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The analysis of cell damage of liver and kidney among alcoholics in Yogyakarta, Indonesia

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ABSTRACT

Alcohol tends to disrupt the organs function of human body, even can cause serious and chronic damage. In Indonesia, the data on cell damage taken from organs including the livers and kidneys among alcoholics are still practically unknown. The aim of this study was to compare the differences of organs' cell disturbance between alcoholics and non-alcoholics in Yogyakarta, Indonesia. A crosssectional study was conducted among 197 people in Yogyakarta, including 96 alcoholics and 101 non-alcoholics. The material of the study was taken from venous blood samples. A kinetic photometric test was conducted to obtain data on blood-chemical markers' value of livers (SGOT, SGPT, and GGT), and kidneys (BUN and serum creatinine). The data werethen analyzed by Chi square test.From a total of 96 alcoholics, 83.6% are males and 16.4% are females who consumed alcohol for an average of 16 years, with 51.6% routinely consuming it daily. The kind of alcohol they consumed was single-brand (59.8%). Based on 25th percentile (GGT), on 50th percentile (SGOT, GGT), and on 75th percentile (SGPT, GGT), the alcoholics had higher proportion value of liver marker disturbance than non-alcoholics which was statistically significant ($p<0.05$). Based on the 50th and 75th percentiles, the alcoholics also had higher proportion value on kidney marker (BUN) damage than non-alcoholics which was statistically significant ($p<0.05$). There were significant differences in GFR values between males and females ($p<0.05$), males had kidney cell damage 7.9 times more than females. There was no significant difference in the value of GFR between alcoholics and non-alcoholics. In conclusion, the alcoholics hassignificantly higher proportion value of blood-chemical markers than that non-alcoholics. The GFR values between males and females are also significantly different, and males had kidney cell damage 7.9 times more than that of females.

ABSTRAK

Alkohol dapat merusak fungsi organ tubuh manusia, bahkan dapat menyebabkan kerusakan serius dan kronik. Di Indonesia, data kerusakan sel yang diambil dari organ hati dan ginjal pada alkoholik tidak diketahui. Tujuan penelitian ini adalah membandingkan perbedaan gangguan sel pada organ antara alkoholik dan non-alkoholik di Yogyakarta, Indonesia. Studi potong lintang dilakukan pada 197 orang di Yogyakarta, terdiri dari 96 alkoholik dan 101 non-alkoholik. Material diambil dari sampel darah vena. Tes fotometrik kinetik dilakukan untuk mendapatkan data nilai penanda kimia darah pada hati (SGOT, SGPT, dan GGT), dan ginjal (BUN dan kreatinin serum). Data kemudian dianalisis menggunakan uji Chi square.Dari total 96 alkoholik, 83,6% adalah laki-laki dan 16,4% adalah perempuan, yang rata-rata mengkonsumsi alkohol selama 16 tahun, dan 51,6% rutin mengkonsumsi tiap hari. Macam alkohol yang dikonsumsi adalah satu merk (59,8%). Berdasar 25 persentil (GGT), 50persentil (SGOT, GGT), dan 75 persentil (SGPT, GGT), alkoholik mempunyai proporsi yang lebih tinggi dan bermakna secara statistik terhadap gangguan nilai penanda hepar dibandingkan non-alkoholik ($p<0.05$). Berdasar 50 dan 75 persentil, alkoholik juga mempunyai nilai proporsi yang lebih tinggi secara bermakna($p<0.05$) pada penanda kerusakan ginjal (BUN) dibandingkan non- alkoholik, Terdapat perbedaan bermakna nilai GFR antara laki-laki dan perempuan ($p<0.05$). Laki-laki mempunyai kerusakan sel ginjal 7.9 kali lebih besar dibandingkan perempuan. Tidak terdapat perbedaan bermakna pada nilai GFR antara alkoholik dan non-alkoholik. Dapat disimpulkan bahwa alkoholik mempunyai proporsi nilai penanda kimia darah lebih tinggi secara bermakna dibandingkan non- alkoholik. Nilai GFR antara laki-laki dan perempuan juga berbeda bermakna, dan laki-laki mempunyai kerusakan sel ginjal 7.9 kali dibandingkan perempuan.

Keywords:
alcohol
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INTRODUCTION

Drinking alcohol can increase the risk of developing liver disease and cause damage to important parts of the body. Alcohol is the leading cause of the 25% increase in deaths from liver disease in the UK over the past decade (from 9231 in 2001 to 11,575 in 2009). Liver disease has become more prevalent in the younger population, with more than one in ten deaths of people at age 40, and most of them are alcohol-related.[1]

Among the 228,864,000 people in the Indonesian population, 72% are above 15 years old. From this age group, there are about 21.9% of males and 3% of females who have consumed alcohol. Accumulatively, they account for about 12.3% of the population above 15 years old. In 2001, the number of males and females who drink excessive alcohol, consuming at least 60 grams of pure alcohol per week, accounted for 7.3% and 0.0% of the population, respectively. Total amount of alcohol consumption per capita is 4.47 liters of pure alcohol from 2003-2005. From morbidity data, the number of alcohol-related diseases in the adult population in Indonesia in 2004 was 0.61% (male) and 0.08% (female). Alcohol continues to kill more than 3.3 million people worldwide every year. The death rate due to alcohol consumption is far above the death rate of AIDS, TB, and violence victims combined.[1]

Indonesians, who are mostly Muslim, do not have an alcohol consumption culture.However, in reality,there are some ethnicities in Indonesia that have traditional alcoholic beverages. Yogyakarta, as a student, cultural, and tourism city, in an effort to improve local revenue, also experiences alcohol abuse. Alcohol abuse results in many victims being brought to the hospital after a liquor party (from abusing alcohol), causing even death. In February 2016 in the Sleman region, 26 people died due to mixed alcohol *oplosan* (a mixed liquid containing methanol), and two of them when examined at Dr. Sardjito General Hospital, Yogyakarta had positive alcohol blood results. The impact of alcohol abuse clearly undermines the nation's future because as work productivity declines, health will also deteriorate and eventually burden the country's economy.[2]

Most of the deaths associated with alcohol consumption are caused by injury from accidents, cancer, cardiovascular diseases, and liver cirrhosis. The liver is the most susceptible organ to alcohol-induced damage.Alcoholic liver disease is mainly caused by excessive alcohol consumption.[3,4] Alcohol abuse in Indonesia, in addition to causing death from overdose or drinking *oplosan*, could also damage liver cells, causing cirrhosis of the liver and eventually, death. Even thoughthere are plenty of alcohol abuse cases in Indonesia, only few researchers have studied the damaging effects of alcohol on liver and kidney cells. In response to this public health concern, this study aimed to study the liver and kidney cells' damage in alcoholics in Yogyakarta.[5]

MATERIALS AND METHODS

Subjects

From November 2014 to July 2015, we conducted a cross-sectional analytic study. Ninety-six alcoholics and 101 non-alcoholic adults of Javanese ethnic participated in the study. Before collecting the data, all participants were asked to sign informed consent forms. The study had been approved by the Medical and Health Research Ethics Committee of the Faculty of Medicine, Public Health and Nursing, Universitas Gadjah Mada in Yogyakarta. Questionnaires identified and listed all subject characteristics such as gender, age, medical history, and alcohol drinking history referring

to Alcohol Dependence Score (ADS) instructions.

Determination of liver and kidney cell damage

We collected 3 mL of blood samples from each participant. Blood chemistry analysis of Serum Glutamic Oxalocetic Transaminase (SGOT), Serum Glutamic Pyruvic Transaminase (SGPT) and Gamma Glutamyl Transferase (GGT) assessed liver cell damages.[6] We used Diasys ALAT (GPT) FS reagents for SGPT levels, Diasys ASAT (GOT) FS reagents for SGOT levels, Diasys Gamma-GT FS reagents for GGT levels to measure serum liver enzyme levels. Measurement of SGOT, SGPT, GGT levels using these reagents was diagnostic for quantitative in vitro measurement in serum or plasma with photometric systems. Optimized UV-test method used was based on the International Federation of Clinical Chemistry and Laboratory Medicine.

Serum urea and creatinine measurements required serum samples or plasma heparin. We collected 3-5 mL of venous blood on a red or green covered tube (heparin) to avoid hemolysis. We centrifuged the blood then separated the serum / plasma for examination. Measurement of serum urea level used DiaSys Urea FS reagent, while serum creatinine level used DiaSys Creatinine FS reagent. Levels of urea (BUN) and creatinine were measured by colorimetric methods using a photometer or chemical analyzer.[7,8]

Data analysis

Depending on the types of data, the data were presented as mean ± standard deviation (SD) or as median (minimum-maximum). We analyzed the differences between blood chemistry percentile values of SGOT, SGPT, GGT, BUN, and creatinine between groups using unpaired t-test. The differences between groups were considered statistically significant if pvalue < 0.05.

RESULTS

The results showed that there aresignificant differences in age and sex between the alcoholic and non-alcoholic groups, with p-values of 0.025 and 0.001, respectively. Data are presented in TABLE 1.

TABLE 1. Age and sex of alcoholics and non-alcoholicsJavanese people in Yogyakarta

Variable	Alcoholic	Non-alcoholic	p
Age (mean±SD year)	44.27±13.57	48.73±14.29	0.025
Sex n (%)			
• Male	83 (66.4)	42 (33.6)	0.001
• Female	14 (18.7)	61 (81.3)	
Total n (%)	97 (48.5)	103 (51.5)	

Note: SD = standard deviation; p < 0.05 = significant difference

We presented the characteristics of alcoholics in this study in TABLE 2. Most alcoholics (83.5%) were male, 51.6% had daily consumption, and 59.8% consumed non-mixed alcohol.

TABLE 2. Characteristics of alcoholics with Javanese ethnic(n=97) in Yogyakarta, Indonesia

Variable	Alcoholics n (%)
Gender	
• Male	81 (83.5)
• Female	16 (16.5)
Age range (year)	
• Minimum	19
• Maximum	69
Age group based on Hurlock criteria	
• Adolescent (14-21 years)	3 (3.1)
• Adult (22-60 year)	81 (83.5)
• Elderly (>60 year)	13 (13.4)
Duration of alcohol consumption (year)	
• Minimum	1
• Medium	16
• Maximum	49
Frequency of alcohol consumption	
• Days	49 (51.6)
• Months	31 (32.6)
• Years	9 (9.5)
• Occasionally	6 (6.3)
Alcohol type	
• Mixed	39 (40)
• Non-mixed	58 (59.8)

The relationship between age and duration of alcohol consumption is presented in FIGURE 1. There was a significant relationship between age and duration of alcohol consumption ($p< 0.001$).

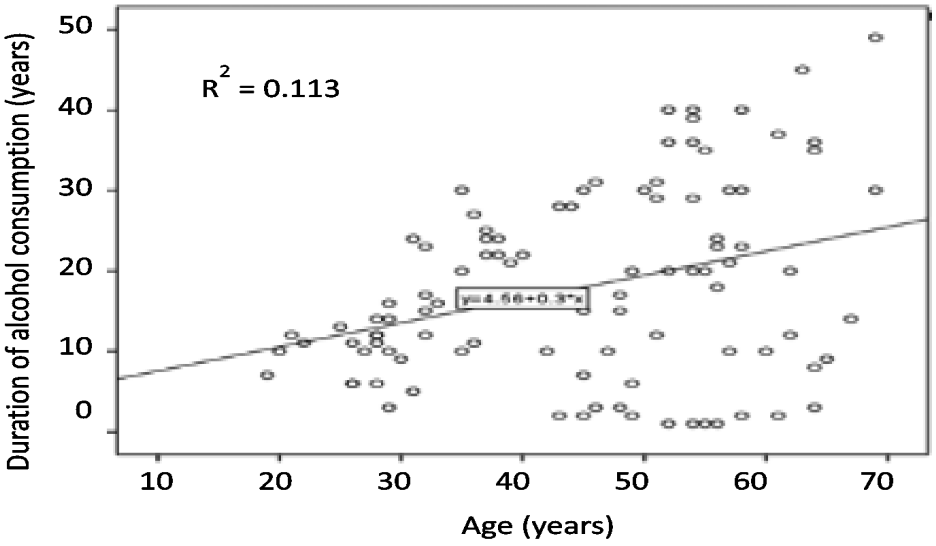

FIGURE 1. Relations between age and alcohol consumption duration

The SGOT blood chemistry percentile values of alcoholics and non-alcoholics are presented in TABLE 3. The SGOT 50% percentile value showed significant differences between alcoholics and non-alcoholics (p = 0.004). Percentile values of 25% SGOT and 75% SGOT did not show significant differences with p-values of 0.253 and 0.196, respectively.

The SGPT blood chemistry percentile values in alcoholics and non-alcoholics are presented in TABLE 4. Percentile value of 75% SGPT showed significant differences between alcoholics and non-alcoholics (p=0.032). Percentile values of 25% SGOT and 50% SGPT did not show any significant differences with p-values of 0.429 and 0.138, respectively. Percentile values of GGT blood chemistry for alcoholics and non-alcoholics are presented in TABLE 5. Percentile values of 25%, 50%, and 75% GGT showed significant differences between alcoholics and non-alcoholics with p-values of 0.036, 0.019, and 0.006, respectively.

TABLE 3. The percentile value of means of SGOT blood chemistry between alcoholics and non-alcoholics

Variable	SGOT-25		Total n (%)	p	OR
	≤ 18 n (%)	> 18 n (%)			
Alcoholic	77(39.1)	19(9.6)	96(48.7)		
Non-Alc	76(38.6)	25(12.7)	101(51.3)	>0.05	1.333
Total	153(77.7)	44(22.3)	197(100.0)		
	SGOT-50				
	≤ 21 n (%)	> 21 n (%)			
Alcoholic	59(58.4)	37(38.5)	96(48.7)		
Non-Alc	42(41.6)	59(61.5)	101(51.3)	<0.05	2.240
Total	101(51.3)	96(48.7)	197(100.0)		
	SGOT-75				
	≤ 25 n (%)	> 25 n (%)			
Alcoholic	30 (54.5)	66(46.5)	96(48.7)		
Non-Alc	25(45.5)	76(53.5)	101(51.3)	>0.05	1.382
Total	55(27.9)	142(72.1)	197(100.0)		

TABLE 4. Percentile value of means of SGPT blood chemistry among alcoholics and non-alcoholics

Variable	SGPT-25		Total n (%)	p	OR
	≤ 13 n (%)	>13 n (%)			
Alcoholic	78(39.6)	18(9.1)	96(48.7)		
Non-Alc	80(40.6)	21(10.7)	101(51.3)	>0.05	1.138
Total	158(80.2)	39(19.8)	197(100.0)		
	SGPT-50				
	≥ 16 n (%)	< 16 n (%)			

Alcoholic	55(27.9)	41(20.8)	96(48.7)		
Non-Alc	49 (24.9)	52(26.4)	101(51.3)	>0.05	1.424
Total	104(52.8)	93(47.2)	197(100.0)		
	SGPT-75				
	≤ 21.5 n (%)	> 21.5 n (%)			
Alcoholic	30 (15.2)	66(33.5)	96(48.7)		
Non-Alc	19(9.6)	82(41.6)	101(51.3)	<0.05	1.962
Total	49(24.9)	148(75.1)	197(100.0)		

TABLE 5. Mean percentile value of GGT blood chemistry between alcoholics and non-alcoholics

Variables	GGT-25		Total n (%)	p	OR
	≤ 15 n (%)	>15 n (%)			
Alcohol	85 (43.1)	11(5.6)	96(48.7)		
Non-Alc	78 (39.6)	23(11.7)	101(51.3)	<0.05	2.279
Total	163(82.7)	34 (17.3)	197(100.0)		
	GGT-50				
	≥ 20 n (%)	< 20 n (%)			
Alcohol	56(28.4)	40(20.3)	96(48.7)		
Non-Alc	43(21.8)	58(29.4)	101(51.3)	<0.05	1.888
Total	99(50.3)	98(49.7)	197(100.0)		
	GGT -75				
	≤ 28.5 n (%)	> 28.5 n (%)			
Alcohol	32(28.4)	64(20.3)	96(48.7)		
Non-Alc	17 (8.6)	84 (42.6)	101(51.3)	<0.05	2.471
Total	49(24.9)	148(75.1)	197(100.0)		

Percentile values of BUN blood chemistry between alcoholics and non-alcoholics are presented in TABLE 6. Percentile values of 50% and 75% BUN showed significant difference between alcoholics and non-alcoholics with p-values of 0.027 and 0.003, respectively. Percentile value of 25% BUN showed no significant difference with p-value of 0.303. Percentile values of creatinine blood chemistry between alcoholics and non-alcoholics are presented in TABLE 7. Percentile values of 25%, 50%, and 75% creatinine showed no significant differences between alcoholics and non-alcoholics with p-values of 0.742, 0.513, and 0.091, respectively.

The values of GFR in alcoholics and non-alcoholics by sex are presented in TABLE 8. There was significant difference in the value of GFR between alcoholics and non-alcoholics by sex, with pvalue 0.001. Male alcoholics tended to have damaged kidney cells 7.9 times greater than that of females.

TABLE 6. Mean percentile value of BUN blood chemistry between alcoholics and non-alcoholics

Variables	BUN-25		Total n (%)	p	OR
	≤ 26.745 n (%)	> 26.745 n (%)			
Alcohol	69(35.0)	27(13.7)	96(48.7)		
Non-Alc	79(40.1)	22(11.2)	101(51.3)	>0.05	0.712
Total	148(75.1)	49(24.9)	197(100.0)		
	BUN-50				
	≥ 34.27 n (%)	< 34.27 n (%)			
Alcohol	56(28.4)	40(20.3)	96(48.7)		
Non-Alc	43(21.8)	58(29.4)	101(51.3)	<0.05	1.888
Total	99(50.3)	98(49.7)	197(100.0)		
	BUN-75				
	≤ 39.36 n (%)	> 39.36 n (%)			
Alcohol	33(16.7)	63(32.0)	96(48.7)		
Non-Alc	16(8.2)	85(43.1)	101(51.3)	<0.05	2.783
Total	49(24.9)	148(75.1)	197(100.0)		

TABLE 7. Percentile value of mean of creatinine between alcoholics and non-alcoholics

Variables	Creatinine -25		Total n (%)	p	OR
	≤ 0.82 n (%)	> 0.82 n (%)			
Alcohol	76(38.6)	20(10.1)	96(48.7)		
Non-Alc	78 (39.6)	23(11.7)	101(51.3)	>0.05	1.121
Total	154(78.2)	43(21.8)	197(100.0)		
	Creatinine -50				
	≥ 0.90 n (%)	< 0.90 n (%)			
Alcohol	52(26.4)	44(22.3)	96(48.7)		
Non-Alc	50(25.4)	51(25.9)	101(51.3)	>0.05	1.205
Total	102(51.8)	95(48.2)	197(100.0)		
	Creatinine-75				
	≤ 0.995 n (%)	> 0.995 n (%)			
Alcohol	29(14.7)	67(34.0)	96(48.7)		
Non-Alc	20(10.2)	81(41.1)	101(51.3)	>0.05	1.753
Total	49(24.9)	148(75.1)	197(100.0)		

TABLE 8. GFRdata in alcoholics and non-alcoholics

GFR criteria*	Gender	Alcoholic n (%)	Non-alcoholics n (%)	Total n (%)	p	OR
>90	Sex					
	• Male	54(48.6)	28(25.2)	82(73.9)		
	• Female	3(2.7)	26(23.4)	29(26.1)	<0.05	16.7
	Total	57(51.4)	54(48.6)	111(100.0)		
60-89	Sex					
	• Male	20(31.7)	12(19.1)	32(50.8)		
	• Female	5(7.9)	26(41.3)	31(49.2)	<0.05	8.7
	Total	25(39.7)	38(60.3)	63(100.0)		
45-59; 30-44	Sex					
	• Male	7(41.2)	3(17.6)	10(58.8)		
	• Female	3(17.6)	4(23.6)	7(41.2)	>0.05	3.1
	Total	10(58.8)	7(41.2)	17(100.0)		
15-29	Sex					
	• Male	1(20.0)	0(0.0)	1(20.0)		
	• Female	3(60.0)	1(20.0)	4(80.0)	>0.05	1.3
	Total	4(80.0)	1(20.0)	5(100.0)		
<15 or ondialysis	Sex					
	• Male	0(0.0)	1(100.0)	1(100.0)		
	• Female	0(0.0)	0(0.0)	0(0.0)		
	Total	0(0.0)	1(100.0)	1(100.0)		
Total	Sex					
	• Male	82(41.6)	43(21.8)	125(63.5)		
	• Female	14(7.1)	58(29.4)	72(36.5)	<0.05	7.9
	Total	96(48.7)	101(51.3	197(100.0)		

*GFR criteria: >90 (normal kidney function); 60-89 (mildly reduced kidney function); 45-59; 30-44 (moderately reduced kidney function); 15-29 (severely reduced kidney function); <15 or on dialysis (very severe, or end-stage kidney failure)

TABLE 9. GFRdata of alcoholics and non-alcoholics

GFR criteria*	Alcoholics n (%)	Non-Alcoholics n (%)	Total n (%)	p
>90	57(28.5)	54(27.0)	111(55.5)	
60-89	24(12.0)	36(18.0)	60(30.0)	
45-59; 30-44	13(6.5)	8(4.0)	21(10.5)	>0.05
15-29	0(0.0)	1(0.5)	1(0.5)	
<15 or on dialysis	3 (1.5)	4 (2.0)	7 (3.5)	
Total	97(48.5)	103(51.5)	200(100.0)	

*GFR criteria: >90 (normal kidney function); 60-89 (mildly reduced kidney function); 45-59; 30-44 (moderately reduced kidney function); 15-29 (severely reduced kidney function); <15 or on dialysis (very severe, or end-stage kidney failure)

The values of Glomerular Filtration Rate (GFR) in alcoholics and non-alcoholics are presented in TABLE 9. There was no significant difference in the value of GFR between alcoholics and non-alcoholics, with *p*-value 0.326.

DISCUSSION

The alcoholics from this study were mostly male (TABLE 1). Males have 8.176 times greater frequency than females to drink alcohol, with confidence interval of 95% between 4.079 and 16.386. This finding was probably due to the fact that males have more outdoor activity, thus they were more vulnerable of being influenced by negative environmental factors. This study was in accordance to the data from the Basic Health Research (*Riset Kesehatan Dasar*) which showed that former alcoholic rates in Indonesia for males and females were 21.9% and 3%, respectively.[9] Male alcoholics also have 7.4% higher burden of disease ratio related to alcohol compared to female alcoholics who only have 1.4%.[4]

From 97 participants (100%), we obtained 16 female participants (16.5%). This implied that alcohol drinking behavior was spreading in Javanese society. When we conducted sampling, we found an alcoholic couple, a husband and wife, who happened to have a breastfeeding toddler. This finding was a very concerning condition because alcohol not only can influence the alcoholics, but also their children, the future generation of Indonesia. The government has attempted to tackle the problems of abuse of alcohol with a number of regulations for alcohol. However, the Presidential Regulation 2013 was limited to the supervision of alcohol distribution.[10]

Data from the Ministry of Health Republic of Indonesia (2009) stated that most alcoholics were male. It could be implied that environmental factors contributed to the abuse of alcohol. From our interviews, we found that some alcoholics would only drink on Saturday night or in a celebratory event, while others who make a living on the street such as some street performers, beggar or homeless people, have difficulties avoiding alcohol abuse.

Most of the alcohol was non-combined (59.8%), as shown in TABLE 2. Obtaining the amounts of alcohol was difficult because most of the time, the alcoholics drank their alcohol together. This presented as a limitation for our study. This result was different compared to studies conducted in western countries with a high level of per capita consumption.[4] Although blood chemistry of 25% and 75% SGOT values showed no significant differences between alcoholics and non-alcoholics, it was shown that there was a greater tendency of liver damage for alcoholics (1.333-1.382 times higher) compared to non-alcoholics. As shown in TABLE 3, it could be explained that there was a tendency of liver cell damage in adult age, explaining the relationship between age of starting drinking and the duration of drinking as shown in FIGURE 1. There was a significant relationship between age and duration of alcohol drinking in which older age was associated with longer duration of alcohol drinking. Blood chemistry value of 50% SGOT showed significant difference between alcoholic and non-alcoholic samples.

As shown in TABLE 4 and TABLE 5, the blood chemistry values of 25% and 50% SGPT showed no significant differences between alcoholics and non-alcoholics. However, there was higher chance for alcoholics to suffer liver cell damage (1.138-1.424) compared to non-alcoholics. Blood chemistry value of GGT showed significant difference at percentile values of 25%, 50%, and 75%. This finding might imply that GGT was more sensitive than SGOT and SGPT in assessing liver cell damage.The most common causes of mild to moderate

increase in these liver enzymes (SGOT and SGPT) are fatty liver, alcohol abuse and other health problems such as diabetes mellitus and obesity. Chronic ethanol use is known to easily trigger an increase in serum GGT. Furthermore, there is a positive correlation between ethanol intake and serum GGT activity.

Serum SGOT and SGPT levels were elevated 2-3 times higher than normal values. In addition, -GT (gamma glutamyl transferase/GGT) and alkali phosphatase levels also showed ½ to 1 time higher than normal levels. SGOT and SGPT are among the transaminase enzyme group produced in the liver. These two indicators would elevate during liver damage process. Normal value of SGOT is 10-40 U/L for males and 7-35 U/L for females whereas normal value of SGPT is 15-40 U/L for males and 13-35 U/L for females. In addition, normal value of GGT for males is 9-49 U/L, while for females it is 9-45 U/L, with an ordinal scale.[11]

Gamma glutamyl transferase examination is the measurement of GGT level in blood. GGT is an enzyme found in most body tissues; however, it has higher concentration in the liver, bile ducts, and kidneys. GGT is produced by the bile system and presence in the blood vessels is a sensitive marker for bile duct damage and could be used to evaluate liver cell damage.A long-term and excessive alcohol intake can cause an increase in GGT levels. In alcoholics, serum GGT can help distinguish the patients with or without liver disease.

Alcohol cessation and its relation with GGT could be explained as follows: a) GGT level would decline into its normal level within 2-3 weeks after cessation.A study conducted by Allen *et al.*[12] showed that increased serum GGT levels are the most widely used indicator for alcohol abuse. GGT levels usually rise after several weeks of alcohol intake. When drinking alcohol stops for 2-6 weeks, generally GGT levels decrease to within the normal range, since the half-life of GGT is about 14-26 days;[12]b) It helps to differentiate individuals with or without liver disease and its relation toward the declining of GGT level. c) GGT value which was continuously abnormal without exposure of ethanol indicates liver disease, usually when GGT level was initially 8-10 times higher than normal value and the increase persisted after 6-8 weeks of alcohol cessation; and d) if the initial GGT level was only 2-3 times the normal value and it returned to normal after alcohol cessation, the individual might be considered negative from liver disease.[13]

The comparison of BUN level between alcoholics and non-alcoholics shows that there was a tendency for alcoholics to have higher BUN level. There are significant differences between alcoholics and non-alcoholics (TABLE 6). From the calculation of odds ratio, alcoholics have higher risk (1.888-2.783) of having higher BUN level. This finding was in contrast with the study by Somba[14] stating that there was an increase in creatinine level accompanied by normal BUN level. This might be due to different confounding factors such as obscurity of the type of alcohol and the frequency of alcohol consumption.[14,15]

Basically, alcohol consumption will negatively impact on the human body, including the kidneys.[16]This study obtained results showing that creatinine serum for alcoholics and non-alcoholics had no significant differences. However, alcoholics were having higher risk (1.121-1.753) to experience creatinine serum level escalation than non-alcoholics (TABLE 7).

The estimated GFR is the best test for measuring the level of kidney function and determining stages of kidney disease. The estimated GFR can be calculated from the results of the blood creatinine test, age, body size and gender. Estimated GFRs can be used to determine the progression of kidney disease

and help doctors to plan treatment. If the GFR is low, the kidneys are not functioning properly. Early detection of kidney disease gives better possibility of stopping its development.[17]The GFRs in this study were mostly normal (>90) at around 57%, and slightly affected kidney function (60-89) at approximately 25%. Statistically, the estimated GFR show a significant difference between alcoholics and non-alcoholics, by sex. Whereas the estimated GFRs based on sex showed that male alcoholics tend to have damaged kidney cells greater than females.

In the estimated normal GFR and mild renal cell damage, there were significant differences in males and females ($p<0.05$), with the tendency of renal cell damage in males 16.7 times and 8.7 times greater than females. In the estimated value of moderate and mild GFR cell damage, there were no significant differences in males and females ($p<0.05$), with a tendency towards kidney cell damage in males 3.1 times and 1.3 times greater than females. In this study, there was no severe renal cell damage in alcoholic drinkers, while among non-alcoholic drinkers there was 1, so that it cannot be analyzed. In total the GFR values of male alcohol drinkers reflect a tendency for damaged kidney cells 7.9 times greater than females (TABLE 8). The results of this study GFR values in alcoholics and non-alcoholics showed no significant differences (TABLE 9), in contrast to the research conducted by Chung *et al.*[18] showing that chronic alcoholic drinkers of South Taiwan's native Pai-Wan experienced the estimated GFR value which was significantly higher than non-alcoholic drinkers.[18]

Chi square statistical test measured correlation between duration of alcohol consumption and serum creatinine level. Results showed no significant differences between duration of alcohol consumption and serum creatinine level in subjects with history of alcohol consumption. It was also in accordance to the correlation of duration of alcohol consumption and BUN value. This might be due to difference in frequency of alcohol consumption, type of alcohol, amount of drink, or the way of consumption. However, this study lacked those aforementioned data.

Limitations of this research include the unavailability of ultrasonographic data to support the cell damage findings within the liver. This limitation occurs because the subjects were not treated inside any hospital facility. Besides, the inclusion and exclusion criteria were less specific encouraging the emergence of many confounding factors during the study. Hence, stratified random sampling was used to reduce the confounding factors. The addition of inclusion and exclusion factors and also the use of sampling method was sub-optimal due to the obstacles in recruiting participants. Implementation of standardized questionnaire was done in hope of completing the rest of the study, but we encountered many incomplete data, namely: type of alcohol consumed, frequency of alcohol consumption, and amount of alcohol consumed. This might be due to various obstacles such as communication barriers between interviewer and respondents and inability of the respondents in recollecting their history of alcohol consumption.

CONCLUSION

In conclusion, the alcoholics has significantly higher proportion value of blood-chemical markers than that non-alcoholics in Yogyakarta Special Region. The GFR values between males and females are also significantly different, and males had kidney cell damage 7.9 times more than that of females.

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