Journal of Asian Multicultural Research for Medical and Health Science Study
ISSN: 2708-972X
Vol. 5 No. 1, 2024 (page 001-006)
DOI: https://doi.org/10.47616/jamrmhss.v5i1.480

The Impact of Alcohol Consumption Duration and Types on Colorectal Cancer: Insights from a Cross-Sectional Study in Medan, Indonesia

Irma Yanti Rangkuti[1]

[1]Universitas Islam Sumatera Utara, Indonesia

Received: January 1, 2024 Received in Revised: February 2, 2024 Accepted: February 10, 2024

Abstract

Colorectal cancer is the third most common type of cancer in the world, in Indonesia it is in the fourth highest position. Nearly 1 million deaths a year were due to colorectal cancer. Alcohol increases the risk of developing colorectal cancer. This study aims to analyze differences in duration of alcohol consumption in people with and without colorectal cancer and to analyze the relationship between type of alcohol and the incidence of colorectal cancer. The research was carried out at the Digestive Surgery Polyclinic, H. Adam Malik Hospital, Medan. Using primary and secondary data involving 31 subjects. Subjects aged 30-80 years, 21 men, 10 women, 80.6% suffered from colorectal cancer. 28 people (90.3%) consumed type A alcohol the most. The mean duration of alcohol consumption in colorectal cancer sufferers and those without colorectal cancer was 14.76 ± 3.33 years and 11.00 ± 3.688 years. There is a difference in the duration of alcohol consumption in people with and without colorectal cancer, and the type of alcohol consumed is not related to the incidence of colorectal cancer.

Keywords: colorectal cancer, alcohol, consumption duration

Introduction

Colorectal cancer is a disorder of the colon or rectum due to uncontrolled proliferation of colon gland epithelial cells. Genetic and environmental factors determine the development of colorectal cancer (Hossain et al., 2022). Currently colorectal cancer is the third most common type of cancer in the world according to Global Burden of Cancer (GLOBOCAN) data released by the World Health Organization (WHO) in 2020 with more than 1.9 million cases after breast cancer and lung cancer. Meanwhile, deaths caused by colorectal cancer are in second place with 935,173 deaths a year (WHO, 2020). New cases of colorectal cancer in Indonesia were recorded at 33,427, with a death rate of 17,786 (WHO, 2021).

Developing countries have the highest risk of suffering from colorectal cancer with a sedentary lifestyle, obesity, high consumption of red meat, drinking alcohol and smoking, adopting a western lifestyle (Arnold et al., 2017; Sawicki et al., 2021; Wong et al., 2021). Data from the Central Statistics Agency shows that alcohol consumption by residents aged ≥ 15 years in the last year was 0.33 liters per capita (Badan Pusat Statistik Indonesia, 2022).

Recent studies have clearly proven that alcoholism is associated with many types of cancer (Varghese & Dakhode, 2022). Multi-ethnic research on whites, Hawaiians, Latinos, Americans and Japanese shows that alcohol consumption is associated with the risk of colorectal cancer (Park et al., 2019). Other research also showed that alcoholics are at high risk of suffering from colorectal cancer (Lin et al., 2020). A meta-analysis study conducted by Bagnardi et al. showed there was an association of cancers and levels of alcohol drinking, which was represented by relative risk of cancer with intake of levels of alcohol, ranged from 1.14 to 8.32 (Bagnardi et al., 2015). Based on ethanol concentration, alcoholic drinks are divided into types A, B and C (Indonesia, 2012). Colorectal cancer is a chronic disease associated with the duration of alcohol

consumption so researchers want to analyze the differences in the duration of alcohol consumption in colorectal and non-colorectal cancer sufferers as well as the relationship between the type of alcohol and the incidence of colorectal cancer. The research was carried out at H. Adam Malik Hospital Medan, a type A hospital in North Sumatra. Through this study, it is hoped that the public can take steps to prevent the incidence of colorectal cancer by stopping alcohol consumption as soon as possible.

Methods

The study used a cross sectional design. The research subjects were 31 people. The sampling technique is non purposive sampling. This research uses primary and secondary data. Primary data is the results of interviews with patients registered in the medical records of the digestive surgery polyclinic at H. Adam Malik General Hospital, Medan in 2020-2022. Interviews with patients consist of questions regarding the duration of alcoholic beverage consumption and the type of alcoholic beverage consumed. Secondary data in this study is medical record data of patients registered at the H. Adam Malik Medan digestive surgery clinic in 2020-2022 at H. Adam Malik Hospital Medan.

The research variables consisted of duration of alcohol consumption (numerical data) and type of alcohol (categorical data) as independent variables and the dependent variable was the incidence of colorectal cancer (categorical data).

Data were analyzed univariately and bivariately, and data normality tests were carried out. Differences in duration of alcohol consumption between groups of subjects suffering from colorectal cancer and those without colorectal cancer were analyzed using the unpaired T test. The relationship between types of alcohol and the incidence of colorectal cancer was tested using the Chi-Square test.

Results and Discussion

This study shows the sociodemographics of the research subjects, differences in duration of alcohol consumption in groups suffering from colorectal cancer and those without colorectal cancer as well as the relationship between the type of alcohol and the incidence of colorectal cancer.

Sociodemography

The research subjects were 31 people, the majority were men (67.7%), average age 57.13 ± 11.436 years, most consumed type A alcohol (28%), and 25 people (80.6%) suffered from cancer. Colorectal (Table 1).

Table 1. Sociodemographics of research subjects

Characteristics	F	Percentage (%)
Sex		
Man	21	67.7
Woman	10	32.3
Age (years)		
Min 30		
Max 80		
Average (mean ± SD) years : 57,13 ± 11,436		
Type of alcohol		
A	28	90,3
B	2	6,5
C	1	3,2

Colorectal cancer		
Yes	25	80,6
No	6	18,4

Differences in duration of alcohol consumption in groups suffering from colorectal cancer and those not suffering from colorectal cancer

Before carrying out the difference test, the normality of the data is first tested. The normality test shows that the data is normally distributed, $p > 0.05$ (Table 2).

Table 2. Data normality test using the Shapiro-Wilk Test

Group	Statistic	P
Colorectal cancer	0,952	0,285
No Colorectal cancer	0,924	0,537

Because the data were normally distributed, differences in duration of alcohol consumption between groups suffering from colorectal cancer and those without colorectal cancer were analyzed using the unpaired T test, $p = 0.021$. There is a difference in the duration of alcohol consumption in groups who suffer from colorectal cancer and those who do not. The average duration of alcohol consumption in subjects suffering from colorectal cancer and those not suffering from colorectal cancer was 14.76 ± 3.333 years and 11.00 ± 3.688 years (Table 3).

Table 3. Cross tab of duration of alcohol consumption with the incidence of colorectal cancer and the p value of the unpaired T test

	Colorectal cancer	F	Average (Mean ± Std. deviatio)	P
Duration of alcohol consumption (years)	Ya	25	14.76 ± 3,333	0,021
	Tidak	6	11.00 ± 3,688	

Relationship between types of alcohol and the incidence of colorectal cancer

The relationship between the type of alcohol consumed and the incidence of colorectal cancer was tested using the Chi Square test, obtained $p = 0.476$ ($p > 0,05$). There was no significant relationship between the type of alcohol and the incidence of colorectal cancer (Dahlan, 2010) (Hastono, 2007) (Table 4). Type A alcohol was most frequently consumed in colorectal cancer sufferers and also in the group without colorectal cancer.

Table 4. Cross tab of types of alcohol with the incidence of colorectal cancer and the p value of the Chi-square test

		Colorectal cancer		Total	p
		Yes	No		
Type of alcohol	A	23	5	28	0,476
	B	1	1	2	
	C	1	0	1	
Total		25	6	31	

Alcoholic drinks are classified into several types according to the ethanol content contained in them, namely groups A, B and C. Class A alcoholic drinks contain 1-5% ethanol, for example, beer, palm wine, and others. Group B is a type of alcohol that contains more than 5% ethanol,

for example, sake, soju, wine, red wine, and others. Group C is a type of alcohol with an ethanol content of more than 20-55%, for example, tequila, vodka, rum, whiskey, and others (Indonesia, 2012).

Colorectal cancer is caused by many factors, around 10% of patients with colorectal cancer have a predisposition to family syndrome mutations such as Lynch syndrome and familial adenomatous polyposis. The development of colorectal cancer is caused by a combination of various factors including environmental influences, genetic susceptibility, and immune mechanisms. Environmental factors that contribute to an increased risk of colorectal cancer are a diet of processed/grilled red meat, fatty foods, low in fiber and folate. Other suspected risk factors are comorbidities such as obesity, inflammatory bowel diseases, type 2 diabetes, smoking, circadian rhythm disorders due to night shift work, and the presence of pathogenic microbiota (Johnson et al., 2015; Johnson et al., 2021).

Ethanol is broken down in the liver into acetaldehyde and acetate (Marchitti, Brocker, Stagos, & Vasiliou, 2008; Seitz & Stickel, 2007). Acetaldehyde, formed through various processes, can cause DNA mutations and oxidative stress in the colon, initiating cancer. It disrupts DNA synthesis and repair, induces mutations, and binds to proteins and DNA, forming stable DNA adducts. Chronic alcohol consumption contributes to cancer through direct and indirect mechanisms (Johnson et al., 2021; Rossi et al., 2018; Seitz & Stickel, 2007) .

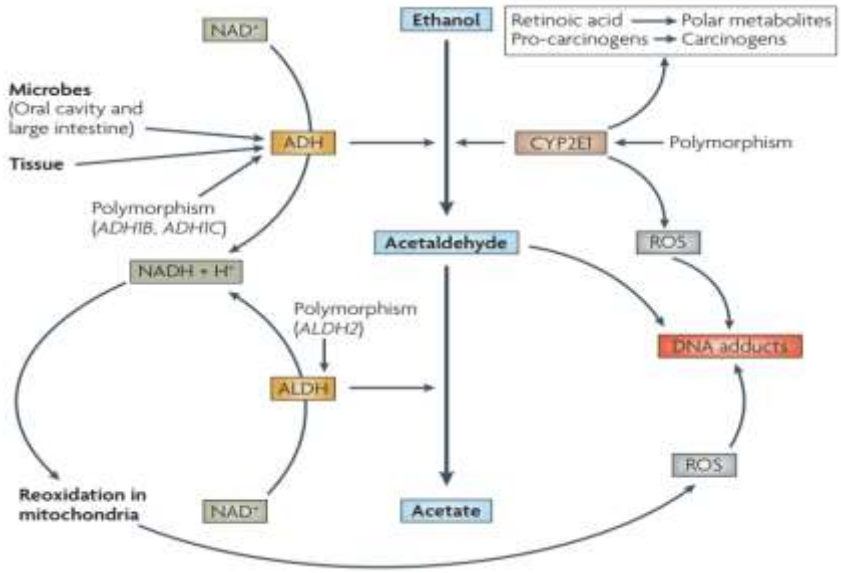

Figure 1. Ethanol metabolism and its involvement in carcinogenesis (Seitz & Stickel, 2007)

Locally, ethanol affects the colorectal mucosa, disrupts the local microbiome, and induces tissue inflammation. Ethanol increases susceptibility to carcinogenesis by activating enzymes that produce procarcinogens, altering carcinogen metabolism, and interfering with DNA repair (Johnson et al., 2021; Seitz & Stickel, 2007). Single nucleotide polymorphisms in specific genes lead to individual variations in acetaldehyde production and oxidation, influencing cancer risk. Polymorphisms in the CYP2E1 enzyme and its increased activity, induced by chronic ethanol consumption, play a role in ROS formation and the activation of various procarcinogens (Seitz & Stickel, 2007).

Furthermore, acetate, a metabolite of ethanol, is linked to colorectal cancer incidence as it is transformed into Acetyl Co-A, a crucial metabolite for cancer cell growth, particularly under hypoxic conditions. The large amounts of acetate produced during chronic alcohol

consumption can fuel the formation of colorectal cancer cells, especially during hypoxia and fasting (Kamphorst et al., 2014; Yoshii et al., 2015).

Conclusion and Suggestions

The findings from the comprehensive analysis presented in this article indicate that the duration of alcohol consumption diverges between individuals with colorectal cancer and those without, while the spesific type of alcohol consumed does not appear to be linked to the onset of colorectal cancer. Given the detrimental effects of alcohol on overall health, particularly digestive well-being, it is advisable to steer clear of its consumption for the sake of mantaining a healthy body.

Acknowledgments

We would like to thank to director of Rumah Sakit H. Adam Malik Sumatera Utara who permitted our research and for institution Fakultas Kedokteran Universitas Islam Sumatera Utara for facilitating the administration of this study.

References

Arnold, M., Sierra, M. S., Laversanne, M., Soerjomataram, I., Jemal, A., & Bray, F. (2017). Global patterns and trends in colorectal cancer incidence and mortality. *Gut*, *66*(4), 683–691.

Badan Pusat Statistik Indonesia. (2022). *Konsumsi Alkohol Oleh Penduduk Umur ≥ 15 Tahun Dalam Satu Tahun Terakhir (Liter Per Kapita), 2020-2022*.

Bagnardi, V., Rota, M., Botteri, E., Tramacere, I., Islami, F., Fedirko, V., Scotti, L., et al. (2015). Alcohol consumption and site-specific cancer risk: A comprehensive dose-response meta-analysis. *British Journal of Cancer*, *112*(3), 580–593. Nature Publishing Group. Retrieved from http://dx.doi.org/10.1038/bjc.2014.579

Dahlan, M. S. (2010). *Mendiagnosis dan Menatalaksana 13 Penyakit Statistik: Disertai Aplikasi Program Stata*. Jakarta: Sagung Seto.

Hastono, S. P. (2007). *Analisis Data Kesehatan*. Jakarta: Universitas Indonesia.

Hossain, M. S., Karuniawati, H., Jairoun, A. A., Urbi, Z., Ooi, D. J., John, A., Lim, Y. C., et al. (2022). Colorectal Cancer : A Review of Carcinogenesis , Global. *Cancer*, *14*(1732), 1–25.

Indonesia, M. P. R. (2012). Peraturan Menteri Perindustrian Republik Indonesia Tentang Pengendalian dan Pengawasan Industri Minuman Beralkohol.

Johnson, C. H., Dejea, C. M., Edler, D., Hoang, L. T., F., A., Santidrian, Felding, B. H., et al. (2015). Metabolism Links Bacterial Biofilms and colon Carcinogenesis. *Cell Metabolism*, *21*(6). Retrieved from http://europepmc.org/backend/ptpmcrender.fcgi?accid=PMC5604322&blobtype=pdf

Johnson, C. H., Golla, J. P., Dioletis, E., Singh, S., Ishii, M., Charkoftaki, G., Thompson, D. C., et al. (2021). Molecular mechanisms of alcohol-induced colorectal carcinogenesis. *Cancers*, *13*(17), 1–17.

Kamphorst, J. J., Chung, M. K., Fan, J., & Rabinowitz, J. D. (2014). Quantitative analysis of acetyl-CoA production in hypoxic cancer cells reveals substantial contribution from acetate. *Cancer & Metabolism*, *2*(1), 1–8.

Lin, T. C., Chien, W. C., Hu, J. M., Tzeng, N. S., Chung, C. H., Pu, T. W., Hsiao, C. W., et al. (2020). Risk of colorectal cancer in patients with alcoholism: A nationwide, population-

based nested case-control study. *PLoS ONE*, *15*(5), 1–12. Retrieved from http://dx.doi.org/10.1371/journal.pone.0232740

Marchitti, S. A., Brocker, C., Stagos, D., & Vasiliou, V. (2008). Non-P450 Aldehyde Oxidizing Enzymes: The Aldehyde Dehydrogenase Superfamily. *Expert Opin Drug Metab Toxicol*, *4*(6), 1–7. Retrieved from https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3624763/pdf/nihms412728.pdf

Park, S. Y., Wilkens, L. R., Setiawan, V. W., Monroe, K. R., Haiman, C. A., & Le Marchand, L. (2019). Alcohol Intake and Colorectal Cancer Risk in the Multiethnic Cohort Study. *American Journal of Epidemiology*, *188*(1), 67–76.

Rossi, M., Anwar, M. J., Usman, A., Keshavarzian, A., & Bishehsari, F. (2018). Colorectal cancer and alcohol consumption—populations to molecules. *Cancers*, *10*(2).

Sawicki, T., Ruszkowska, M., Danielewicz, A., Nied´zwiedzka, E., Przybyłowicz, T. A., & E., K. (2021). A Review of Colorectal cancer in Terms of Epidemiology, Risk Factors, Development, Symptoms and Diagnosis. *Cancers*, *13*(2025), 1–23.

Seitz, H. K., & Stickel, F. (2007). Molecular mechanisms of alcohol-mediated carcinogenesis. *Nature Reviews Cancer*, *7*(8), 599–612.

Varghese, J., & Dakhode, S. (2022). Effects of Alcohol Consumption on Various Systems of the Human Body: A Systematic Review. *Cureus*, *14*(10), 8–13.

WHO. (2020). Colorectal Cancer. *International Agency for Research on Cancer*.

WHO. (2021). Cancer incident in Indonesia. *International Agency for Research on Cancer*.

Wong, M. C. S., Huang, J., Lok, V., Wang, J., Fung, F., Ding, H., & Zheng, Z. J. (2021). Differences in Incidence and Mortality Trends of Colorectal Cancer Worldwide Based on Sex, Age, and Anatomic Location. *Clinical Gastroenterology and Hepatology*, *19*(5), 955-966.e61. Elsevier, Inc. Retrieved from https://doi.org/10.1016/j.cgh.2020.02.026

Yoshii, Y., Furukawa, T., Saga, T., & Fujibayashi, Y. (2015). Acetate/acetyl-CoA metabolism associated with cancer fatty acid synthesis: Overview and application. *Cancer Letters*, *356*(2), 211–216. Elsevier Ireland Ltd. Retrieved from http://dx.doi.org/10.1016/j.canlet.2014.02.019