the world's population, transmitted mainly through mouth-to-mouth and fecal-to-mouth contact, and can be diagnosed with either invasive tests or non-invasive tests such as stool Antigen [10,11].

Helicobacter pylori infection triggers inflammatory responses in the gastric epithelium, resulting in immune cell activation and release of proinflammatory cytokines. These immune cells can also generate reactive oxygen species (ROS) as part of the inflammatory process, leading to oxidative stress within the gastric tissue [12,13].

Since pesticide exposure and Helicobacter pylori infection are common environmental risk factors among Egyptian farmers, having a significant impact on human health, this study was conducted to investigate the individual and combined effects of pesticide and H. pylori infection on biochemical parameters. The study is expected to provide valuable knowledge into the synergistic effects and to identify potential biomarkers for early detection and monitoring of the health impacts of these factors.

MATERIAL AND METHODS

Study population: -

This cross-sectional study enrolled 150 participants who were divided into an unexposed control group (n=64) and an exposed group (n=86). The control and exposed groups were further stratified based on Helicobacter pylori infection status, into 4 subgroups: A) unexposed/uninfected (n=32), B) unexposed/infected (n=32), C) exposed/uninfected (n=33), and D) exposed/infected (n=53). Participants were matched across all subgroups for demographic and clinical characteristics including sex, age, smoking status, socioeconomic status, body mass index and nutritional status. Individuals with medical conditions or taking medications known to affect the biochemical parameters of interest were excluded.

Sample collection and preparation: Approximately 5 milliliters of venous blood were collected from each participant into sterile vacutainer plain tube, the blood samples were allowed to clot at room temperature, then centrifuged at 3000 rpm for 15 minutes to separate the serum. In addition to a random stool sample for Helicobacter pylori detection.

Laboratory measurements: -

Biochemical parameter determination: -

Liver profile including Alanine aminotransferase, Aspartate aminotransferase,

Total protein, Albumin, Alkaline phosphatase and Gamma-glutamyl transferase in addition to Creatinine, Magnesium, Calcium and Iron were analyzed using fully automated analyzer Cobas c311/501 (Roche diagnostics, Germany). Serum electrolytes, including sodium, potassium, ionized calcium, chloride, and blood pH, were measured b ion-selective electrode auto analyzer (Cornley-K-Lite 5, Meizhou, Cornley Hi-Tech Co Ltd, China).

Oxidative stress biomarker:

Glutathione S-transferase (GST) as an antioxidant enzyme and malondialdehyde (MDA) as a lipid peroxidation product and oxidative stress marker were determined colorimetrically, in accordance with previously reported methods [14,15], using a commercial kit (Biodiagnostic, Egypt) and a plate reader (BMG Labtech, FLU Ostar Omega, Germany). The results were expressed in IU/L.

Exposure biomarker: as a biomarker of organophosphate and carbamate pesticides exposure Butyrylcholinesterase (BChE) was measured colorimetrically using as a substrate butyrylthiocholine iodide. The measurements were performed on an ERMA AE-600N spectrophotometer (ERMA Inc., Tokyo, Japan) using assay kit from Centronic GmbH, Germany [16]. The results were expressed in IU/L.

Infection biomarker: Helicobacter pylori stool Antigen as an infection biomarker was detected using the H. Pylori Antigen Quick Test Cassette (Right Sign, China), which is a rapid immunochromatographic test for the qualitative detection of H. pylori antigen in stool.

Ethical approval:

Informed consent was obtained from all participants prior to enrollment. The study protocol was approved by the ethics review board of Faculty of Medicine at Zagazig University.

Statistical analysis: -

Results were reported in means ± SEM (Standard Error of Mean) for continuous variables. The value of $P < 0.05$ was used to indicate statistical significance. Post hoc testing was performed for inter group comparisons using the least Significant Differences (Duncan) test Differences between groups were determined by one–way analysis of variance (ANOVA). Prior to ANOVA, the Shapiro-Wilk test verified that the data were normally distributed and Levene's test confirmed homogeneity of variance among groups. P-values less than 0.05 were considered statistically significant.

RESULTS

In this comparative cross-sectional study, 86 farmers chronically exposed to mixed pesticides along with 64 matched healthy unexposed control were enrolled, each group were subdivided according to Helicobacter pylori infection. All participants were male, residing in the same area, and shared similar nutritional and lifestyle backgrounds. Table 1 demonstrates demographic characteristics of the study population and revealing comparability between the study groups with respect to age, BMI, smoking status, or prevalence of H. pylori infection, as all p-values exceeded 0.05. The unexposed group had an H. pylori infection rate of 50%, while the exposed group had a non-significantly higher infection rate of 61.6%. The average pesticide exposure in hours per week and the average years of work experience were comparable between the two exposed subgroups C and D. The farmers were exposed occupationally to different types of pesticides mainly organophosphates, carbamates, and pyrethroids as demonstrated in Table 2.

Histopathological results:

Table 3 shows the effects of pesticide exposure and H. pylori infection on markers of oxidative stress and cholinesterase activity in the study groups. MDA, a product of lipid peroxidation, was significantly increased in group B (11.60 ± 3.89 IU/L), group C (12.90 ± 4.51 IU/L), and group D (14.08 ± 4.50 IU/L) compared to the control group A (8.82 ± 2.04 IU/L) with p values $p=0.030$ for A vs. B, $p<0.000$ for A vs. C, $p<0.000$ for A vs. D, and $p=0.030$ for B vs. D. Similarly, GST activity, an indicator of antioxidant response, was significantly reduced in the group C (253±160 IU/L) and group D (245 ± 139 IU/L)

compared to controls A (410 ± 302 IU/L) with p values p=0.005 for B vs. D, p=0.001 for A vs. D). BChE, a measure of cholinesterase enzyme activity, showed a dramatic step-wise decrease from control groups A and B (5907 ± 5889, 5536 ± 869 IU/L) to exposed groups C and D (4318 ± 677, 4240 ± 563 IU/L), with highly significant reductions compared to controls (p<0.001).

Table 4 shows the effects of pesticide exposure and H. pylori infection on the biochemical profile of the study groups. Liver markers did not differ significantly between groups. Sodium, potassium, chloride and blood pH were comparable amongst the study groups. Creatinine levels were significantly higher in the exposed groups C and D versus the unexposed group A, with p-values of p=0.04 and p=0.001, respectively. Total calcium and ionized calcium levels showed a progressive decline from groups A to D, with group C and D exhibiting significantly lower levels than group A (p=0.003 and p=0.000 for total calcium; p=0.036 and p=0.001 for ionized calcium). Serum magnesium was also significantly reduced in groups C and D relative to group A (p=0.009 and p=0.001). Lastly, serum iron levels exhibited a step-wise decrease from groups A to D, with group B, C and D having significantly lower levels than group A (p=0.034 for B vs A; p=0.022 for C vs A; p=0.001 for D vs A).Total , ionized calcium, magnesium and iron levels showed a progressive decline from groups A to D, with group C and D exhibiting significantly lower levels than group A. Lastly, serum iron levels exhibited a step-wise decrease from groups A to D, with group B, C and D having significantly lower levels than group A (p=0.034 for B vs A; p=0.022 for C vs A; p=0.001 for D vs A).

DISCUSSION

Since pesticide exposure and Helicobacter pylori infection are common risk factors among agricultural workers in Egypt, the current study was conducted to investigate the individual and combined effects of pesticide exposure and Helicobacter pylori infection on biochemical parameters in Egyptian farmers. The observed elevation in malondialdehyde (MDA) levels in groups B, C, and D suggests that pesticide exposure and Helicobacter pylori infection induce oxidative stress in farmers. This is likely due to the generation of reactive oxygen species (ROS) from both sources, which can damage cellular components, leading to lipid peroxidation, as previously reported and consistent with the findings of other studies [17-22] Glutathione-s-transferase (GST) activity decreased significantly in exposed groups (C and D) supporting the role of oxidative stress in the observed biochemical changes. GST is an essential antioxidant enzyme that plays an important role in protecting the body from damage caused by xenobiotics and other toxic substances, A decrease in GST activity can make the body more susceptible to the harmful effects of these substances [23]. Previous studies documented decrement in GST in pesticides exposed workers [24-26] and H. pylori infected patients [27-29]. The dramatic declines observed in butyrylcholinesterase (BChE) activity are consistent with the known anticholinesterase toxicity of organophosphate and carbamate pesticides [30]. Since these pesticides target cholinesterase enzymes, BChE has been considered a useful biomarker for exposure [31]. Its reduction could lead to increased accumulation of pesticides in the body and potentiate their toxic effects. These findings were in line with several previous studies [32-34]. The significant increase in creatinine levels along with declines in calcium, magnesium, and iron levels observed in group B and even more prominently in group D, suggest pesticide exposure, especially when combined with Helicobacter pylori infection, may impair kidney function and disrupt mineral homeostasis. Several studies have reported renal impairment in workers exposed to pesticides [35-38], which may be due to induced oxidative stress [39]. Similarly, Pan et al. in 2019 considered H. pylori infection a risk factor for renal damage as well [40]. Medithi et al. (2022) recorded low levels of calcium and magnesium in pesticide-exposed individuals [41]. Previous studies have also reported a significant decrease in trace elements in H. pylori-infected patients,

attributing this to changes in elemental metabolism, malnutrition, absorption difficulties, and oxidative stress, all of which are possible effects of H. pylori infection [42-43].

CONCLUSION

The study found that Egyptian farmers face a "double burden" from occupational pesticide exposure and endemic H. pylori infection, as both factors individually, and more prominently synergistically, disturbed biomarkers of oxidative stress, antioxidant defense, kidney function, and mineral balance, leading to a range of health problems. So, more Integrated interventions were needed to improve farmer health, such as reducing pesticide exposures and providing access to testing and treatment for H. pylori infection. Biomarker monitoring is also recommended for early identification of farmers demonstrating biochemical effects. Finally, because H. pylori is so prevalent in Egypt, it should be considered when studying biochemical changes and oxidative stress status in other similar studies.

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Table 1: Demographic Characteristics of the Study Population

Group	Control (N=64)		Exposed (N=86)		P-value
Subgroup	Non infected A (n=32)	Infected B (n=32)	Non infected C (n=33)	Infected D (n=53)	
Age (years)	45.53 ± 8.31	43.64 ± 7.08	43.34 ± 7.59	40.56 ± 9.25	NS[1]
BMI (Kg/m2)	26.55 ± 2.58	26.83 ± 2.47	27.21 ± 2.68	27.33 ± 3.49	NS[1]
Smoking (n/%)	22 (31.3%)	15 (53.1%)	21 (36.4%)	29 (45.3%)	NS[1]
Exp. (hrs./week)	----------	----------	11.8 ± 4.6	12.2 ± 5.2	NS[2]
Exp. (years)	----------	----------	19.2 ± 5.2	19.9 ± 5.6	NS[2]
H. pylori (n%)	32(50%)		53(61.6%)		NS[3]

SD (standard deviation), BMI (body mass index), n (number), Exp. (exposure), hrs. (hours), [1] by ANOVA, [2] by independent sample t- test,[3] using chi-square tests.

Table 2: List of Frequently Used Pesticides Reported by Exposed Farmers

Chemical class	Use	Common name
Organophosphates	Insecticide	Prothiofos, Profenofos, Malathion, Diazinon, Dimethoate, Chlorpyriphos, Fenamiphos.
Carbamates	Insecticide	Carbosulfan, Methomyl
Pyrethroid	Acaricide	Abamectin
Pyrethroid	Insecticide	Lambda-cyhalothrin
Neonicotinoides	Insecticide	Acetamiprid
Dithiocarbamates	Fungicide	Mancozeb
Benzimidazole	Fungicide	Carbendazim
Dinitroanaline	Herbicide	Pendimethalin
Triazole	Fungicide	Penconazole
Pyrazole	Acaricide	Fenpyroximate
Thioureas	Fungicide	Thiophanate- methyl

Table 3. Oxidative And Exposure Markers.

Group	Control (N=64)		Exposed (N=86)		P- value
Subgroup	Non infected A (n=32)	Infected B (n=32)	Non infected A (n=33)	Infected B (n=53)	
MDA (IU/L)	8.8± 2.0	11.6 ± 3. 9	12.9 ± 4.5	14.1 ± 4.5	$0.030^{1,5}$,$0.000^{2,3}$
GST (IU/L)	410 ± 302	302± 127	253±160	245 ± 139	0.005^{2}, 0.001^{3}
BChE (IU/L)	5907 ± 589	5536 ± 869	4318 ± 677	4240 ± 563	$0.000^{2,3,4,5}$

Data tabulated as mean ± standard deviation, NS (non-significant), n (number), A (unexposed-uninfected), B (unexposed-infected), C (exposed-uninfected), D (exposed-infected), [1] (comparison between group A and B), [2] (comparison between group A and C), [3] (comparison between group A and D), [4] (comparison between group B and C), [5] (comparison between group B and D)

Table 4. Biochemical Parameters Evaluated in the Study Groups

Group	Control (n=64)		Exposed (n=86)		P- value
Subgroup	Non infected A (n=32)	Infected B (n=32)	Non infected A (n=33)	Infected B (n=53)	
ALT(IU/L)	22.5 ± 4.2	22.7 ± 4. 7	24.3 ± 6.1	25.3 ± 7.5	NS
AST(IU/L)	22.7 ± 3.3	24.1 ± 3.7	25.3 ± 6.6	26.3 ± 8.9	NS
Alb (g/dl)	4.3 ± 0.2	4.8 ± 2.8	4.2 ± 0.2	4.9 ± 5.1	NS
TP(g/dL)	7.5 ± 0.34	7.5 ± 0.3	7.5 ± 0.3	7.5 ± 0.3	NS
ALP(IU/L)	88.3 ± 21.3	83.1 ± 23.1	88.3 ± 25.2	96.1 ± 29.4	NS
GGT(IU/L)	20.2 ± 5.2	21.0 ± 5.8	20.6 ± 3.5	21.6 ± 5.7	NS
LDH(IU/L)	332± 56	336 ± 47	351 ± 51	360 ± 69	NS
Creat(mg/dL)	1.00 ± 0.08	1.04 ± 0.11	1.07 ± 0.13	1.09 ± 0.12	$<0.05^{2}$, $<0.01^{3}$
Na(mmol/l)	140.3 ± 3.3	135.8 ± 2.2	140.8 ± 3.4	140.2 ± 3.8	NS
K(mmol/l)	4.10 ± 0.26	4.21 ± 0.27	4.28 ± 0.39	4.09 ± 0.30	NS
p H	7.37 ± 0.03	7.39 ± 0.05	7.38 ± 0.05	7.39 ± 0.07	NS
Ca(mg/dl)	9.46 ± 0.23	9.19 ± 0.53	9.13 ± 0.45	8.93 ± 0.67	$<0.01^{2,3}$
Ca^{++}(mg/dl)	4.53 ± 0.23	4.34 ± 0.43	4.24 ± 0.42	4.14 ± 0.54	$<0.01^{2,3}$
Cl (mmol/l)	104.6 ± 2.3	105. 5 ± 2.2	104.8 ± 2.1	105.1 ± 2.3	NS
Mg(mg/dl)	2.20 ± 0.20	2.13 ± 0.21	2.04 ± 0.22	2.03 ± 0.20	$<0.01^{2,3}$
Iron(ug/dl)	84.3 ± 24.8	67.1 ± 25.8	66.3 ± 19.3	58.6 ± 27.7	$<0.05^{1,2}$ $<0.001^{3}$

Data represented as mean ± standard deviation, NS (non-significant), n (number), A (unexposed-uninfected), B (unexposed-infected), C (exposed-uninfected), D (exposed-infected), 1 (comparison between group A and B), 2 (comparison between group A and C), 3 (comparison between group A and D), 4 (comparison between group B and C) 5 (comparison between group B and D)