RESEARCH ARTICLE

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Evaluation of Inflammatory Biomarkers *Gal-1* and *IL-1β* and Liver Enzyme Activities in Gastric Cancer Patients Pre- and Post-Chemotherapy: A Case-Control Study

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Abstract

Objective: The objective of the present study was to measure the serum levels of two inflammatory biomarkers, galectin-1 (*Gal-1*) and interleukin-1 beta (*IL-1β*), as well as liver enzyme activities, in gastric cancer (GC) patients before and after chemotherapy, and to compare these findings with healthy controls. **Methods:** The study included 75 gastric cancer patients and 50 controls. Serum levels of *IL-1β* and *Gal-1* were measured using the ELISA method, and liver enzyme activity was assessed using the kinetic chromogenic method with commercial kits. **Results:** Baseline levels of *Gal-1* and *IL-1β* were significantly higher in patients compared to controls (38.1 ng/ml vs 16.1 ng/ml; p <0.001 for *Gal-1*, 86.3 pg/mL vs 18.8 pg/mL; p <0,001 for *IL-1β*). And also Baseline levels of ALT, AST and ALP was significantly higher in the patient group pages Following chemotherapy, levels of *IL-1β* significantly decreased to 67.9 pg/ml (P < 0.001) and *Gal-1* showed a modest decrease to 37.2 ng/ml (not significant, P = 0.06). Although reduced by about 10%, both biomarkers remained significantly higher compared to controls (P < 0.001). In contrast, the activities of liver enzymes significantly increased after chemotherapy. **Conclusion:** Both *Gal-1* and *IL-1β* play pivotal roles in causing inflammation during the development of GC, and their expression may represent an integrated systemic inflammatory process in GC patients regardless of gastrectomy as well as chemotherapy. In addition, the combination chemotherapy in treatment of GC led to hepatotoxicity as suggested by sera hepatic enzyme activities during and following therapy.

Keywords: Gastric cancer- *Gal-1*- *IL-1β*- liver enzymes- chemotherapy

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Introduction

Gastric cancer (GC) remains the fifth frequent common cancer in incidence and mortality around the world, affecting 4.9% of total cases and 6.8% of all cancer-related deaths globally. The established high-risk factors include Helicobacter pylori infection, specific dietary habits and smoking, a positive correlation with obesity and older age as inherited genetic predispositions [1]. In Iraq, GC accounted for over 1,200 new cases and more than 1,000 deaths in 2022 [2]. Inflammation is a critical promoter of cancer progress since it operates the generation of tumor microenvironment (TME) with inflammatory elements like vasculature endothelial cells, fibroblasts, and immune cell. This environment presents high levels of immune checkpoints like programmed death receptor-1 (PD-1) ligand 1 (PD-L1), which inhibit anti-tumoral immune responses and also pro-inflammatory secretions as, chemokines, cytokines and growth factors [3]. Interleukin-1β (*IL-1β*) might have an effect on carcinogenesis although the current evidence suggested that *IL-1β* was associated with cancer, especially solid tumor and in particular with GC [4,5]. By binding to its receptors IL1R1 and IL1RAcP on both tumor and immune cells, this cytokine activates a range of transcription factors including NF-κB and AP-1 which in turn induces multiple inflammatory genes that foster tumor cell invasion, proliferation and angiogenesis [6]. Furthermore, *IL-1β* expression is related to tumor staging in breast cancer and esophageal carcinoma [7, 8].

They are able to linkage between intracellular organelles and from inside the nucleus or cytoplasm to the extracellular space that the presence of galectins, which refers to a family of galactoside-binding lectins found in various tissues that possibly affect immune functions through by involving microenvironmental cues [9]. Diabetes and pancreatitis patient commonly exhibit elevated peritoneal *Gal-1*, which counter-modulates *Gal-1* as enhancement of the major membrane proteins CD45 (FIG. 2b) and CD43 on T cells. This interaction leads to TAM-mediated T cell apoptosis and -migration, thereby inhibiting the local tumor-specific immune response [10, 11]. *Gal-1* also contributes to the creation of an immunosuppressive microenvironment by activating regulatory T cells and cancer-associated fibroblasts, which

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enhances immune evasion by tumor cells [12, 13].

Serum levels of *Gal-1* have been positively correlated with histopathological features in several cancers, including ovarian cancer and GC [14, 15]. However, only a few studies have examined changes in *Gal-1* expression before and after chemotherapy. For example, a study by Zhu et al. [16], reported a significant decrease in *Gal-1* expression in patients with squamous cervical cancer following chemotherapy. Conversely, Funkhouser et al. found that serum concentrations of *Gal-1* increased after chemotherapy in patients with breast cancer [17].

Chemotherapy has made significant progress in lowering the risk of GC recurrence. The first-line treatment typically involves a combination of fluoropyrimidine and platinum-based drugs [18]. The mechanism of action of cytotoxic chemotherapy involves interfering with nucleic acid synthesis and cell division. As a result, liver toxicity is a common side effect of anti-tumor chemotherapy, often leading to elevated liver enzyme levels [19, 20].

Acknowledging the pro-inflammatory significance of *IL-1β* and *Gal-1* in GC, our study aims to evaluate and compare the serum levels of *IL-1β*, *Gal-1*, and liver enzymes in GC patients versus healthy individuals.

Materials and Methods

Patients with GC from Imam Al-Hussein Medical City in Karbala, Iraq, were enrolled in the study, along with an age- and sex-matched healthy control group. Key patient characteristics including age, sex, histopathological findings, and chemotherapy details were obtained from medical records. Patients with metastases, liver disease, sepsis, autoimmune disorders, or a history of alcohol abuse were excluded from the study.

Blood samples were collected at baseline from both study groups and from post-chemotherapy patients. Samples were drawn into plain tubes to isolate serum, which was then used to measure the concentrations of inflammatory markers (*IL-1β* and *Gal-1*) and liver enzymes, including alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP).

Serum levels of *IL-1β* and *Gal-1* were measured using the ELISA technique with commercial kits from ZellBio®, and readings were taken using the Biotek® Model 1800 ELISA reader. Liver enzyme activity was measured using the kinetic colorimetric method with commercial kits from BioSystems®, and analyzed using** a Mindray BS-380 clinical chemistry analyzer.

Ethic approval

The study was approved by the Human Ethics Committee of Imam Al-Hussein Medical City in Karbala, Iraq. All participants were informed about the study and gave written informed consent before enrollment. Confidentiality of patient information was strictly ensured throughout the research.

Statistical analysis

Descriptive statistics (mean and standard deviation) were used to summarize age, sex, serum levels of *Gal-1*, *IL-1β*, and liver enzyme levels. Comparisons between patients and controls were conducted using the Chi-square test for categorical variables and the independent Student's t-test for continuous variables. Changes in serum levels of *Gal-1*, *IL-1β*, and liver enzymes from baseline to post-chemotherapy in the patient group were analyzed using the paired Student's t-test. All data analyses were performed using IBM SPSS Statistics (version 24) for Windows.

Results

Our case-control study included 75 patients with GC and 50 healthy controls, with no significant differences in age or sex between the groups (Table 1). All patients underwent either total or partial gastrectomy followed by chemotherapy regimens consisting of a combination of 5-fluorouracil (5-FU), oxaliplatin, and irinotecan.

Table 1. Main Characteristic Data

	Patients	Controls	P. value
Number	75	50	
Age (mean ± SD)	54.28 ± 12	56.12 ± 11.6	0.39*
Sex			
Female	20 (26.7%)	15 (30%)	0.68**
Male	55 (73.3%)	35 (70%)	

Statistical analysis was done using t-test and Chi-square test; P-values were 0.39 for age and 0.68 for sex.

Table 2. Histopathological Data of GC Patients

Charecterestic	
TNM Staging	
I	2 (2.7%)
II	39 (52%)
III	34 (45.3%)
Grade	
I	33 (44.0%)
II	28 (37.3%)
III	14 (18.7%)
T	
T2	15 (20.0%)
T3	37 (49.3%)
T4	23 (30.7%)
N	
N0	11 (14.7%)
N1	42 (56%)
N2	15 (20%)
N3	7 (9.3%)
Lauren classification	
Intestinal	43 (57.3%)
Diffuse	23 (30.7%)
Mixed	9 (12.0%)

T, tumor invasion; N, regional lymph node metastasis. Statistical analysis for categorical variables like TNM staging, tumor grade, T and N classification, and Lauren classification was performed using the Chi-square test. Percentages were reported for each category.

Table 3. Differences of *IL-1β* and *Gal-1* Levels within Patient Group (Pre- vs Post-Chemotherapy) and between Groups (Patient Group vs Control Group).

Marker	Patient group		Within-patient Δ	P. value (within)**	Control group	Between-group Δ	P. value (between)*
	Pre (Mean ± SD)	Post (Mean ± SD)			Mean ± SD		
IL-1β (pg/ml)	86.3 ± 19.5	67.9 ± 20.4	-18.3	< 0.001	18.83 ± 3.00	67.4 (Pre)	< 0.001
						49.11 (Post)	< 0.001
Gal-1 (ng/ml)	38.1 ± 10.4	37.2 ± 8.6	-0.89	0.063	16.13 ± 2.45	21.9 (Pre)	< 0.001
						21.10 (Post)	< 0.001

**, paired sample t-test; *, independent sample t-test; Δ, mean difference (within patient or between group); pre, pre-chemotherapy; post, post-chemotherapy; *Gal-1*, galectin-1; *IL-1β,* interleukin-1beta

Histopathological data are shown in Table 2.

Comparison of study variables between pre-chemotherapy and controls

At baseline, serum levels of *Gal-1* and *IL-1β* in patients were significantly higher 38.1 ng/ml and 86.3 pg/ml, respectively compared with controls, who had levels of 16.1 ng/ml and 18.8 pg/ml (P < 0.001) (Table 3). Similarly, serum levels of ALT, AST, and ALP were significantly elevated in the patient group (73.9 IU/ml, 106.4 IU/ml, and 128.6 IU/ml, respectively) compared with the control subjects (41.4 IU/ml, 55.4 IU/ml, and 95.3 IU/ml, respectively) (P < 0.001) (Table 4).

Comparison of study variables post-chemotherapy with their baseline levels

Post-chemotherapy, the serum level of *IL-1β* (67.9 pg/ml) was significantly lower than the baseline level (86.3 pg/ml) (P < 0.001). In contrast, *Gal-1* levels showed a slight, non-significant decrease from 38.1 ng/ml at baseline to 37.2 ng/ml post-chemotherapy (P = 0.063) (Table 3). Additionally, serum levels of ALT, AST, and ALP increased slightly after chemotherapy (74.7 IU/ml, 108.1 IU/ml, and 131.5 IU/ml, respectively) compared to baseline levels (73.9 IU/ml, 106.4 IU/ml, and 128.6 IU/ml, respectively), with these changes reaching statistical significance (P < 0.001) (Table 4).

Comparison of Gal-1 and IL-1β levels between post-chemotherapy and controls

Serum levels of *Gal-1* and *IL-1β* in post-chemotherapy patients (37.2 ng/ml and 67.9 pg/ml, respectively) were still significantly higher than those in the control group (16.1 ng/ml and 18.8 pg/ml, respectively) (P < 0.001) (Table 3).

Discussion

Inflammation is a key driver in the progression of gastric adenocarcinoma, with chronic inflammation caused by Helicobacter pylori induced gastritis widely recognized as a major contributing factor in its initiation [21]. Studies have shown that Helicobacter pylori colonization is associated with an increased expression of *Gal-1* and *IL-1β* in gastric tissue [22, 23]. Moreover, several studies have reported that the expression of *Gal-1* and *IL-1β* is significantly higher in gastric cancer (GC) tissue compared to normal gastric tissue [24, 25], these findings will likely account for the elevated serum levels of them in GC patients. We confirmed that both *Gal-1* and *IL-1β* levels in sera of GC patients 38.1 ng/ml, 86.3 pg/ml, respectively were elevated significantly compared with control group (16.1 ng/ml,18.8 pg/ml; P <0.001). Both of these observations resonate vis-à-vis the Blair et al. An example of this can be found in Vila-Perelló [26], that reported increased serum levels of *Gal-1* in breast and lung cancer patients compared to healthy controls. Similarly, Bosch et al. [27], demonstrated that serum GaI-1 in patients with pancreatic cancer was significantly higher than those of healthy controls. Furthermore, Deans et al. In addition, up-regulation of *IL-1β* both at mRNA and protein levels is described in patients with gastroesophageal cancer [28]. After completion of chemotherapy, at the time of serum collection, the levels of pro-inflammatory cytokine *IL-1β* declined from 86.3 pg/ml (baseline) to 67.9 pg/ml whereas that of *Gal-1* exhibited a sub effective decrease from 38.1 ng/ml (baseline) to 37.2 ng/ml. These observations are supported by Masoodi et al. Ovarian cancer patients showed reduced serum *Gal-1* levels upon chemotherapy in a report by Rubinstein et al. [29]. Although the ir-S-CRP level decreased after

Table 4. Differences of Liver Enzymes Activities within Patient Group (Pre- vs Post-Chemotherapy) and between Groups (Patient Group vs Control Group).

Marker	Patient group		Within-patient Δ	P. value (within)**	Control group Mean ± SD	Between-group Δ	P. value (between)*
	Pre (Mean ± SD)	Post (Mean ± SD)					
ALT (IU/ml)	73.89 ± 18.19	74.72 ± 18.14	0.84	< 0.001	41.44 ± 11.05	32.45 (Pre)	< 0.001
AST (IU/ml)	106.36 ± 8.99	108.14 ± 9.23	1.78	0.002	55.4 ± 5.32	50.96 (Pre)	< 0.001
ALP (IU/ml)	128.63 ± 16.56	131.51 ± 17.08	2.88	< 0.001	95.34 ± 19.54	33.29 (Pre)	< 0.001

**, paired sample t-test; *, independent sample t-test; Δ, mean difference (within patient or between group); pre, pre-chemotherapy; post, post-chemotherapy; ALT, alanine aminotransferase; AST, aspartate aminotransferase; ALP, alkaline phosphatase.

chemotherapy (37.2 ng/ml for *Gal-1* and 67.9 pg/ml for *IL-1β*), serum levels of these inflammatory markers were still elevated when compared with control group values (*Gal-1* = 16.1 ng/ml, P <0.001; *IL-1β* sat= 18.8 pg.ml, P <0.001). These results suggest that serum levels of *Gal-1* and *IL-1β* could represent systemic inflammatory soluble biomarkers in GC [29].

At baseline, the activities of liver enzymes in patient group were beyond normal limits (73.9 IU/L for ALT, 106.4 IU/L for AST and 128.6 for ALP) (P <0.001), suggesting some hepatic complications related to gastrectomy [30]. There were the elevated liver enzyme activities after chemotherapy (ALT 74.7 IU/L, AST 108.1 IU/L and ALP131.5 IU/L). Similarly, hepatic steatosis has been described with irinotecan and 5-FU in the literature, as well as oxaliplatin-induced hepatic sinusoidal injury in other studies. Furthermore, it is well established that both 5-FU and oxaliplatin induce oxidative stress, which contributes to hepatic steatosis development. These results support the notion that this chemotherapy combination increases the risk of hepatotoxicity [31, 32].

In conclusion, *Gal-1* and *IL-1β* play a significant role in inflammation during the progression of gastric cancer (GC), with their serum levels reflecting the overall systemic inflammatory status in these patients, even after gastrectomy and chemotherapy. Moreover, the combination chemotherapy used in GC treatment increases the risk of hepatotoxicity, as evidenced by persistently elevated liver enzyme activities following treatment.

Author Contribution Statement

Muslim Mohammed: Supervision, Conceptualization, Methodology, Data curation, Writing – original draft, Validation, Project administration, Funding acquisition, Alaa khalaf, Noor R: Methodology, Software, Writing – original draft, Fadhil Abb: Investigation, Resources..

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If any scientific Body approved it/ if it is part of an approved student thesis

This study is not part of a student thesis or academic dissertation and has not been submitted to any scientific body for prior approval. It is an independent clinical research conducted by the authors.

How the ethical issue was handled (name the ethical committee that approved the research)

The ethical aspects of this research were reviewed and approved by the Ethical Committee of Imam Al-Hussein Medical City in Karbala, Iraq. All participants provided informed consent prior to enrollment, and the study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Was the study registered in any registration dataset (for clinical trials, guidelines, meta-analysis)

No, this study was not registered in any clinical trial registry or international database, as it was an observational study and not a clinical trial, guideline, or meta-analysis.

Conflict of interest

Authors declared that they have no conflict of interest.

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