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Low Serum Anti Mullerian Hormone as a Risk Factor for Cardiovascular Disease in Child-Bearing Age Women

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Abstract

Introduction
Cardiovascular disease (CVD) is the leading cause of global death. Incidence of having CVD is influenced by general risk factors and gender-related risk (preeclampsia, miscarriage, premature birth, contraception and hormonal therapy). Women of childbearing age have lower risk of CVD than men. Anti Mullerian Hormone (AMH) can be used to predict CVD. This study aimed to know association between Serum AMH level with CVD.

Methods
This study was an observational analytic, cross-sectional study. Subjects study is women with childbearing age (18-44 years old) who doing laboratory examination from May until November 2024. The exclusion criteria were patients who under chemoradiation therapy, pregnancy and have history of fertility disorder such as polycystic ovarian syndrome (PCOS). Data was subjected to bivariate testung chi square and multivariate regression logistic with Statistical Package for the Sociak Sciences (SPSS) version 29.0.

Results
A total of 57 subjects were being analyzed. Bivariate analysis showed that Serum AMH (p<0,001) have correlation with CVD. Multivariate analysis shows that Serum AMH <0,69 ng/mL (p<0,001; RR=34,049) were independently associated with CVD.

Conclusions
Serum AMH <0,69 ng/mL (PR:34,05; 95% CI: 6,245–185,630, p=<0,001) increased risk of having CVD in women with childbearing age.

Keywords: Anti-Mullerian Hormone; Cardiovascular Disease; Child-Bearing Age Women; Risk Factor

Introduction

Cardiovascular disease (CVD) is a group of heart and blood vessels' disorder, consisting of four entities; coronary heart disease, cerebrovascular disease, peripheral artery disease (PAD) and aortic atherosclerosis. In 2018, CVD was the leading cause of death globally with 18.6 million deaths (*World Health Organization*, 2021; Zhao *et al*., 2021). From 1990 to 2019, CVD deaths increased from 5.6 to 10.8 million. Indonesia is the sixth country with the highest number of CVD deaths in the worlds with the estimated number 375,479 deaths (Muharram et al., 2024; Vaduganathan et al., 2022; World Health Organization, 2021; Zhao, 2021a).

Risk factor for CVD include gender, age, environment and habits (Frąk et al., 2022; Ben Dhaou, Scott and Orr, 2024). The incidence of CVD is higher in men than women in childbearing age, but nowadays the incidence of CVD in women are increased from 27% in 2007 become 32% in 2010 (Okoth et al., 2020). The incidence of CVD was increased by the women's specific risk factors; pregnancy related factors (preeclampsia, miscarriage, premature birth, gestational diabetes and hypertension), contraception, hormonal therapy, polycystic ovarian syndrome (PCOS), autoimmune, age of menarche and menopause (Mehta et al., 2023; Tschiderer et al., 2023). The risk of having CVD in women gradually increase with age (Zhao, 2021; Anggelina, Kristina and Wiedyaningsih, 2023). Women who have experienced menopause have higher risk of CVD than childbearing age women (Rajendran

et al., 2023).

Various parameters have been used to predict incidence of CVD in childbearing age women, such as anti mullerian hormone (AMH). Anti mullerian hormone is a glycoprotein transforming growth factor β that play role in the development of primordial follicles (Shrikhande, Shrikhande and Shrikhande, 2020; Zeng et al., 2022). Low Serum AMH level associated with decreased estradiol level. Estradiol act as an atheroprotective by reducing inflammatory mediator and cytokine also reducing oxidative stress. It inhibiting smooth muscle cell proliferation and reducing cholesterol and macrophages accumulation to the arterial wall. Low estradiol level increased inflammatory responses and leukocyte adhesion to the endothelium and stimulate plaque formation (Figueroa-Vega et al., 2015; Looby et al., 2016). Studies found that high AMH level in PCOS patients increase the risk of insulin resistance, cytokines release and associated with CVD ((et al., 2017); Pratama *et al.*,2024). Contratory to that, low AMH level ini premenopause increase risk of having CVD. Until now, there has been no clear cut off AMH level that increase risk of having CVD. Hingga saat ini, belum ada *cut off* yang jelas dan studi mengenai AMH pada PKV di Indonesia. This study aims to provide an overview the role of AMH level in incidence of CVD. This study aims to determine the cut off AMH level that increased risk of CVD in childbearing age women. Furthermore, this cut off will be compared with another established CVD risk factors such as dyslipidemia, obesity and age.

Methods

Observational analytic was being conducted in Dr. Moewardi Regional Hospital from May until November 2024. This was a cross-sectional study with subjects were childbearing age women who come to clinical pathology department. Women who under chemoradiation therapy, pregnant and have history of fertility disorder such as polycystic ovarian syndrome (PCOS) were excluded from the study. A total subjects of 57 patients were being analyzed. Clinical characteristic (age, body mass index and patients' diagnosis) and lipid profile [cholesterol total level, low density lipid (LDL) cholesterol level, high density lipid (HDL) cholesterol level and trigliserid level] were collected from hospital information system (HIS) and laboratory information system (LIS).

Lipid profile being categorized with total cholesterol cut off ≥ 240 mg/dL, LDL cholesterol cut off ≥190 mg/dL, HDL cholesterol cut off < 40 mg/dL and trigliserid cut off ≥200 mg/dL. Lipid profile data was being taken at the same day with serum was taken. Subjects' serum that has been collected was stored in -80^{o}C refrigerator then being analyzed by Vidas Biomeriux IVD 3002596. AMH levels were measured in ng/mL units by ELFA method. IBM-SPSS version 29 was used to perform bivariate and multivariate analyses in this study. Nominal variable was described as frequencies and percentages, while continous variable was going under normality test. The normality test that used in this study was Kolmogorov-Smirnov because the number of sample is more than 50 sample. If the data was not normally distributed, log transformation was being performed. Non normal distributed data was described with median and being analyzed with mann whitney test. While normal distributed data being analyzed by independent sample t test. Data with nominal variable being analyzed with chi square. The data was analyzed with a significant level of $p < 0.05$ with a 95% confidence interval.

This study was approved by the Health Research Ethics Committee of Dr. Moewardi Hospital with ethical clearance number: 2.464/XI/HREC/2025.

Results

Subjects of 57 women aged 18-44 years old; 25 patients having CVD and 32 patients non CVD. Subjects characteristics can be seen in Table 1. The median age of CVD subjects were 42 years old and CVD were 33 years old. Arround 52.6% of the study subject included in obesity, 14% were overweight, 29.8% normoweight and 3.6% were underweight. Subjects' profile lipid the being categorized by dyslipidemia or not; 52.6% population having dyslipidemia and 47.4% were not having dyslipidemia. The mean AMH level in CVD population was 0.59 ± 0,12 ng/mL while in non CVD population was 1,14 ± 0,10 ng/mL.

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Characteristics	Total (n=57)		Childbearing Age Women		p
	n	%	CVD n = 25 (43,9%)	Non CVD n = 32 (56,1%)	
Age (years)			42 (26-44)	33 (18-44)	**<0,001[b,*]**
BMI (kg/m²)					
Obesity	30	52,6	33,2 (25,6-48,9)	28,37 (25,3-38,2)	0,198
Overweight	8	14	23,90 ± 0,13	24,41 ±0,42	0,191
Normoweight	17	29,8	20,6 (19,1-22,6)	20,82 (18,5-22,9)	0,820
Underweight	2	3,6	17,48	17,5	0,317
Dyslipidemia					**0,038*,c**
Yes	30	52,6			
No	27	47,4			
AMH (ng/mL)			0,59 ± 0,12	1,14 ± 0,10	**<0,001[a,*]**

Table 1. Subject characteristics

Abbreviation : BMI (body mass index), AMH (anti mullerian hormone), kg/m2 (kilogram per meter persegi), ng/mL (nanogram/mililiter), aindependent t test, bmann whitney test, cchi square *significant in p < 0,05

The BMI variable then being converted into a nominal categorical variable by categorized as obesity and non obesity. The AMH level variable was being converted into nominal categorical variable using receiver operating characteristics (ROC) curve analysis. Figure 1 shows the ROC curve analysis for the AMH and CVD. The cut off Serum AMH that being used in this study was 0.69 ng/mL with an area under curve (AUC) 0.841, sensitivity 84.4% and specificity 84%.

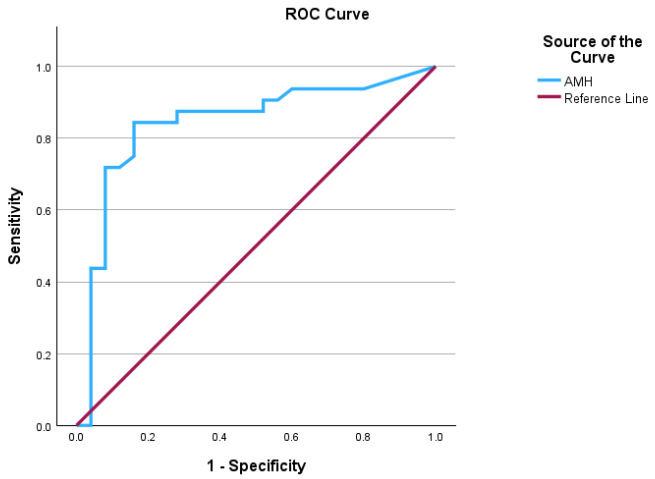

Chart 1. ROC variable curve AMH and CVD

Bivariate analysis showed that all study variables had a significant differences in CVD and non CVD population. The study subjects who ≥40 years old were 24 (42.1%) subjects and 33 (57.9%) subjects were under 40 years old. The p-value in the chi-square test was 0.017, indicating that there was a significant difference in the age between the CVD and non-CVD populations. The study subjects with obesity were 17 (56.7%) having CVD and 13 (43.3%) subjects did not have CVD. The p-value in the chi-square test for the obesity variable was 0.038, indicating that there was a statistically significant difference between CVD and non-CVD subjects. The study subjects with dyslipidemia were 17 (56.7%) having CVD and 13 (43.3%) subjects did not have CVD. There was a statistically significant

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difference between the dyslipidemia variables in the CVD and non-CVD populations, with p-value 0.038. Arround 26 (45.6%) subjects having low level Serum AMH (<0.69 ng/mL) while 31 (54.4%) subjects were having higher Serum AMH (≥0.69 ng/mL), and 20 of 26 subjects (76.9%) having CVD and 6 (23.1%) subjects with low Serum AMH level did not have CVD. There was a significant difference between AMH and CVD with p value <0.001.

Characteristic	n	%	Childbearing Age Women CVD n=25 (43,9%)	Non CVD n=32 (56,1%)	*p value*	PR	95% CI
Age (years)					**0,017***	2,292	1,211-4,335
≥40	24	42,1	15 (62,5)	9 (37,5)			
<40	33	57,9	9 (27,3)	24 (72,7)			
Obesity					**0,038***	2,186	1,074-11,492
Yes	30	52,6	15 (62,5)	9 (37,5)			
No	27	47,4	9 (27,3)	24 (72,7)			
Dyslipidemia					**0,038***	2,186	1,074-4,447
Yes	30	52,6	17 (56,7)	13 (43,3)			
No	27	47,4	7 (25,9)	20 (74,1)			
AMH (ng/mL)					**<0,001***	5,962	2,332-15,238
< 0,690	26	45,6	20 (76,9)	6 (23,1)			
≥ 0,690	31	54,4	4 (12,9)	27 (87,1)			

Table 2. Bivariate analysis of various independent variable on CVD

Abbreviation : % (percent), n (total), PR (prevalence ratio), AMH (anti mullerian hormone),
*significant in p <0.05

Table 3 presents the results of backward stepwise logistic regression analyzed the relationship between predictor variables obesity, dyslipidemia and AMH with the incidence of CVD in the study. The obesity variable has a statistically significant difference with prevalence ratio (PR) value of 7.172 (95% CI: 1.319 - 38.991, p = 0.023), indicating that obesity increased CVD events by 7.172 times compared to the non-obese population. The AMH variable has a statistically significant different with PR value of 34.049 (95% CI: 6.245 - 185.630, p = <0.001), indicating that Serum AMH level <0.69 ng / mL increased the CVD events 34.049 times than subjects with serum AMH levels ≥0.69 ng / mL.

Tabel 3.

	Characteristics	SE	PR	95% CI		*p-value*
				Lower	*Upper*	
***Step* 1**	Obesity	0,879	6,046	1,081	33,825	**0,041***
	Dyslipidemia	0,834	2,589	0,505	13,267	0,254
	Age	0,836	2,803	0,545	14,414	0,217
	AMH	0,890	22,326	3,903	127,707	**<0,001***
***Step* 2**	Obesity	0,872	6,720	1,217	37,103	**0,029**
	Dyslipidemia	0,764	2,042	0,456	9,135	0,351
	AMH	0,879	28,550	5,098	159,883	**<0,001***
***Step* 3**	Obesity	0,865	7,172	1,319	38,991	**0,030***
	AMH	0,897	34,049	6,245	185,630	**<0,001***

Table 3. Multivariate analysis of various independent variable on CVD

Abbreviation : AMH (anti mullerian hormone), CI (confidence interval), SE (standard error), PR (prevalence ratio), step (langkah), * significant in p < 0.05

Discussion

The results of the logistic regression test analysis showed that lower Serum AMH levels (cut off < 0.69 ng/mL) were 34.049 times more at risk of developing CVD (95% CI: 6.245 - 185.630, p = <0.001). Decreased AMH levels are associated with decreased estradiol levels in childbearing age women (Verit et al., 2016; El Khoudary et al., 2023). Estradiol plays a role in initiating mitochondrial respiration so that decreased estradiol levels result increased absorption of calcium ions by mitochondria resulting opening of mitochondrial permeability transition pores and increased release of cytochrome C accompanied by increased ROS formation which triggers heart cell apoptosis (Mahmoodzadeh and Dworatzek, 2019; Ventura-Clapier et al., 2019). Study by Rios et al.,(2020) stated that low AMH levels are associated with increased levels of LDL cholesterol, triglycerides and insulin resistance. Low Serum AMH level also associated with cardiometabolic risk factors. Research by Güler et al., (2020) stated that lower Serum AMH levels are associated with increased risk of CVD in pregnant women. Likewise, research conducted by Perry et al., (2016) stated that higher AMH levels have a lower risk of ischemic heart disease.

Contrary in subjects with PCOS, higher Serum AMH level increased risk having CVD. AMH will bind to the AMHR2 receptor in gonadotropin releasing hormone (GnRH) neurons, thereby increasing the frequency of GnRH pulsations which results in increased LH and increased AMH. AMH levels suppress the expression of the aromatase enzyme in granulosa cells and reduce the conversion of androgens to estrogen. Hyperandrogen conditions caused adipose tissue dysfunction (hypertrophy, hypoperfusion, hypoxia and apoptosis), released inflammatory cytokines and insulin resistance (Pratama et al., 2024). Increased Serum AMH levels also increased NF-kB level which play a role in atherosclerosis (Lambrinoudaki et al., 2020; Barton et al., 2023). Contra with our study, research by von Berg, et al., (2022) showed that AMH had no effect on cardiometabolic health.

In addition to the AMH variable, the obesity variable was independently associated with the incidence of CVD (p = 0.030), subjects with BMI > 25 kg / m2 were 1.319 times more at risk of having CVD than non-obese subjects (95% CI: 1.319–38.991, p = 0.023). The mechanism of obesity causing CVD is because of obesity was associated with other conditions that become CVD's risk factors such as; hypertension, dyslipidemia, type 2 diabetes mellitus (Manrique-Acevedo et al., 2020; Lopez-Jimenez et al., 2022; Ojalehto et al., 2023). Obesity was associated with increased accumulation of free fatty acids which increased the production of pro-inflammatory mediators and recruitment of pro-inflammatory macrophages. Excessive inflammatory conditions will increased oxidative stress and tissue damage (Koliaki et al., 2019; Lopez-Jimenez et al., 2022). Obesity can decreased AMH levels which cause decreased GnRH stimulation which results in decreased LH and FSH and increased leptin levels. Increased leptin levels will eventually cause leptin resistance and subsequently insulin resistance. Insulin resistance caused hyperinsulinemia and disrupts the glucose metabolism process. Insulin resistance wass associated with chronic mild inflammation and the production of proinflammatory cytokines such as TNF-α, IL-6, IL-8, plasminogen activator inhibitor-1 (PAI-1) and monocyte chemoattractant protein-1 (MCP-1). The increase in proinflammatory cytokines is also accompanied by an increase in C-reactive protein (CRP). Chronic mild inflammation accompanied by the involvement of the immune system (macrophages) results in endothelial dysfunction and atherosclerosis (Kosmas et al., 2023; Eng et al., 2024).

Research by Lopez-Jimenez et al., (2022) stated that BMI> 25 kg / m2 was associated with CVD mortality, especially CHD and ischemic stroke. A cohort study by Ojalehto et al., (2023) stated that obesity at the age of 40-64 years increased the risk of having CVD. Research by Khan et al., (2018) revealed that increased BMI was associated with increasing incidence of PKV, BMI ≥40 increases the risk of PKV 3.14 times higher compared to normoweight subjects (95% CI: 2.48-3.97). Cohort study by Almuwaqqat et al., (2024) increased BMI was associated with an increased risk of having CVD by 16%

[hazard ratio (HR), 1.16; 95% CI: 1.13-1.19]. Although BMI was associated with the incidence of CVD, other studies by Peters et al., (2018) and Darbandi et al., (2020) revealed that waist to hip ratio (WHR) and waist circumference (WC) better describe the risk of CVD than BMI, because WHR and WC reflect increased adipocytes in the abdominal area which correlated with chronic inflammation (Khan et al., 2018; Peters et al., 2018; Darbandi et al., 2018).

Dyslipidemia had a significant difference in the population with and without PKV ($p = 0.038$). Dong et al., (2021) also stated that triglyceride, total cholesterol and HDL levels were correlated with CVD and have statistically significant difference. The increased lipids and lipoproteins concentration in the blood, was related to the process of atherosclerosis. Cholesterol deposits in the intimal arteries cause lesions of endothelial cells in the blood vessel wall which induce the release of ROS which induces the formation of oxidative stress and impaired left ventricular function. LDL oxidation and modification of the heart structure increase endothelial cell permeability and increase the expression of adhesion molecules, chemotaxis proteins and monocytes which will accumulate oxidized lipoproteins and form foam cells which initiate the formation of atherosclerosis. Triglycerides can cause atherosclerosis because of the triglyceride's ability to enter the intimal arteries and combine with atherosclerotic plaques to become larger. In addition, lipoprotein lipase activity found in endothelial cells and the intima will degrade triglycerides and release free fatty acids and inflammatory monoacylglycerols. Fat accumulation causes smooth muscle cell proliferation and inflammatory cell activation which results in plaque formation and blood vessel necrosis (Nordestgaard, 2016; Addisu et al., 2023; Du and Qin, 2023; Abera et al., 2024).

HDL cholesterol plays a protective factor against CVD. HDL cholesterol has the ability to transport excess cholesterol found in peripheral tissues back to plasma and bring it back to the liver to be metabolized by bile salts before being excreted (Ouimet et al., 2019; Hedayatnia et al., 2020). Increased LDL cholesterol levels are associated with an increased risk of CVD (Stanciulescu et al., 2023). A cohort study by Hedayatnia et al., (2020) showed that increased total cholesterol, LDL and triglycerides were associated with increased risk of CVD (HR: 1.53, 95% CI: 1.18-1.98; $p<0.01$; HR: 1.54, 95% CI: 1.19-2; $p<0.01$; HR: 1.57, 95% CI: 1.27-2.03; $p<0.01$). A study by Abera et al., (2024) stated that dyslipidemia (abnormality in one of the lipid levels), especially decreased HDL cholesterol, is associated with the risk of heart disease. In contrast to this study, a study by Güleç et al., (2020) showed that HDL cholesterol levels >80 mg/dL increase the risk of CVD. Research by Kim et al., (2023) stated that increasing HDL cholesterol levels ≥60 mg/dL increased the risk of having CVD (HR: 1.15; 95% CI: 1.05-1.25; $p<0.001$). This difference is caused by oxidative modification of HDL cholesterol which results in damage to the lecithin-cholesterol acyltransferase (LCAT) binding site in apolipoprotein A-1. The LCAT enzyme is an enzyme that is useful in cholesterol esterification and the HDL maturation process. ApoA-1 dysfunction results in decreased HDL biogenesis and results in HDL containing Trp72 which is a pro-inflammatory compound (Güle et al., 2020; Liu et al., 2022; Kim et al., 2023).

Bivariate test in this study showed that age had a statistically significant difference in the CVD and non-CVD populations ($p=0.017$). Increasing age is associated with changes and functional structure of the heart; systolic, diastolic and electrical dysfunction of the heart. Changes in cardiac structure and function are accompanied by accumulation of increased oxidative stress, ROS and chronic inflammation resulting in CVD (Qu et al., 2024; Zhao et al., 2024). Based on the study of Rodgers et al., (2019) the risk of CVD increases with age. According to several studies, this increase in cardiovascular risk begins at the age of 40 in women (World Health Organization, 2021; Wahabi et al., 2023). The age of 40 in women is marked by perimenopause conditions associated with decreased estrogen levels (a protective factor for CVD) resulting in an increased risk of having CVD (Gao and Moodie, 2022; Lange-Maia et al., 2023).

Conclusions

This study demonstrated that serum AMH levels were independently associated with cardiovascular disease (CVD) events. AMH levels <0.69 ng/mL increased the risk of CVD

by 34.049 times compared to AMH levels ≥0.69 ng/mL (95% CI: 6.245–185.630; p < 0.001). Obesity was also identified as a significant risk factor, increasing the likelihood of CVD by 7.172 times compared to non-obese individuals. These findings highlight the significance of AMH as a potential biomarker for early identification of CVD risk among women of childbearing age, addressing the need for accessible and preventive screening tools in this population.

However, this study has limitations, including the single-center design and the relatively small sample size, which may not represent broader population characteristics. Future studies should utilize prospective or retrospective cohort designs and involve multicenter samples to obtain more generalizable findings. Further research is also needed to explore additional parameters, such as waist-to-hip ratio (WHR) or other biomarkers commonly available in rural healthcare settings, to enrich the understanding of CVD risk assessment among women of reproductive age.

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