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Research Article

Association between Dietary Acid Load and Risk of Gastric Cancer: A Case-Control Study

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ABSTRACT

Background and objective: The association between Dietary Acid Load (DAL) and the risk of various cancers has been conflicting. Furthermore, no study has examined the association between DAL and the risk of Gastric Cancer (GC) so far. This investigation was conducted to assess the association between dietary acid load and GC risk.

Methods: In this case-control study, 184 patients with newly diagnosed gastric cancer and 2763 healthy controls were enrolled. A validated Diet History Questionnaire (DHQ) was applied for dietary assessments. DAL was calculated by computing amounts of protein intake into potassium contents. Logistic regression was used to define the association between DAL and GC risk.

Results: Mean age of participants was 56.26; of them, 31.7% were women. There was a positive association between DAL and GC risk (OR 4.59; 95% CI 2.61-8.07; P-trend<0.001, for T2 versus T3). In the full model, adjusted for all covariates, including BMI, DAL was positively associated with the risk of GC (OR 3.55; 95% CI 1.89-6.99; P-trend <0.001).

Conclusion: We found that DAL was associated significantly with greater gastric cancer odds in this case-control study. This finding supports the current recommendation for healthy eating to lower the risk of cancer incidence.

Keywords: Diet; Stomach cancer; Case-control; Women; Cancer

INTRODUCTION

Gastric Cancer (GC) is the fifth most prevalent cancer worldwide [1], while it is the third cause of death from cancer [2] which indicates a poor prognosis for this cancer. This emphasizes the importance of prevention and early detection strategies. To this end, the main risk factors of gastric cancer should be recognized. Smoking, *H. pylori* infection, heavy alcohol intake, and industrial chemical exposures are established risk factors for GC [3]. Nutritional factors are considered important risk factors [4], but there is no strong evidence except for body fatness [5] and food preserved by salting [6]. However, evidence on the association between intakes of several nutrients or food groups with GC is not convincing and unable researchers to make a firm conclusion.

Although GC incidence decreases in several developed countries, it remains high in some countries, including Iran [7]. It is the first common cancer death, the first common cancer in men, and the second common cancer in women in Iran [8]. The high prevalence of gastric cancer and the different dietary habits make Iran excellent for studying the association between dietary habits and GC risk. We conducted a hospital-based case-control

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study to investigate the association of Iranian dietary patterns with the GC risk. Existing evidence studied the association of sole foods with gastric cancer [9-11]. However, several foods are consumed mixed, and these foods contain thousands of nutrients and chemicals, which are considered to have interactive effects. Clearly, studies on individual foods hardly consider the synergistic or inhibitory effect. Furthermore, there are many undiscovered compounds in our diet which could have unknown effects on our health [12-15]. Dietary Acid Load (DAL) is defined as the acidity potential of a diet and is considered to be associated with the risk of several diseases. Food groups such as meat, cereal, and eggs could increase the DAL score of a diet, and fruit and vegetable play a reverse role [16-19].

Dietary acid load is defined by the balance of acid-producing food like meat, eggs, cereal grain, and base-producing foods such as fruits and vegetables [20]. It is suggested that consuming a high potassium base-producing diet can decrease the incidence of gastric cancer and other mortality-related conditions [21].

Earlier studies have demonstrated the association between DAL and the risk of various types of cancers. However, these results were conflicting and inconsistent. However, according to our current information, no studies have investigated the association between DAL and GC risk worldwide. This study was done to assess the association between dietary acid load and gastric cancer odds in Iran.

MATERIALS AND METHODS

Study population: This case-control study was conducted between May 2010 and June 2012 in the cancer research institute, Imam Khomeini complex, Tehran university of medical sciences, Tehran, Iran. Patients diagnosed with GC, referred from all-around Iran to Imam Khomeini hospital, took part. Patients who were pathologically diagnosed with GC in less than six months and with no previous history of any cancer were identified as eligible for the current study. Case employment was based on the convenience sampling method. A non-random sampling method was applied for enrolling 276 controls among healthy people. The control group has been chiefly picked out among relatives of patients in other hospital wards. The response rate was 95% in cases and 70% in controls.

Dietary assessment: Dietary patterns were determined by applying a 146 items, semi-quantitative, dish-based Diet History Questionnaire (DHQ). Comprehensive information about this questionnaire, including its design, development, and validity, will be explained subsequently. DHQ was completed by performing a face-to-face interview. Controls were asked to report their last 12 months' dietary intake in the form of Iranian home scales like a spoon, plate, bowl, and scoop. Reported amounts were converted to grams/day by household equipment. Patients with gastric cancer were asked to recall their intake before the cancer symptoms showed off. Total daily energy and macro and micronutrients were calculated using food consumption tables provided by the United State Department of Agriculture (USDA). Dietary Acid Load as NEAP (daily dietary Net Endogenous Acid Load Production) was estimated by dividing the amount of protein intake into potassium through the following formula: NEAP=(54.5*g protein/potassium meq)-10. Tertile cut-off points of these dietary scores were obtained based on scores in control subjects to avoid potential bias that might arise due to patient changes in dietary intakes.

In terms of the validity of the DHQ, we found a high correlation coefficient between nutrient estimations by DHQ and those from multiple 24 h recalls. This was the case for energy, protein, carbohydrate, protein, fiber, vitamin A, carotene, niacin, folate, vitamin B_{12}, biotin, vitamin C, sodium, magnesium, iron, zinc, and selenium ($r>0.5$ for all).

Assessment of gastric cancer: Gastric cancer was diagnosed based on a gastroscopic or surgical biopsy that an expert pathologist did. Patients were included if they were diagnosed with stomach cancer histologically, considering the definition of gastric cancer provided by the second edition of the International Classification of Diseases for Oncology (ICDO code c16). Only patients who had been diagnosed during one preceding year to the date of the interview were eligible for participating in the present study.

Assessment of covariates: A trained bachelor of health sciences conducted an organized upstanding interview. Demographic and general information, including gender, marital status, education level, residential places, and smoking and drinking habits, were derived from the questionnaire. Since body weight status is influenced by gastric cancer, the actual weight of patients was not considered in this study. Usual weight and height were collected by asking participants about their weight before cancer diagnosis individually Body Mass Index (BMI) was calculated by dividing weight in kilograms by height in meters squared. Patients were categorized as current smokers and non-smokers according to their reported smoking habits last year. Ten milliliters of venous blood samples were taken from all attendants at both fasting and non-fasting status to examine for *H. pylori* infection. ELISA kits were applied to measure serum samples for IGF antibodies. Serologic examinations were carried out by experienced technicians who were aware of neither the study setting nor the participants' case/control status. The *H. pylori* antibody test was repeated in a random collection of serums to prove validity. The existence of antibodies and sero-positivity of y more than 0, 87 was considered positive.

Statistical analysis: Dietary acid load was categorized into tertiles, and participants' dietary intake was compared across these tertiles using one-way ANOVA. Categorical variables were compared using the *Chi-square* test, and one-way ANOVA was used to compare continuous variables. Logistic regression was used to define the association between DAL and GC risk. In first model, we adjusted for age (continuous), sex (male/female) and energy intake (continuous). Further adjustments were done for education (illiterate/literate), marital status (married/single), *H. pylori* infection (positive/negative), alcohol intake (continuous) and smoking status (smoker/nonsmoker) in the second model. Additional control was applied in the third model for BMI.

RESULTS

This case-control study enrolled 184 patients with newly diagnosed gastric cancer and 273 healthy controls. The mean age of study participants was 56.6 (men: 58.5 and women: 52.6). Almost 32% of study participants were women. The general characteristics of participants are shown in Table 1. Age has been significantly different across tertile of dietary acid load in controls (57.3 ± 11.6 versus 49.0 ± 11.8). Participants in tertile 3 of dietary acid load have remarkably lower alcohol intake than those in tertile 1 (9.2 ± 95.2 versus 1.7 ± 10.7). Other characteristics of participants were not significantly different in tertile of DAL in neither cases nor controls.

Table 1: General characteristics of participants across tertile of dietary acid load[a].

Characteristics	Controls				Cases			
	T1 (n=91)	T2 (n=91)	T3 (n=91)	P[b]	T1 (n=31)	T2 (n=64)	T3 (n=89)	P[b]
Age (years)	57.3 ± 11.6	53.3 ± 10.7	49.0 ± 11.8	<0.001	61.3 ± 13.1	60.6 ± 13.1	62.6 ± 12.4	0.62
Alcohol intake (g/day)	3.2 ± 18.4	0.31 ± 1.8	1.8 ± 9.4	0.28	0.13 ± 0.73	0.8 ± 0.52	1.2 ± 10.9	0.09
BMI (kg/m^2)	25.5 ± 4.4	25.7 ± 5.7	25.7 ± 5.3	0.93	27.1 ± 4.6	26.8 ± 4.6	25.3 ± 5.4	0.1
Gender (male %)	67 (73.6)	53 (58.2)	54 (59.3)	0.06	22 (70.9)	48 (75.0)	68 (76.4)	0.83
Marital status (married %)	20 (21.9)	28 (30.8)	23 (25.3)	0.39	21 (67.7)	36 (56.3)	66 (74.2)	0.07
Education (literate %)	71 (78.0)	61 (67.0)	71 (78.0)	0.15	14 (45.2)	28 (43.8)	26 (29.2)	0.11
Smoking (yes %)	24 (26.4)	31 (34.1)	27 (29.7)	0.53	12 (38.7)	29 (45.3)	45 (50.6)	0.5
H. pylori infection (positive %)	44 (48.4)	49 (53.9)	59 (64.8)	0.07	15 (48.4)	24 (37.5)	33 (37.1)	0.51

Note: [a]Reported figures are means ± SDs unless indicated.

[b]Obtained from *Chi-square* test for categorical and one-way ANOVA for continuous variables.

The dietary intakes of participants are demonstrated in Table 2. The higher dietary acid load was associated with higher intakes of energy, proteins, fats, fiber, zinc, selenium, red meat, grains, fruits, vegetables, dairy, and fish in controls. In patients, only intake of selenium, grains, vegetables, and dairy was significantly different in tertile of DAL.

Table 2: Dietary intakes of participants across tertile of dietary acid load[a].

Characteristics	Controls				Cases			
	T1 (n=91)	T2 (n=91)	T3 (n=91)	P[b]	T1 (n=31)	T2 (n=64)	T3 (n=89)	P[b]
Energy (kcal/day)	2540.1 ± 961.3	3044.3 ±1140.6	2880.3 ± 1135.2	0.006	2246.7 ± 1394.6	2271.3 ± 1016.2	2541.4 ± 1178.3	0.34
Protein (% of energy)	13.3 ± 4.4	16.3 ± 3.5	16.5 ± 3.6	<0.001	17.4 ± 4.4	18.7 ± 3.6	18.7 ± 4.5	0.3
Fats (% of energy)	25.5 ± 10.4	32.4 ± 7.9	30.6 ± 9.7	<0.001	29.7 ± 7.3	31.2 ± 7.5	30.8 ± 7.3	0.66

Carbohydrates (% of energy)	55.3 ± 9.9	53.1 ± 7.4	54.3 ± 9.2	0.24	54.4 ± 7.8	52.8 ± 6.8	52.9 ± 7.9	0.57
Dietary fiber (g/day)	23.5 ± 8.9	22.8 ± 9.5	18.9 ± 7.9	0.001	19.8 ± 13.9	17.9 ± 7.8	16.6 ± 8.3	0.24
Zink (mg/day)	11.5 ± 5.5	14.3 ± 5.6	12.9 ± 4.9	0.002	10.4 ± 5.2	11.9 ± 4.7	12.3 ± 5.1	0.17
Selenium (µg/day)	78.4 ± 39.9	96.9 ± 40.9	111.4 ± 67.7	<0.001	59.2 ± 43.8	62.3 ± 36.7	87.8 ± 61.2	0.002
Red meat (g/day)	15.6 ± 20.6	15.8 ± 17.8	19.9 ± 24.4	0.009	12.2 ± 12.7	16.1 ± 17.4	17.7 ± 18.9	0.33
Grains (g/day)	152.8 ± 101.4	199.4 ± 126.6	271.6 ± 222.8	<0.001	75.3 ± 80.7	104.9 ± 102.1	188.6 ± 213.2	<0.001
Fruits (g/day)	589.4 ± 368.1	584.4 ± 352.3	349.9 ± 262.2	<0.001	483.3 ± 407.2	399.7 ± 253.8	280.3 ± 187.1	<0.001
Vegetables (g/day)	457.2 ± 213.9	377.4 ± 210.1	267.7 ± 176.0	<0.001	320.3 ± 260.4	262.2 ± 166.8	195.1 ± 110.3	<0.001
Dairy (g/day)	852.2 ± 652.5	1214.6 ± 758.1	960.5 ± 611.2	0.001	919.7 ± 580.4	1150.9 ± 537.5	1020.6 ± 619.7	0.16
Low-fat dairy	525.9 ± 600.3	630.0 ± 738.2	556.3 ± 609.1	0.524	556.1 ± 519.4	734.7 ± 602.3	512.0 ± 571.8	0.06
Fish (g/day)	7.3 ± 3.5	8.3 ± 5.5	11.2 ± 10.4	<0.001	10.8 ± 9.8	9.3 ± 6.5	10.3 ± 7.0	0.62
Nuts (g/day)	7.7 ± 19.8	8.6 ± 21.6	3.8 ± 6.6	0.14	5.9 ± 18.6	4.7 ± 7.4	3.4 ± 3.9	0.38
Legume (g/day)	32.4 ± 25.4	35.6 ± 30.4	36.3 ± 26.6	0.59	32.7 ± 29.1	32.0 ± 21.9	35.1 ± 25.8	0.73

Note: [a]Reported figures are means ± SDs.

[b]Using one-way ANOVA.

Crude and adjusted odds ratios of GC across tertile of dietary acid load are provided in Table 3. In the crude model, we found a positive association between DAL and GC risk (OR 2.87; 95% CI 1.74-4.74; P-trend <0.001). The association strengthened after adjusting for age, sex, and energy intake (OR 4.59; 95% CI 2.61-8.07; P-trend <0.001). Even after additional adjustment for other potential confounders, DAL was positively associated with the risk of GC (OR 3.56; 95% CI 1.89-6.69; P-trend <0.001). Additional adjustments for BMI did not significantly affect this association.

Table 3: Odds ratio and 95% Confidence Interval (CIs) for gastric cancer across tertile of dietary acid load.

	T1	T2	T3	Ptrend[a]
Cases/control (273/184)	91/31	91/64	91/89	-
Crude	1	2.06 (1.23-3.47)	2.87 (1.74-4.74)	<0.001
Model A[b]	1	3.05 (1.72-5.39)	4.59 (2.61-8.07)	<0.001
Model B[c]	1	2.52 (1.34-4.79)	3.56 (1.89-6.69)	<0.001
Model C[d]	1	2.49 (1.32-4.72)	3.55 (1.89-6.96)	<0.001

Note: [a]Trend based on median values of each tertile.

[b]Adjusted for age (continuous), sex (male/female) and energy intake (continuous).

[c]Further adjusted for education (illiterate/literate), marital status (married/single), alcohol intake (continuous), *H. pylori* infection (positive/negative), and smoking status (smoker/nonsmoker).

[d]Additionally, controlling for BMI (continuous).

DISCUSSION

In the current case-control study, which is the first to the best of our knowledge, we investigated the association between dietary acid load and gastric cancer odds. We found a significant positive association between DAL and the risk of GC. This association even got stronger after adjusting for several potential confounders.

Previous studies have assessed the association between acid-producing foods and the risk of several cancers. However, there is no report available about the association with stomach cancer. In a case-control that reported the association of dietary acid load with the risk of breast cancer, Safabakhsh, et al., failed to find a significant association between high DAL and risk of breast cancer. Using the NEAP score, Alvaro et al. demonstrated a positive association between DAL and lung cancer. However, when the same investigators assessed DAL using the PRAL score, they failed to find any association. In a longitudinal study, Li-Wei-shi, et al., examined the association with the risk of pancreatic cancer. They found a significant positive association between DAL and risk of pancreatic cancer, either by Potential Renal Acid Load (PRAL) or NEAP score. Although assessment of DAL with risk of cancers is interesting, linking this exposure variable with the risk of gastric cancer is of high importance, particularly in an area where gastric cancer is highly prevalent and several dietary factors, including dietary acid load, may significantly contribute to this risk. Therefore, it is highly recommended that future studies examine this association in other regions using well-designed prospective cohort studies to confirm our findings.

Although there is no well-known mechanism through which dietary acid load might affect the risk of gastric cancer, there are some clues in this regard. Acidogenic diets may lead to a rise in cortisol levels, which can take part in carcinogenesis. Moreover, non-hormonal factors might be responsible for this observation. Some studies suggest that metabolic acidosis caused by acidogenic diets stimulates cancer metastasis. The inflammatory effects of dietary acid load are also investigated in various studies. Circulating levels of C-Reactive Protein (CRP) have increased after consuming an acidogenic diet in a randomized clinical trial. In addition, an increase in oxidative stress by diets full of red meat and processed food may also explain the association between dietary acid load and the risk of gastric cancer.

There are several strengths in the current study. Among them is the first study assessing the relationship between dietary acid load and risk of gastric cancer; adjusting for several covariates would make the results more validated. However, our study also has limitations: Although various probable confounders were considered in the present study, residual confounders cannot be ignored. In the case-control studies, we may face selection and recall biases. In addition, the use of FFQ for dietary assessment might result in some sort of misclassification. We used nutrient composition tables of foods of USDA, instead of local Iranian foods, in this study which can be considered a further limitation.

CONCLUSION

In conclusion, we found a positive association between dietary acid load and gastric cancer odds using a case-control design. This finding supports the current recommendation for healthy eating to lower the risk of cancer incidence. However, longitudinal studies are necessary to examine this issue further, particularly in other regions of the world where gastric cancer is prevalent. Assessment of the hypothesis in gastric precancerous lesions, including intestinal metaplasia and atrophic gastritis, may also help understand the mechanism behind this association.

In conclusion, we found that dietary acid load increases the risk of gastric cancer. These findings indicate that adherence to a healthy diet with an appropriate acid-base balance might help prevent cancer related conditions. Further studies, especially cohort studies, are required to confirm this finding. It is also essential to study the role of dietary load in the cardia and non-cardia stomach cancer.

HIGHLIGHTS

Gastric Cancer (GC) is the fifth most prevalent cancer worldwide. At the same time, it is the third cause of death from cancer. This indicated the low survival rate of this cancer. This emphasizes the importance of prevention and early detection strategies.

We found a positive association between dietary acid load and gastric cancer odds using a case-control design.

Adherence to a healthy diet with an appropriate acid-base balance might help prevent cancer-related conditions.

CONSENT FOR PUBLICATION

No data has been published by the name of the participants.

AVAILABILITY OF DATA AND MATERIALS

Data of this study are available on a reasonable request by KZ.

COMPETING INTERESTS

None of the authors declared conflicts of interest.

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AUTHORS' CONTRIBUTION

FT and KZ designed and developed the original study. SN, HM, and BS supervised sampling and the interviews. FT conducted the primary analysis of data. AE led analyzing process and drafting of this paper. FD developed the draft. All authors approved the final version of the manuscript.

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ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was conducted according to the guidelines laid down in the declaration of Helsinki and all procedures involving human subjects/patients were approved by the Ethics Committee of Cancer Research Center, Tehran University of Medical Sciences (no.17198). Written informed consent was obtained from all subjects.

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