



Metabolic Syndrome as a Risk Factor for Gastric Cancer by Gender

**Youngran Yang¹, Gum Mo Jung²*

1. School of Nursing, Research Institute of Nursing Science, Jeonbuk National University, 567 Baekje-daero, Deokjin-gu, Jeonju-si, Jeollabuk-do 561-756, Republic of Korea
2. Division of Gastroenterology, Department of Internal Medicine, Presbyterian Medical Center, Jeonju, Republic of Korea

***Corresponding Author:** Email: youngran13@jbnu.ac.kr

(Received 12 Dec 2019; accepted 26 Dec 2019)

Dear Editor-in-Chief

The relationship between metabolic syndrome and gastric cancer has not been well-known yet and the existing studies are also not consistent by gender (1,2). We aimed to understand the association between metabolic syndrome, metabolic components, and the risk of gastric cancer incidences according to gender by using the integrated data from a community-based cohort study in Ansong - Ansan as a part of the Korean Genome and Epidemiology study (KoGES). Case of gastric cancer was confirmed by self - questionnaire asking if the participants diagnosed as gastric cancer at the point of survey each time. Metabolic syndrome was defined by the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III) (3). Multivariate-adjusted Cox proportional regression analysis computed hazard ratios (HRs) and 95% confidence intervals (CIs).

The average follow-up duration of 9,937 participants aged 40-69 was 10.8 yr (107,488 person-years at risk). Twenty nine cases (0.6%) in women and 59 cases (1.2%) of gastric cancer in men were identified during the follow-up.

The finding of this study indicated a positive association between elevated fasting glucose levels and gastric incidence in women; this is consistent with studies that revealed a significant association of glucose levels and gastric cancer in women (2).

High glucose concentrations may be toxic to endothelial cells due to DNA damage, leading to mutational changes in oncogenes and tumour suppressor genes (4).

Our study found that high blood pressure is associated in women, but not in men. The 11 national prospective population-based cohort in Norway stated that self-reported hypertension history is related to an increased risk gastric adenocarcinoma in women (HR 2.41, 95% CI 1.44-4.03) (Table 1). It is plausible that increased levels of calcium are likely to be involved in the pathogenesis of hypertension and in the early events of cell proliferation (5).

In this study, low HDL cholesterol was associated with the risk of gastric cancer development in men. HDL cholesterol is essential for maintaining the normal cell cholesterol homeostasis by removing excess cholesterol from an intracellular space (6). Thus, the association of HDL cholesterol and gastric cancer needs to be investigated. The results of this study can be applied toward the development of public health programs for the management of metabolic health and prevention of gastric cancer. Further research is needed to clarify the mechanisms of association significant in this study and the gender difference between metabolic components and the risk of gastric cancer.



Table 1: Hazard ratios for the development of gastric cancer

<i>Variable</i>	<i>Women</i>		<i>Men</i>	
	Adjusted HR (95% CI)	<i>P</i> -value	Adjusted HR (95% CI)	<i>P</i> -value
Metabolic syndrome				
No	Reference	.725	Reference	0.983
Yes	1.19 (0.49-2.78)		1.01 (0.51-2.01)	
Number of composites of metabolic syndrome				
0	Reference		Reference	
1	2.28 (0.45-11.59)	.320	1.41 (0.65-3.09)	.340
2	4.25 (0.86-21.09)	.077	1.64 (0.71-3.76)	.245
3	3.25 (0.58-18.29)	.181	1.85 (0.73-4.70)	.194
4	2.85 (0.40-20.17)	.293	0.00 (0.00-0.00)	.937
5	8.60 (0.98-75.24)	.052	4.40 (0.53-36.75)	.171
Fasting glucose (mg/dl)				
<100	Reference		Reference	
100-125	0.00 (0.00-0.00)	0.963	0.73 (0.26-2.05)	0.556
126-139	7.80 (1.73-35.09)	0.007*	0.00 (0.00-0.00)	0.973
≥140	2.42 (0.32-18.26)	0.390	0.57 (0.08-4.14)	0.575
Total Cholesterol (mg/dl)				
<200	Reference		Reference	
200-239	0.68 (0.28-1.61)	0.676	1.08 (0.60-1.96)	0.800
≥240	0.29 (0.04-2.23)	0.236	0.47 (0.11-1.99)	0.308
LDL Cholesterol (mg/dl)				
<100	Reference		Reference	
100-129	1.60 (0.65-3.93)	0.305	0.79 (0.43-1.45)	0.445
130-159	0.53 (0.15-1.84)	0.318	0.57 (0.26-1.24)	0.157
≥160	0.27 (0.03-2.24)	0.225	0.48 (0.14-1.62)	0.235
Triglyceride (mg/dl)				
<149	Reference		Reference	
150-199	2.69 (1.17-6.20)	0.020*	1.18 (0.58-2.39)	0.651
200-499	0.51 (0.11-2.30)	0.378	1.40 (0.73-2.69)	0.307
≥500	11.19 (2.40-52.31)	0.002*	0.00 (0.00-0.00)	0.965
HDL Cholesterol ^a				
No	Reference		Reference	
Yes	1.10 (0.51-2.35)	0.809	2.38 (1.35-4.21)	0.003*
Waist circumference ^b				
No	Reference		Reference	
Yes	0.70 (0.25-1.96)	0.499	0.63 (0.27-1.46)	0.277
Blood pressure ^c				
No	Reference		Reference	
Yes	2.35 (1.00-5.50)	0.049*	0.83 (0.48-1.43)	0.502

Adjusted for demographic and socioeconomic information (age, education level, marital status, and income), lifestyle (drinking, smoking, and physical activity), and waist to height ratio (WHtR)

^aLess than 40 mg/dl for men and less than 50 mg/dl for women, or being medication treatment

^bMore than 90 cm for men and 85 cm for women

^cSystolic blood pressure of 130mmHg or more, diastolic blood pressure:85mmHg or more or on medication treatment

**P*< .05

Acknowledgements

This research was supported by Basic Science Research Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education (NRF-2017R1D1A3B03028701).

Conflict of interest

The authors declare that there is no conflict of interest.

References

1. Esposito K, Chiodini P, Colao A, Lenzi A, Giugliano D (2012). Metabolic syndrome and risk of cancer: a systematic review and meta-analysis. *Diabetes Care*, 35(11):2402-2411.
2. Lindkvist B, Almquist M, Bjørge T, et al (2013). Prospective cohort study of metabolic risk factors and gastric adenocarcinoma risk in the Metabolic Syndrome and Cancer Project (Me-Can). *Cancer Causes Control*, 24 (1):107-116.
3. Grundy SM, Cleeman JI, Daniels SR, et al (2005). Diagnosis and management of the metabolic syndrome: an American Heart Association/National Heart, Lung, and Blood Institute scientific statement. *Circulation*, 112 (17):2735-2752.
4. Lorenzi M, Montisano DF, Toledo S, Barrieux A (1986). High glucose induces DNA damage in cultured human endothelial cells. *J Clin Invest*, 77(1):322-325.
5. Meyer P (1987). Increased intracellular calcium: from hypertension to cancer. *J Hypertens Suppl*, 5(4):S3-4.
6. Eisenberg S (1984). High density lipoprotein metabolism. *J Lipid Res*, 25(10):1017-1058.