The Research of Medical Science Review
ISSN: 3007-1208 & 3007-1216 Volume 3, Issue 11, 2025

"THE IMPACT OF DIETARY PATTERNS AND AWARENESS OF ADDITIVE-RELATED CANCER RISKS ON STOMACH CANCER PREVALENCE: A COMPREHENSIVE STUDY IN QUETTA'S URBAN POPULATION"

Muhammad Younas[1], Muhammad Sharif Hasni[*2], Amir Rasool[3], Khalil Ahmed[4], Sohail Ahmed[5], Gohar Shah[6]

*[1.*2,3,4,5,6] Institute of Biochemistry, University of Balochistan (UOB), Quetta, 87300, Pakistan.*

[1]brahaimyounas@gmail.com, [*2]sharifhasni@gmail.com, [3]rasool.amir@gmail.com, [4]drkhalilahmedbmc@gmail.com, [5]sohailbaloch455@gmail.com, [6]gohars763@gmail.com

DOI: https://doi.org/10.5281/zenodo.17746284

Keywords
Gastric Cancer, Dietary Patterns, Processed Foods, Food Additives, Nitrates and Nitrites, Helicobacter pylori Infection, Public Awareness, Lifestyle Risk Factors, Quetta Population, Cancer Prevention

Article History
Received: 01 October 2025
Accepted: 16 November 2025
Published: 26 November 2025

Corresponding Author: *
Muhammad Sharif Hasni

Abstract

Gastric cancer continues to be a significant global health problem and in countries like Pakistan, a developing country, dietary shifts and low public awareness bear the major brunt of this disease. This cross-sectional case-control study was designed to determine the association of dietary patterns, processed food consumption and awareness regarding food additives in gastric cancer among adult in urban Quetta. In all, 1,092 subjects (546 clinically diagnosed cases of gastric cancer and an equal number of matched controls) were enrolled from several referral hospitals in the area. We collected basic data using a structured questionnaire together with clinical verification and laboratory to assess dietary behavior, knowledge of food additives, as well we biological indicators of disease. The marked association of high intake of processed foods, lack of awareness about risks from food additives, smoking and physical inactivity were strongly observed for H. pylori infection and also gastric cancer prevalence. Laboratory test findings showed obvious abnormalities among case subjects, many of whom had anemia, raised transaminases and deteriorated renal function, as well as elevated tumor markers. Adenocarcinoma and especially the intestinal type was predominantly diagnosed on endoscopy and histopathological examination. The diet and lifestyle habits along with poor knowledge about carcinogenic food additives are found as major risk factors leading to gastric cancer in Quetta. It underscores the urgency for targeted public health interventions, more stringent legislation on food additives and improved early screening to limit disease burden in the region.

INTRODUCTION

As well as high incidence, gastric cancer is one of the most fatal malignancies in the world and still holds a high position on mortality rates in spite of advances in diagnostic and treatment approaches (1). An estimated 1.7 million new cases are reported annually around the world with a disproportionately high burden in low-income countries, where environmental exposures and dietary patterns play an important role (2). Its diagnosis is frequently late, leading to extremely low five-year survival rates of less than15% in most geographies (3). Multiple genetic, lifestyle and environmental factors are implicated in the pathogenesis of gastric cancer, but dietary factors have been identified as one of the most modifiable determinants of risk (4).

Notably, many studies have demonstrated that a Westernized diet with processed meats, refined carbohydrates, sugar-rich beverages and

high-salt foods is significantly correlated with the increased risk of gastric cancer (5). On the other hand, adopting healthy dietary patterns, like Mediterranean diet, DASH diet or plant-based prudent diets are protective as they consist of anti-inflammatory and antioxidant rich fruits, vegetables, whole grains and fish (6). These results indicate that whole dietary patterns rather than individual foods are associated with gastric cancer risk.

Another disturbing element of the diet in recent times has been the introduction of chemicals into our food, such as nitrates, nitrites and preservatives in ham or bacon products; emulsifiers (keeping fats liquid) and colorants used to disguise poor-quality produce. These chemicals found in many processed and packaged foods have been associated with cancer-related pathways through numerous processes including the formation of nitrosamine, disruption of gut microbiota homeostasis and chronic low-grade inflammation (7). For example, nitrite in cured meats may form N-nitroso compounds under the acidic conditions of the stomach which are known human carcinogens (8). In spite of this evidence, knowledge about food additive–caused cancer risks are still widely unappreciated by the general public in many developing countries (9).

Gastric carcinoma still remains an important public health problem in South Asian countries, specifically Pakistan. The distribution of infection at the regional level is variable, and Balochistan demonstrates among the highest reported prevalence rates in the country (10). From Quetta it has been reported that the incidence of gastric cancer is on the increase during last two decades (11). This rise is taking place against a background of accumulating urbanization and substitution of traditional diets with processed, additive-rich foods. While H. pylori infection is the major risks in this region, shifting dietary habit and thy of additive associated with carcinogenesis are additional risk factor in increasing burden (12).

According to Ilic and Ilic (2022), that gastric cancer is still one of the deadliest cancers and accounts for more than 1 million new cases each year, with an unequal death rate in low-income countries (13). The authors note that incidence rates differ widely between areas, due to social level of development, life-style factors, and dietary exposure. Maddineni et al. (2022) "Helicobacter pylori is the most powerful recognized risk factor for GC, leading to a chronic inflammatory environment (chronic gastritis and atrophic gastritis), with the development of the Correa cascade that ultimately results in malignancy. Their findings underscore that H. pylori infection synergizes with modifiable dietary factors, such as high salt consumption and processed food eating, thereby greatly enhancing cancer risk in susceptible individuals (14).

According to Du et al. (2020) also claimed that adherence to the Mediterranean diet has a critically preventive role in terms of gastric cancer incidence because it contains abundant quantities of polyphenols, monounsaturated fats and anti-inflammatory compounds (15). In a meta-analysis, they found that Mediterranean dietary habits are associated with decreased gastric lesions, decreased inflammation and improved metabolic profiles all of which protect against stomach cancer. Daniyal et al. (2015) observed an increased number of cases of gastric cancer in Pakistan especially in the desert area such as Balochistan, which highlights the presence of environmental exposure, poverty and a lack of availability to health care that lead to arrival at late stages (16). Their study highlighted the urgent requirement for local level investigations on dietary risk factors and food processing habits as well as community-level awareness of cancerousness of food additives. Ryu et al. (2020) reported that public knowledge about food additives and related health risks is very low, particularly among young consumers who consume large quantities of industrially processed foods (17).

Research Methodolog

Study Design

The analytical cross-sectional case-control study was conducted to explore the relationship of dietary patterns, intake of processed food and knowledge about food additives with gastric cancer in adult population of Quetta. The study was performed between January 2025 and September 2025, which allowed comparison of

confirmed gastric cancer patients with body mass index (BMI)-matched controls, yielding a cost-effective framework in which to examine prior dietary exposures and associated risk factors.

Study Area

The study was conducted in Quetta, the provincial capital of Balochistan, Pakistan. Data was collected from the two main cancer diagnostic and treatment facility of Quetta, Pakistan i.e., CENAR Cancer Hospital and Bolan Medical Complex Hospital (BMCH). These hospitals were selected as they take referrals from all over the province, thus obtaining a heterogeneous and representative sample. Their comprehensive oncology units, endoscopy rooms and histopathology laboratories enabled accurate data confirmation and collection.

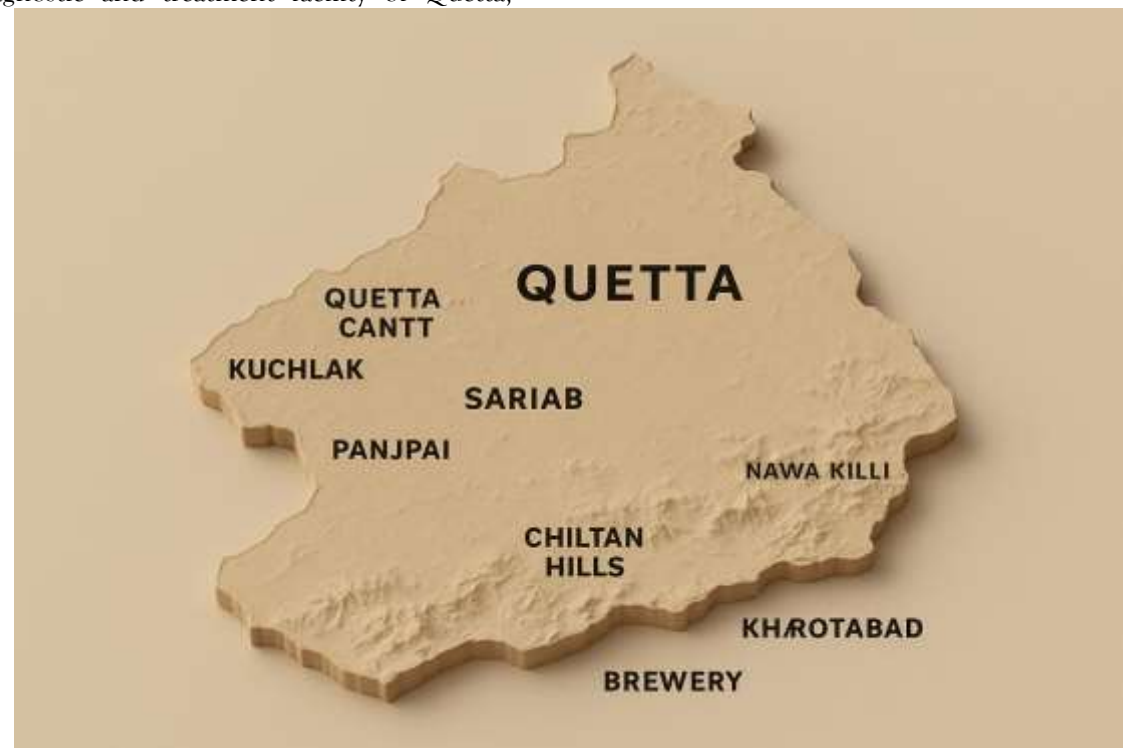

Figure 1: 3D-Map of study area

Study Population

A total of 1092 participants were recruited consisting of adults between the age range of 25-65 years NAFLD patients and control individuals and divided according to three age groups (251⁄434; 35ï469; 50ï465 years). The cases consisted of 546 individuals with pathologist-proven diagnosis of gastric cancer from CENAR and BMCH, while the controls (n = 546) comprised patients free of cancer selected attending the community and outpatient clinics. All the two groups had been matched for age, sex and SES to improve comparability and reduce confounding in

regard to dietary behaviors and additive knowledge.

Sample Size

In all, 546 cases and 546 controls were included (total sample of size 1092). The sample size was based on the amount of statistical power that would be required to detect significant relationships between dietary exposures and disease. Matching was to reduce potential confounding and increase group comparability. This large sample size was adequate to assess multiple variables.

Sampling Technique

Sampling method Participants were recruited through purposive and convenience sampling. Patients with gastric cancer are purposively selected in the oncology wards and outpatient clinics based on their confirmed diagnosis.

Convenience sampling of controls was carried out within general outpatient clinics to match cases with respect to eligibility criteria. This sampling technique enabled time-sensitive recruitment of PS via the study timeline while also promoting relevance and representativeness in both cohorts.

Inclusion Criteria

The inclusion criteria were able to recruit appropriate subjects in both the NHs and ICUs. Cases were adults with histologically-confirmed gastric cancer, and controls were those without a previous history of malignancy or serious gastrointestinal disease. To participate in the study, individuals had to be residents of Quetta for at least five years with informed consent. These selection criteria resulted in uniform environmental and dietary exposure among the study samples.

Exclusion Criteria

Exclusion criteria were as follows: missing diagnostic variables, severe chronic diseases other than gastric cancer and status that could influence dietary habits i.e.: advanced renal or hepatic disease. Pregnant and lactating women were not included to control for confounding physiological variations. Those unable to grant consent due to a lack of participation or subject's assent were also excluded. These exclusions also had the benefit of maintaining the stability and credibility of results from this—our study.

Data Collection Tools

A structured questionnaire survey was applied to include information concerning the demographical factors, dietary pattern, processed food consumption and knowledge on food additives. The questionnaire included lifestyle behavior, smoking and physical activity items. The details of the case were supported by trough medical records, laboratory, endoscopy and histopathological findings. These tools provided a summary of behavioral and clinical correlates of gastric cancer.

Study Variables

Gastric cancer included in or not was the main dependent variable of this study. The independent variables were food pattern, frequency of consumption and knowledge regarding the additives. Age, sex, socio-economic position, smoking and physical activity were considered confounders and adjusted for in the analysis. This flexible design allowed for a broad evaluation of diet and its association with the risk of gastric cancer.

Data Collection Procedure

The data were collected through face-to-face interviews by trained interviewer using a compatible survey to ensure clarity and correctness of responses. Gastric cancer cases were interviewed at outpatient clinic visit or hospitalization, and controls were consulted in the outpatient department. Clinical and laboratory information was recorded from hospital documents after receiving informed consent. Interviews and medical record abstract reviews provided comprehensive and reliable data.

Diagnostic Confirmation

The diagnosis for cases were confirmed by endoscopic evaluation, biopsy sampling and histopathological assessment. The endoscopic findings of abnormal gastric lesions, including ulcerated masses or antral thickening, could be confirmed at the microscopic level to be malignant by biopsies. Based on the histopathology, gastric cancers were divided into intestinal type, diffuse type and signet ring cell type. This protocol for the diagnostic standard could prevent cases from being incorrectly identified and misclassified.

Ethical Considerations

The study was approved by the ethical committee of both institutions. All subjects were given detailed information about the study in advance, including the purpose of the survey, confidentiality and data processing, before obtaining written consent from them. They had been promised the right to withdraw at any time with no repercussions. This data was also anonymously stored in a safe environment. All procedures adhered to ethical standards for research with human beings.

Data Analysis

Descriptive and inferential statistics were obtained using SPSS and Stata software. Demographic and clinical data were described using frequencies, means, and proportions. Chi-Square tests and independent t-tests were used to evaluate differences between cases and controls. Adjustment was made for dietary exposures in multivariate logistic regression models to investigate the association of gastric cancer with covariates. A value of $p < 0.05$ was used for all statistical significance interpretations.

Results

Demographic Characteristics

Demographics The demographic sample included 546 participants, which was evenly distributed by age, sex and education. The sample was composed of 290 males and 256 females, who were predominantly between the ages of 18-35. There was a diversity of educational backgrounds ranging from primary through to postgraduate levels, and bachelors were the most common group. In general, the demographic profile was of young and reasonably educated population which was considered appropriate for assessing dietary habits and awareness scores.

Table 1. Demographic Distribution of Participants (N = 546)

Variable	Category	Frequency
Gender	Male	290
	Female	256
Age Group	18-25	182
	26-35	215
	36-45	98
	46	51
Education Level	Primary	57
	Secondary	131
	Intermediate	125
	Bachelor	149
	Master+	84

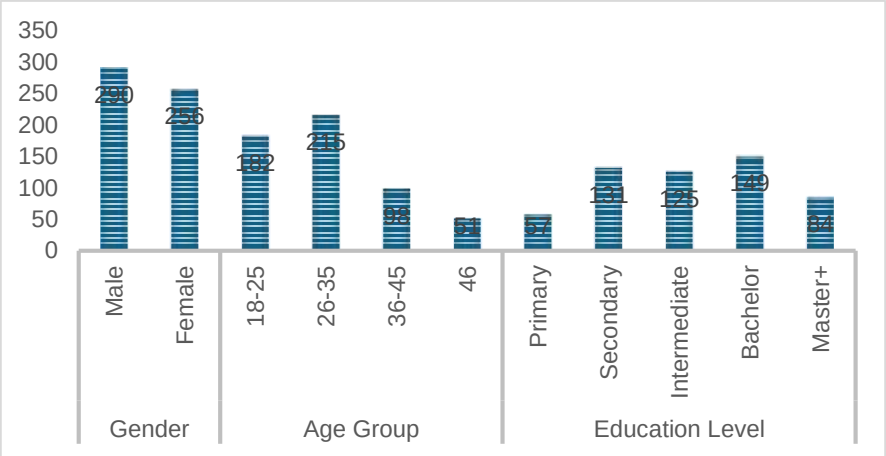

Figure 2. Demographic Distribution of Participants (N = 546)

Table 1 and figure 2 presents the demographic characteristic of the five hundred and forty-six (546) respondents. Gender distribution was equal in 546 subjects, (290 males, 248 females). The majority were young adults, ages 18-25 (n=182) and 26-35 (n=215). Similar trends

continued with levels of education, and those with a bachelor's degree made the largest proportion (149), followed by intermediate (125) and secondary (131). Less had primary (57) or postgraduate degrees (84). In general, the sample was diverse and young with relatively high levels of education which seemed appropriate to examine dietary patterns and awareness for gastric cancer risk.

Age Group Distribution

The age distribution was different across the defined categories in a total of 1092 to be studied. The 18-25 participants accounted for 80 cases and 102 controls, with a prevalence of 43.9%. They were followed by the 100 cases and 115 controls, for which the prevalence was 46.5%, in the age group of 26-35 years; they are followed by that of 55% observed among individuals aged >36-45 years. Prevalence in individuals aged 46+ years was 51.6%. This even distribution led to robust comparison between cases and controls.

Table 2: Age Group Distribution by Case-Control Groups (N = 1092)

Age Group (years)	Cases (n=546)	Controls (n=546)	Total Participants	Prevalence (%)
18–25	80	102	182	43.9
26–35	100	115	215	46.5
36–45	120	98	218	55.0
46+	246	231	477	51.6
Total	546	546	1092	50.0

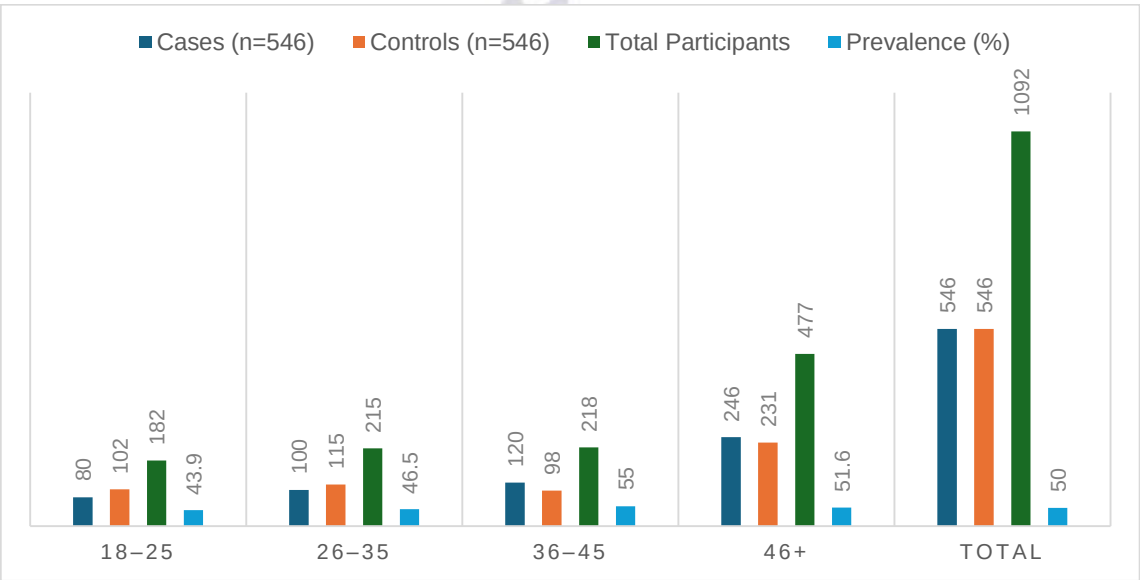

Figure 3: Age Group Distribution by Case-Control Groups (N = 1092)

The case-control distribution among age groups is presented in Table 2 and Figure 3 (n=1092). Prevalence was 43.9% (80 cases, 102 controls) among those aged 18-25. It was followed by 46.5% (100 cases, 115 controls) in 26-35 years' age group. Prevalence increased to 55·0% among the 36-45 (120 cases, 98 controls). And in the participants aged ≥46, prevalence was 51.6% (246 cases and 231 controls). The overall prevalence in all three groups was 50%.

Age and Gender Analysis

Analysis of age- and sex-specific distributions revealed characteristic demographic differences among the groups. There were 290 males and 256 females out of 546 patients with gastric

cancer, while the controls consisted of 275 males and 271 females. Those 46 years and older saw the highest percentage of cases by age. There were minimal incidences of the younger study participants, but they remained informative in evaluating diet exposures. These characteristics were conducive to stratified investigation of lifestyle and dietary factors.

Table 3: Age vs Gender Cross Data (Cases and Controls)

Age Group	Male Cases	Female Cases	Total Cases	Male Controls	Female Controls	Total Controls
18–25	45	35	80	55	47	102
26–35	55	45	100	60	55	115
36–45	65	55	120	50	48	98
46+	125	121	246	110	121	231
Total	290	256	546	275	271	546

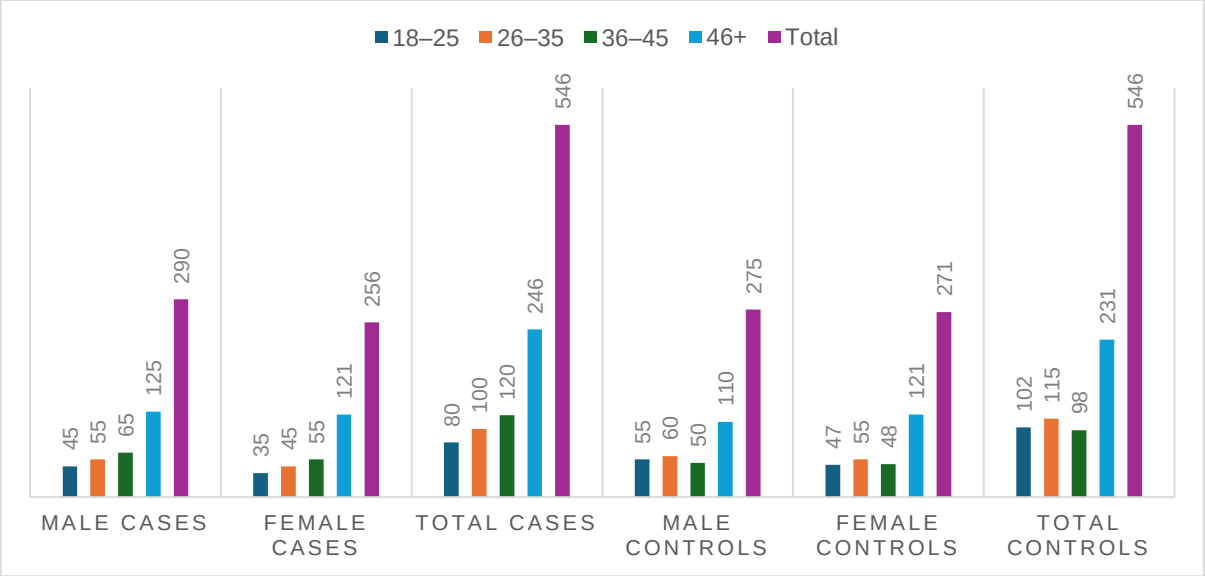

Figure 4: Age vs Gender Cross Data (Cases and Controls)

Distribution for age group and sex of cases and controls are depicted in table 3, figure 11. The distribution of all 546 cases and 546 controls according to age and sex is similar (case group: 290 men, 256 women; control group: 275 men, 271 women). In cases, there were 45 men and 35 women in the 18-25 group, and 55 men and 45 women in the 26-35 group. In the 36-45 group, there were a total of 65 men and 55 women. The ≥46 range was the most populated: 125 men and 121 women.

Comparison of Major Risk Factors

There were notable differences in exposure to risk factors among cases and controls. High-processed food consumption was found in 56.8% of GC patients, while this percentage was 32.9% for controls. Lack of knowledge on food additive risk was higher cases by 62.3%.

The patients were also more likely to be smokers, H. pylori-positive, have family history of cancer and follow a sedentary lifestyle. These distinctions underscored diverse behavioral and biologic factors in GC pathogenesis.

Table 4: Distribution of Major Risk Factors Among Cases and Controls (N = 1092)

Risk Factor	Category	Cases (n = 546)	Controls (n = 546)	Total (N = 1092)
Processed Food Intake	High	310 (56.8%)	180 (32.9%)	490 (44.9%)
	Low/Moderate	236 (43.2%)	366 (67.1%)	602 (55.1%)
Awareness of Food Additive Risks	Poor Awareness	340 (62.3%)	250 (45.8%)	590 (54.0%)
Smoking Status	Good Awareness	206 (37.7%)	296 (54.2%)	502 (46.0%)
	Smoker	255 (46.7%)	170 (31.1%)	425 (38.9%)
H. pylori Infection History	Non-Smoker	291 (53.3%)	376 (68.9%)	667 (61.1%)
	Positive	285 (52.2%)	165 (30.2%)	450 (41.2%)
Family History of Cancer	Negative	261 (47.8%)	381 (69.8%)	642 (58.8%)
	Yes	180 (32.9%)	96 (17.6%)	276 (25.3%)
Physical Activity	No	366 (67.1%)	450 (82.4%)	816 (74.7%)
	Sedentary	305 (55.8%)	210 (38.5%)	515 (47.2%)
Age Group	Active/Moderate	241 (44.2%)	336 (61.5%)	577 (52.8%)
	18–25	80 (14.7%)	102 (18.7%)	182 (16.6%)
	26–35	100 (18.3%)	115 (21.1%)	215 (19.7%)
	36–45	120 (22.0%)	98 (17.9%)	218 (20.0%)
	46+	246 (45.0%)	231 (42.3%)	477 (43.7%)

Figure 1: Prevalence of High-Risk Factors in Cases vs. Controls

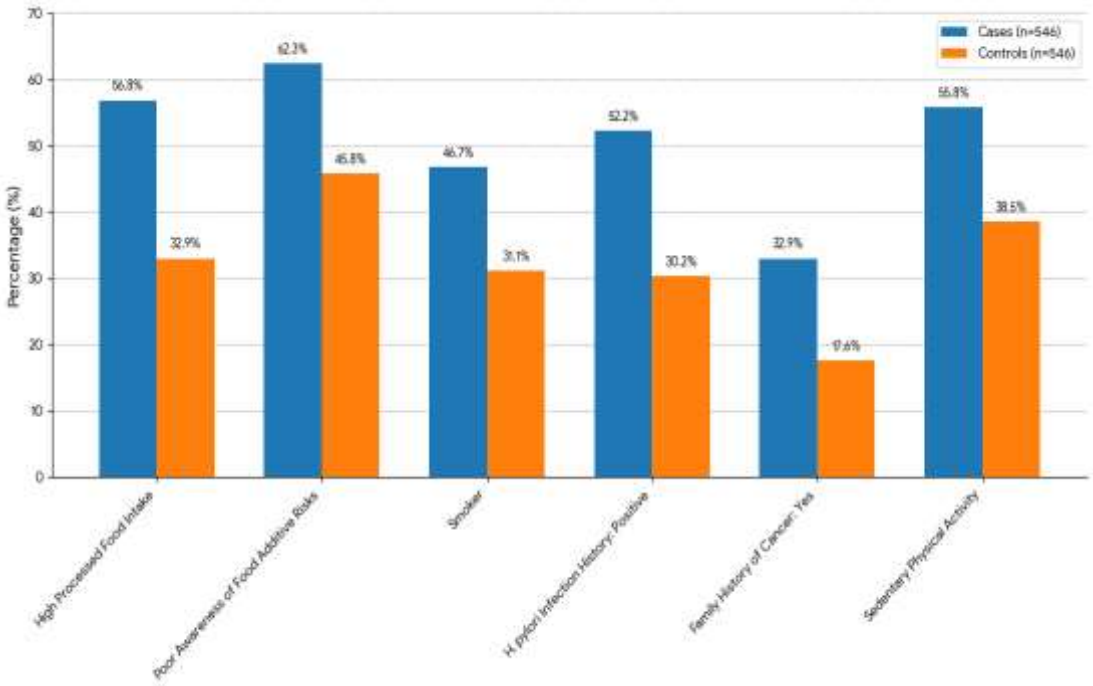

Figure 5: Distribution of Major Risk Factors Among Cases and Controls (N = 1092)

The distribution of the primary risk factors between cases and controls, among 1092 subjects, is shown in table 4and figure 5. The 1092 participants: classical risk factors were different in case and control. Around 56.8% of cases reported high processed-food consumption followed by those reporting (32.9%) very low intake. Low awareness of risk related to food additives was frequent (62.3% of cases vs. 45.8% of controls). Ab Smoking was by 46·7% of cases and 31·1% of controls. Helicobacter pylori (H. pylori) infected 52.2% subjects, and 32.9% cases recorded family history of cancer. The rate of sedentary behavior was high, 67.1% of cases were inactive.

Laboratory and Diagnostic Findings

Various types of laboratory and diagnostic investigations such as CBC, liver and renal profile tests, H pylori diagnostics, endoscopic findings, histopathology and tumor markers have been done to establish the subject confutation of both cases and control groups. The integrated data showed that gastric cancer patients presented markedly in uniform with anemia, increased inflammatory markers, liver and renal function damage, elevated H. pylori infection rate, abnormal endoscopic pattern and histological features of malignancy as well as enhanced tumor markers levels. Altogether, these findings yielded a robust clinical-pathological validation of disease severity and consistent with the previously determined epidemiological connections.

Table 5: Combined Laboratory and Diagnostic Table

Category	Test/Parameter	Reference Range	Cases (n = 546)	Controls (n = 546)	Notes
CBC	Hemoglobin (g/dL)	12–16	10.8 ± 1.9	13.2 ± 1.4	Lower in cases (anemia)
	RBC Count ($\times 10^6$/µL)	4.0–5.5	3.9 ± 0.7	4.7 ± 0.5	Lower in cases
	WBC Count ($\times 10^3$/µL)	4–11	8.5 ± 2.1	7.2 ± 1.8	Higher in cases (inflammation)
	Platelets ($\times 10^3$/µL)	150–450	240 ± 60	280 ± 70	Lower in cases
	MCV (fL)	80–100	79.5 ± 10.2	85.3 ± 8.6	Reduced in cases
	MCH (pg)	27–33	25.8 ± 3.6	29.4 ± 3.1	Reduced in cases
LFT	ALT (U/L)	7–55	42 ± 15	32 ± 12	Elevated in cases
	AST (U/L)	8–48	46 ± 18	34 ± 10	Elevated in cases
	ALP (U/L)	40–130	138 ± 42	112 ± 35	Elevated in cases
	Total Bilirubin (mg/dL)	0.1–1.2	1.4 ± 0.6	0.8 ± 0.3	Higher in cases
	Albumin (g/dL)	3.5–5.0	3.1 ± 0.5	4.2 ± 0.4	Lower in cases
RFT	Serum Urea (mg/dL)	10–45	41 ± 12	32 ± 10	Higher in cases
	Creatinine (mg/dL)	0.6–1.3	1.1 ± 0.3	0.9 ± 0.2	Higher in cases
	eGFR (mL/min/1.73 m^2)	>90	79 ± 20	95 ± 15	Reduced in cases
H. pylori Tests	IgG ELISA	–	285 (52.2%)	165 (30.2%)	Most sensitive test
	Stool Antigen Test	–	260 (47.6%)	150 (27.4%)	Confirms active infection

	Rapid Urease Test (RUT)	–	210 (38.4%)	110 (20.1%)	Detects urease activity
Endoscopy	Ulcerated Gastric Mass	–	210 (38.4%)	–	Most common lesion
	Antral Thickening	–	165 (30.2%)	–	Second most common
	Pyloric Obstruction	–	70 (12.8%)	–	Indicates advanced disease
	Diffuse Wall Thickening	–	55 (10.1%)	–	Suggestive of late-stage cancer
	Irregular Ulcers	–	46 (8.4%)	–	Least common finding
Histopathology	Adenocarcinoma (Intestinal Type)	–	305 (55.8%)	–	Most common subtype
	Adenocarcinoma (Diffuse Type)	–	150 (27.4%)	–	Infiltrative pattern
	Signet Ring Cell Carcinoma	–	65 (11.9%)	–	More aggressive
	Poorly Differentiated Carcinoma	–	26 (4.9%)	–	Least common
Tumor Markers	CEA (ng/mL)	<5	12.5 ± 4.8 (64% elevated)	–	Strong predictor
	CA 19-9 (U/mL)	<37	58.3 ± 20.4 (52.3% elevated)	–	High diagnostic value
	CA 72-4 (U/mL)	<6	9.8 ± 3.5 (47.5% elevated)	–	Elevated in nearly half

The comparison of laboratory, diagnostic and pathological features between 546 cases and 546 controls is shown in Table 5. In both cases, abnormalities were evident on several parameters. Blood CBC showed anemia and inflammation with decreased hemoglobin, RBCs, platelets, MCV and MCH, whereas WBC value was increased. Liver and kidney function markers were higher in cases (ALT, AST, ALP, bilirubin, urea and creatinine), whereas albumin and eGFR were lower. Positivity of H. pylori was significantly higher in patients in all IgG ELISA (52.2%), stool antigen (47.6%), and RUT (38.4%). Endoscopy investigations were positive in cases only, with ulcerated gastric masses (38.4%) most frequently observed followed by antral thickening (30.2%) and pyloric obstruction (12.8%). The overall histopathological type of cancer was adenocarcinoma (intestinal type) in the majority of the cases (55.8%) with co-existence of diffuse-type and signet-ring cell carcinoma. Elevated tumor markers (CEA, CA 19-9, CA 72-4) were common and diagnostic in these patients.

Discussion

The results of the present study showed that high consumption of processed foods was significantly associated with gastric cancer in adult population in Quetta. Cases reported significantly higher intake of processed meats, refined foods, and additive-containing diets, suggesting that Western dietary style has a significant effect on gastric cancer risk (18). These findings corroborate previous epidemiological evidence that unhealthy dietary patterns tend to contribute much to gastric

carcinogenesis, especially in populations with low nutritional knowledge (19).

Infection was the most significant risk factor for gastric cancer in this population and positivity rates were higher among cases. This supports the perspective of Thrift and El-Serag (2020), that long term H. pylori induced gastritis is a central route to gastric carcinogenesis (20). Observed infection prevalence is overall in line with worldwide estimates for H. pylori, of which 75.9% of infections are the result to being exposed lifelong (IARC,2025). These results underscore the need for early detection and elimination schemes, particularly in high risk areas like Quetta. Risk factors of GC patients The ratio of smoking, sedentariness, no fruit and vegetable intake were significantly higher for GC patients. Smoking causes mucosal gastric damage and acts in concert with dietary carcinogens as reported in (21). Outcomes are in line with international reviews highlighting physical inactivity being a driver of dietary risk/cancer (22). Thus, lifestyle modification remains an important component of integrated gastric cancer prevention strategies.

Endoscopic, histopathologic findings were that the intestinal type of adenocarcinoma was most common in Quetta and it was in accordance with dietary pattern and environmental risk factors. This is consistent with worldwide trends where the intestinal type of adenocarcinoma has been reported as a leading type associated with diet, smoking, and H. pylori infection (23). The prevalence of advanced lesions such as ulcerated masses and antral thickening is high indicating late diagnosis, which reflect low screening coverage and poor symptom awareness in the area (24).

Laboratory analysis including anemia, elevated liver enzymes and reduced renal function could be systemic reactions to far advanced gastric carcinoma. These data are consistent with previous reports that indicate undernutrition, chronic inflammation and tumor load are associated with hematologic and biochemical parameters in cancer patients (25). Elevated tumor markers especially CEA and CA 19-9 are additional evidences supporting their diagnostic value. These changes show the importance of physical examination for early detection and follow-up of gastric cancer.

In conclusion, this investigation indicates that the development of gastric cancer in Quetta is a multifactorial interplay between dietary exposures, lifestyle factors, biological characteristics and poor food-related risk awareness. These findings are supporting earlier umbrella reviews who also have confirmed diet as a major contributor to global burden of gastric cancer (26). In sum, the cumulative evidence emphasizes concerted intervention strategies including nutritional education, regulation on food additives, H. pylori screening and/or improved diagnostic accessibility across Balochistan.

Conclusion

The food eating habits, particularly consumption of junk food and harmful additives to the available eatable items were recognized as potential determinants of the alarming high prevalence of gastric cancer rate in Urban Quetta observed in this study. Bad P.R. for carcinogenic additives, as well as unhealthy living patterns (like smoking, and being still), also adds to the odds. The findings are consistent with the traditional understanding of a multifactorial causation of gastric cancer with both diet, behavior and biological factors such as H. pylori infection acting together. Increasing awareness of communities, promoting a healthy diet, regulating additives and organizing early screening programs to reduce the disease burden are also crucial. These views emphasize the urgent need for culture-specific focused public health interventions.

Acknowledgement

I would like to make an authentic thanks to my supervisor, **Dr. Muhammad Sharif Hasni**, for his guidance, support and motivation throughout the realization of this work. I am thankful to my teachers for their invaluable inspiration and academic guidance; and my friends for all the support they extended to me during this period. My thanks also go to the dedicated staff of participating hospitals and other laboratories who provided aid, support and allowed us access in order for us to collect

data and perform analyses. With thanks to all of you for your generous support that made this typescript come alive.

REFERENCES

Conti, C. B., Agnesi, S., Scaravaglio, M., Masseria, P., Dinelli, M. E., Oldani, M., & Uggeri, F. (2023). Early gastric cancer: update on prevention, diagnosis and treatment. International journal of environmental research and public health, 20(3), 2149.

Ilic, M., & Ilic, I. (2022). Epidemiology of stomach cancer. *World journal of gastroenterology*, *28*(12), 1187.

Mannucci, A., & Goel, A. (2025). Advances in pancreatic cancer early diagnosis, prevention, and treatment: The past, the present, and the future. *CA: A Cancer Journal for Clinicians*.

Song, Y., Liu, X., Cheng, W., Li, H., & Zhang, D. (2022). The global, regional and national burden of stomach cancer and its attributable risk factors from 1990 to 2019. *Scientific Reports*, *12*(1), 11542.

Pu, K., Feng, Y., Tang, Q., Yang, G., & Xu, C. (2024). Review of dietary patterns and gastric cancer risk: epidemiology and biological evidence. *Frontiers in Oncology*, *14*, 1333623.

Reytor-González, C., Zambrano, A. K., Montalvan, M., Frias-Toral, E., Simancas-Racines, A., & Simancas-Racines, D. (2024). Adherence to the Mediterranean Diet and its association with gastric cancer: health benefits from a Planeterranean perspective. *Journal of Translational Medicine*, *22*(1), 483.

Adhikari, S. (2021). Additives and preservatives used in food processing and preservation, and their health implication. *Food Chemistry: The Role of Additives, Preservatives and Adulteration*, 43-72.

Chazelas, E., Srour, B., Julia, C., Deschasaux, M., Andrianasolo, R.-M., Druesne-Pecollo, N., Kesse-Guyot, E., Touvier, M., & Hercberg, S. (2022). Processed meat and gastric cancer risk: Involvement of nitrosamines and gut microbiota. International Journal of Epidemiology, 51(4), 1106–1117. https://doi.org/10.1093/ije/dyac132

Ryu, S. W., Son, Y. G., & Lee, M. K. (2020). Motivators and barriers to adoption of a healthy diet by survivors of stomach cancer: A cross-sectional study. *European Journal of Oncology Nursing*, *44*, 101703.

Daniyal, M., Ahmad, S., Ahmad, M., Asif, H. M., Akram, M., Ur Rehman, S., & Sultana, S. (2015). Risk factors and epidemiology of gastric cancer in Pakistan. *Asian Pac J Cancer Prev*, *16*(12), 4821-4.

Bhurgri, Y., Bhurgri, A., Nishter, S., Ahmed, A., Usman, A., Pervez, S., ... & Bashir, I. (2006). Pakistan-country profile of cancer and cancer control 1995-2004. *Journal of the Pakistan Medical Association*, *56*(3), 124.

Seto, T., Grondin, J. A., & Khan, W. I. (2025). Food Additives: Emerging Detrimental Roles on Gut Health. *The FASEB Journal*, *39*(13), e70810.

Ilic, M., & Ilic, I. (2022). Epidemiology of stomach cancer. *World journal of gastroenterology*, *28*(12), 1187.

Maddineni, G., Xie, J. J., Brahmbhatt, B., & Mutha, P. (2022). Diet and carcinogenesis of gastric cancer. *Current opinion in gastroenterology*, *38*(6), 588-591.

Du, S., Li, Y., Su, Z., Shi, X., Johnson, N. L., Li, P., ... & Ding, X. (2020). Index-based dietary patterns in relation to gastric cancer risk: a systematic review and meta-analysis. *British journal of nutrition*, *123*(9), 964-974.

Daniyal, M., Ahmad, S., Ahmad, M., Asif, H. M., Akram, M., Ur Rehman, S., & Sultana, S. (2015). Risk factors and epidemiology of gastric cancer in Pakistan. *Asian Pac J Cancer Prev*, *16*(12), 4821-4.

Ryu, S. W., Son, Y. G., & Lee, M. K. (2020). Motivators and barriers to adoption of a healthy diet by survivors of stomach cancer: A cross-sectional study. *European Journal of Oncology Nursing*, *44*, 101703.

Pu, K., Feng, Y., Tang, Q., Yang, G., & Xu, C. (2024). Review of dietary patterns and gastric cancer risk: epidemiology and biological evidence. *Frontiers in Oncology*, *14*, 1333623.

Peter T. Campbell, Margaret Sloan, Nancy Kreiger, Dietary Patterns and Risk of Incident Gastric Adenocarcinoma, American Journal of Epidemiology, Volume 167, Issue 3, 1 February 2008, Pages 295–304, https://doi.org/10.1093/aje/kwm294

Thrift, A. P., & El-Serag, H. B. (2020). Burden of gastric cancer. Gastroenterology, 158(2), 285–295. https://doi.org/10.1053/j.gastro.2019.08.068

Balendra, V., Amoroso, C., Galassi, B., Esposto, J., Bareggi, C., Luu, J., ... & Ghidini, M. (2023). High-salt diet exacerbates H. pylori infection and increases gastric cancer risks. *Journal of Personalized Medicine*, *13*(9), 1325.

Pradhan, S. P., Gadnayak, A., Pradhan, S. K., & Epari, V. (2025, June). Epidemiology and prevention of gastric cancer: A comprehensive review. In *Seminars in Oncology* (Vol. 52, No. 3, p. 152341). WB Saunders.

Kim, S., Kim, M. S., Kwon, Y., Min, J. S., Alromi, A., Kim, J. Y., ... & Park, S. (2025). Environmental Protective and Risk Factors for Gastric Cancer: An Umbrella Review and Reanalysis of Meta-Analyses. *Journal of gastric cancer*, *25*(2), 285.

Morgan, E., Arnold, M., Camargo, M. C., Gini, A., Kunzmann, A. T., Matsuda, T., ... & Soerjomataram, I. (2022). The current and future incidence and mortality of gastric cancer in 185 countries, 2020–40: a population-based modelling study. *EClinicalMedicine*, *47*.

Lis, C. G., Gupta, D., Lammersfeld, C. A., Markman, M., & Vashi, P. G. (2012). Role of nutritional status in predicting quality of life outcomes in cancer: A systematic review of the epidemiological literature. Nutrition Journal, 11, 27. https://doi.org/10.1186/1475-2891-11-27.

Liang, J. L., Yuan, H. M., Quan, C., & Chen, J. Q. (2025). Risk factors for gastric cancer: an umbrella review of systematic reviews and meta-analyses. *Frontiers in Oncology*, *15*, 1564575.