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ORIGINAL ARTICLE

Association between vitamin D levels, superoxide dismutase (SOD), and malondialdehyde (MDA) with endometriosis

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ABSTRACT

Background and aim: Endometriosis is a chronic condition in which endometrial tissue grows outside the uterus, causing pain and fertility problems. Oxidative stress plays an important role in its pathogenesis, characterized by an increase in free radicals and a decrease in antioxidant activity. SOD functions as the main defense against free radicals, while MDA reflects the level of oxidative damage. In addition, vitamin D has anti-inflammatory and antioxidant effects that may be related to the development of endometriosis. The Aim of this study is to determine the relationship between vitamin D, SOD, and MDA levels and endometriosis.
Methods: A descriptive analytic case–control study was conducted involving 86 women of reproductive age, consisting of patients with histopathologically confirmed endometriosis and controls without endometriosis. Venous blood samples were collected to measure serum vitamin D, SOD, and MDA levels using ELISA. Data were analyzed using independent t-tests, chi-square tests, and receiver operating characteristic (ROC) curve analysis, with statistical significance set at $p < 0.05$.
Results: Vitamin D levels were lower in endometriosis compared to controls (16.4 ng/mL vs. 20.4 ng/mL; $p = 0.003$). SOD levels were also lower in endometriosis compared to controls (28.1 ng/mL vs. 39.6 ng/mL; $p = 0.001$). MDA levels were higher in endometriosis compared to controls (29.1 ng/mL vs. 22.7 ng/mL; $p = 0.024$). Vitamin D deficiency was more common in endometriosis than in controls (58.2% vs. 41.8%, $p = 0.04$). MDA level of 24.1 ng/mL had a sensitivity of 62.0% and specificity of 60.5% in diagnosing endometriosis.

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Conclusions: Patients with endometriosis have lower vitamin D levels, decreased superoxide dismutase, and increased oxidative stress, specifically malondialdehyde. (www.actabiomedica.it)

Key words: endometriosis, vitamin d, superoxide dismutase, malondialdehyde

Introduction

Endometriosis is a gynecological disorder characterized by the growth of endometrial tissue outside the uterine cavity. The most common sites of endometrial growth are the pelvic peritoneum, ovaries, and rectovaginal septum. The prevalence of endometriosis is 10-15% among women of reproductive age and is often underdiagnosed (1). The impact of endometriosis is chronic pelvic pain that can affect quality of life both psychologically and in terms of productivity. This chronic pelvic pain originates from macrophage activity and mast cell activation, which contribute to a continuous inflammatory cycle and oxidative stress. The etiopathogenesis of endometriosis involves abnormal angiogenesis, leading to inflammation and oxidative stress (2). Oxidative stress is a potential component of the pathophysiology underlying endometriosis and is characterized by an imbalance between reactive oxygen species (ROS) and antioxidants. Oxidative stress occurs because ROS are produced in large quantities while antioxidant activity is reduced. Oxidative stress also plays a role in endometriotic pain, but current knowledge regarding its etiology is still lacking.[1] Malondialdehyde (MDA) is the end product of lipid oxidation caused by free radicals and is used as a marker of the oxidative stress process that causes cell membrane damage. Other markers include protein carbonyls and serum iron. Antioxidants are needed to prevent oxidative stress by counteracting free radicals. Antioxidants consist of endogenous antioxidants produced by the body and exogenous antioxidants. Endogenous antioxidant enzymes include superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx). SOD works by converting superoxide anions into less reactive hydrogen peroxide, which is then converted into water (H2O) by catalase or GPx. Exogenous antioxidants are obtained by the body through vitamin D (3). Vitamin D is one of the exogenous antioxidants that acts by stabilizing free radicals by providing hydrogen atoms. In addition, vitamin D, both in its inactive form 25-dihydroxy vitamin D [25(OH)2D] and its active form 1,25-dihydroxyvitamin D [1,25(OH)2D], works by reducing inflammation and preventing oxidative stress. Vitamin D functions as a powerful antioxidant by preventing lipid peroxidation, DNA damage, and protein oxidation, all of which can cause cell and tissue damage (4). The inflammatory process and oxidative stress that occur in patients with endometriosis then produce MCP 1, which can facilitate COX to produce prostaglandin (PGE 2) and other cytokines such as VEGF and NRF, which then stimulate angiogenesis, proliferation, and innervation, triggering chronic pelvic pain. This chronic pelvic pain can cause a decline in quality of life in patients with endometriosis (5). Based on the above explanation, there is a relationship between vitamin D and SOD as antioxidants in preventing increased oxidative stress in endometriosis, which needs to be studied further.

Materials and methods

Study design and subjects

A descriptive-analytic case-control study conducted at Wahidin Sudirohusodo Hospital and its network of hospitals from December 2024 until the sample size is fulfilled. Eighty-six women of reproductive age were recruited and categorized into case groups (women with endometriosis) and control groups (women without endometriosis). The diagnosis of endometriosis was made intraoperatively and confirmed by histopathological examination in patients undergoing surgery. The control group consisted of patients undergoing gynecological surgery for non-endometriosis indications. All women met the age eligibility criteria (15–49 years) and were willing to participate thru written consent. Subjects with cardiovascular disease, chronic infections, autoimmune diseases, cancer, diabetes mellitus, and those taking antioxidants (such as vitamins C or E), glucocorticoids, anticonvulsants, or antiretroviral therapy, as well as pregnant women, were excluded.

Data collection

Upon confirmation of eligibility, venous blood samples (3 cc) were obtained from all subjects in a fasting predetermined manner. Blood samples were collected preoperatively without standardization to menstrual cycle phase. Serum 25-hydroxyvitamin D [25(OH)D], SOD, and MDA levels were measured using commercially available ELISA kits. Blood samples were processed promptly, and serum was stored at 20°C until analysis to minimize degradation, The serum levels of vitamin D, SOD, and MDA were detected with enzyme-linked immunosorbent assay (ELISA) kits according to the manufacturer's instructions. The data of demographic and clinical information were collected by chart reviews and direct interviews, and the laboratory results during hospitalization were reported on a standardized collection form before being analyzed. Malondialdehyde (MDA) levels were measured using the MDA ELISA Kit 96T (E-EL-0060) from Elabscience Biotechnology Co., Ltd. with the competitive ELISA method. This kit has an analytical range of 31.25–2000 ng/mL and a sensitivity of 18.75 ng/mL. Measurement precision is considered good with a coefficient of variation (CV) <10%. Absorbance readings were taken at a wavelength of 450 nm. The 25-hydroxyvitamin D [25(OH)D] level was measured using the 25(OH)D ELISA Kit 96T (CAN-VD-510) produced by Elabscience Biotechnology Co., Ltd. with the competitive ELISA method. This kit has an analytical range of 4–120 ng/mL and demonstrates good precision with intra- and inter-assay coefficients of variation (CV) <10%. SOD levels were measured using the Human SOD1 ELISA Kit 96T (E-EL-H113) produced by Elabscience Biotechnology Co., Ltd., employing the sandwich ELISA method. This kit has an analytical range of 0.156–10 ng/mL and exhibits good precision with intra- and inter-assay coefficients of variation (CV) <10%.

Statistical analysis

Statistical processing data analysis was carried out using SPSS software version 26.0 (SPSS Inc., Chicago, IL, USA). Parametric data were summarized as mean ± SD, and categorical data as frequency (%). Differences in endometriosis vs. control were compared by independent t-test for normally distributed data. Chi-square test was used for categorical variables. Receiver-operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic value, including sensitivity and specificity. Values of $p < 0.05$ were considered to be statistically significant.

Result

Research subject characteristics

This study involved 86 patients consisting of 43 patients with endometriosis and 43 patients as controls. The average age of patients was 31.2 years, where most had normal BMI, were married, nulliparous, non-smokers, and did not receive vitamin D supplementation. Based on age, BMI, marital status, smoking, and vitamin D supplementation, there were no significant differences between subjects with endometriosis and controls. Comparison of research subject characteristics is shown in Table 1.

Comparison of vitamin D, superoxide dismutase, and malondialdehyde levels

Vitamin D levels were significantly lower in the endometriosis group compared with controls (16.4 ng/

Table 1. Characteristics of the study

Variable	Endometriosis (n=43)	Control (n=43)	Total	*p-value*
	n (%)	n (%)	n (%)	
Age (mean + SD)	32.6 + 4.2	31.8 + 4.3	31.2 + 4.5	0.752
BMI				
Underweight	2 (4.7)	1 (2.3)	3 (3.5)	0.748
Normoweight	37 (86.0)	35 (81.4)	72 (83.7)	
Overweight	3 (7.0)	5 (11.6)	8 (9.3)	
Obesity	1 (2.3)	2 (4.7)	3 (3.5)	
Marital status				
Yes	41 (95.3)	38 (88.4)	79 (91.9)	0.237
No	2 (4.7)	5 (11.6)	7 (8.1)	
Parity				
Nullipara	22 (51.1)	23 (53.5)	45 (52.3)	0.717
Primipara	16 (37.2)	13 (30.2)	29 (33.7)	
Multipara	5 (11.6)	7 (16.3)	12 (14.0)	
Smoking				
Yes	2 (4.7)	1 (2.3)	3 (3.5)	0.557
No	41 (95.3)	42 (97.7)	83 (96.5)	
Vitamin D Supplementation				
Yes	2 (4.7)	3 (7.0)	5 (5.8)	0.645
No	41 (95.3)	40 (93.0)	81 (94.2)	

Table 2. Comparison of vitamin D, superoxide dismutase, and malondialdehyde levels

Variable	Endometriosis	Control	*p-value*
	Mean (Min-Max)	Mean (Min-Max)	
Vitamin D (ng/mL)	16.4 (5.9 - 35.3)	20.4 (13.3 - 27.4)	0.003
Superoxide dismutase (ng/mL)	28.1 (11.8 - 48.2)	39.6 (14.9 - 93.0)	0.001
Malondialdehyde (ng/mL)	29.1 (10.1 - 84.8)	22.6 (2.1 - 57.2)	0.024
Independent sample T test; p<0.05			

mL vs. 20.4 ng/mL; p = 0.003) (Table 2; Figure 1). Likewise, SOD levels were significantly lower in patients with endometriosis than in controls (28.1 ng/mL vs. 39.6 ng/mL; p = 0.001) (Table 2; Figure 2). Conversely, MDA levels were significantly higher in the endometriosis group than in the control group (29.1 ng/mL vs. 22.7 ng/mL; p = 0.024) (Table 2; Figure 3).

Vitamin D status in endometriosis

Vitamin D deficiency was significantly more common in endometriosis than in controls (32 (58.2%) vs. 23 (41.8%)). Conversely, normal vitamin D levels were more common in controls than in endometriosis (20 (64.5%) vs. 11 (35.5%)) (p=0.04) (Table 3).

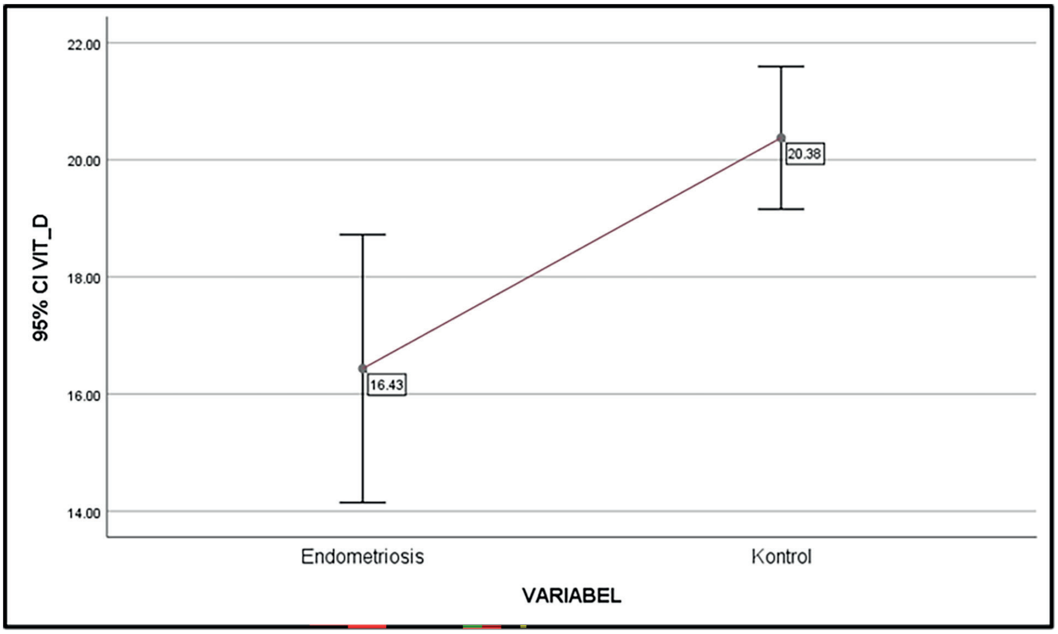

Figure 1. Comparison of mean vitamin D levels in endometriosis and controls. Mean vitamin D levels were significantly lower in endometriosis than in control (16.4 ng/mL vs. 20.4 ng/mL; p = 0.003).

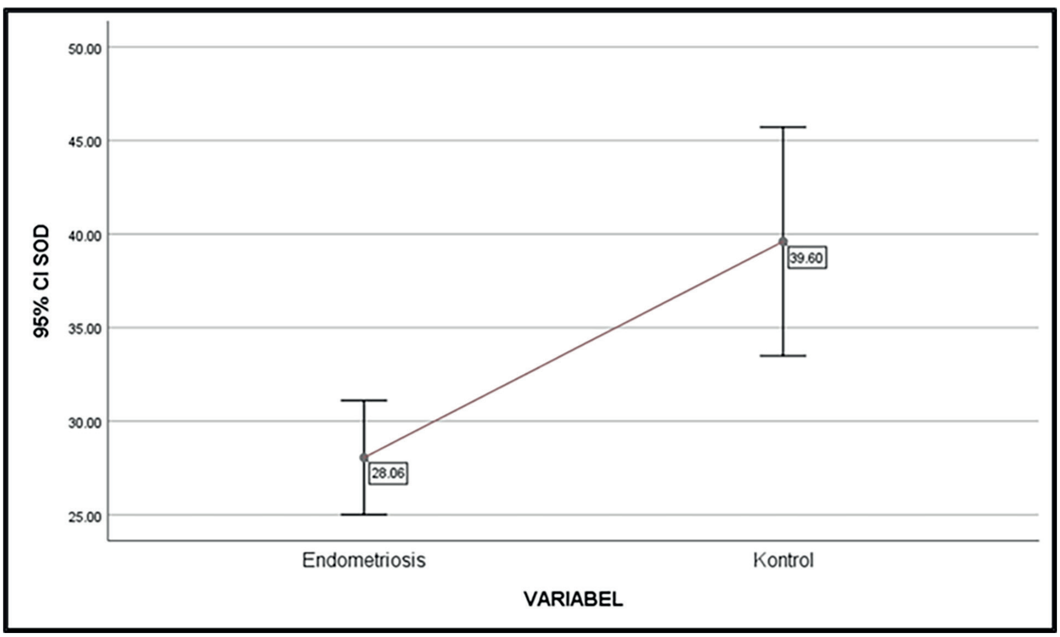

Figure 2. Comparison of mean SOD levels in endometriosis and controls. Mean SOD levels were significantly lower in endometriosis than in controls (28.1 ± 9.9 ng/mL vs. 39.6 ± 19.9 ng/mL; p = 0.001).

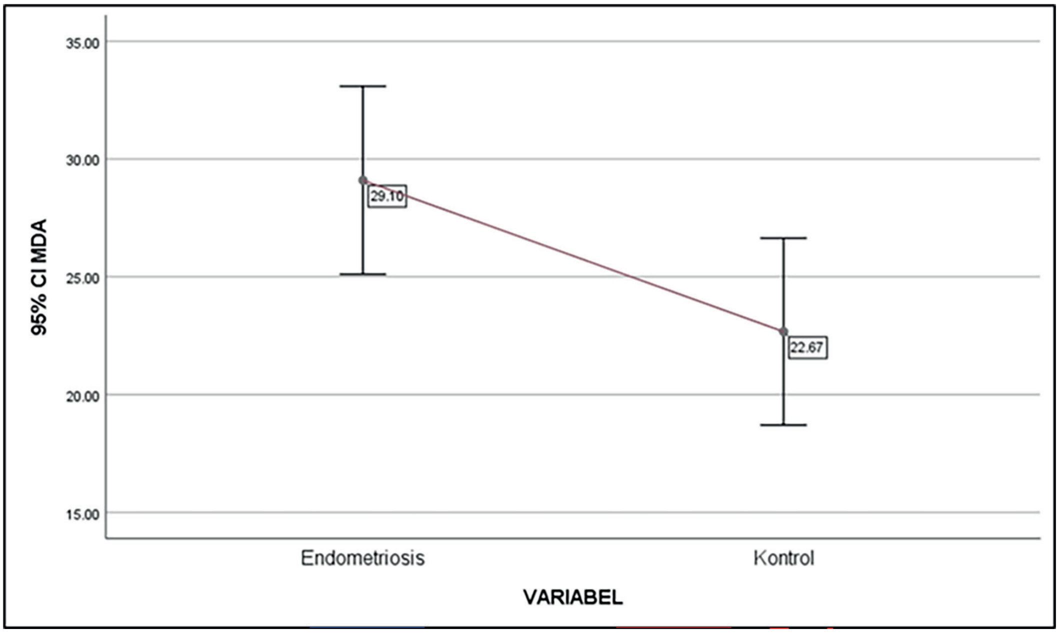

Figure 3. Comparison of mean MDA levels in endometriosis and controls. Mean MDA levels were significantly higher in endometriosis patients than in controls (29.1 ± 13.0 ng/mL vs. 22.7 ± 12.9 ng/mL; p = 0.024).

Table 3. Vitamin D status in endometriosis.

Variable	Vitamin D: Deficiency n (%)	Vitamin D: Normal n (%)	Total	p-value
Endometriosis	32 (58.2)	11 (35.5)	43 (50)	0.04
Control	23 (41.8)	20 (64.5)	43 (50)	
Total	**55 (100)**	**31 (100)**	**86 (100)**	
Chi square test p<0.05				

Superoxide dismutase status in endometriosis

Analysis to determine the cutoff value of SOD and measure sensitivity and specificity for diagnosing endometriosis found that the cutoff value of SOD was 29.8 ng/mL with a sensitivity of 38.5% and specificity of 37.2% in diagnosing endometriosis (Figure 4).

SOD levels <29.8 ng/mL were more commonly found in endometriosis than in controls (23 (62.2%) vs. 14 (37.8%)). Meanwhile, SOD levels >29.8 ng/mL were more commonly found in controls than in endometriosis (29 (59.2%) vs. 20 (40.8%)) (p=0.05) (Table 4).

Malondialdehyde status in endometriosis

Analysis to determine the MDA threshold and assess sensitivity and specificity for diagnosing endometriosis found that the MDA threshold was 24.1 ng/mL with a sensitivity of 62.0% and specificity of 60.5% (Figure 5).

MDA levels <24.1 ng/mL were more commonly found in controls than in endometriosis (26 (61.9%) vs. 16 (38.1%)), while MDA levels >24.1 ng/mL were more commonly found in endometriosis than in controls (27 (61.4%) vs. 17 (38.6%)) (p=0.03) (Table 5).

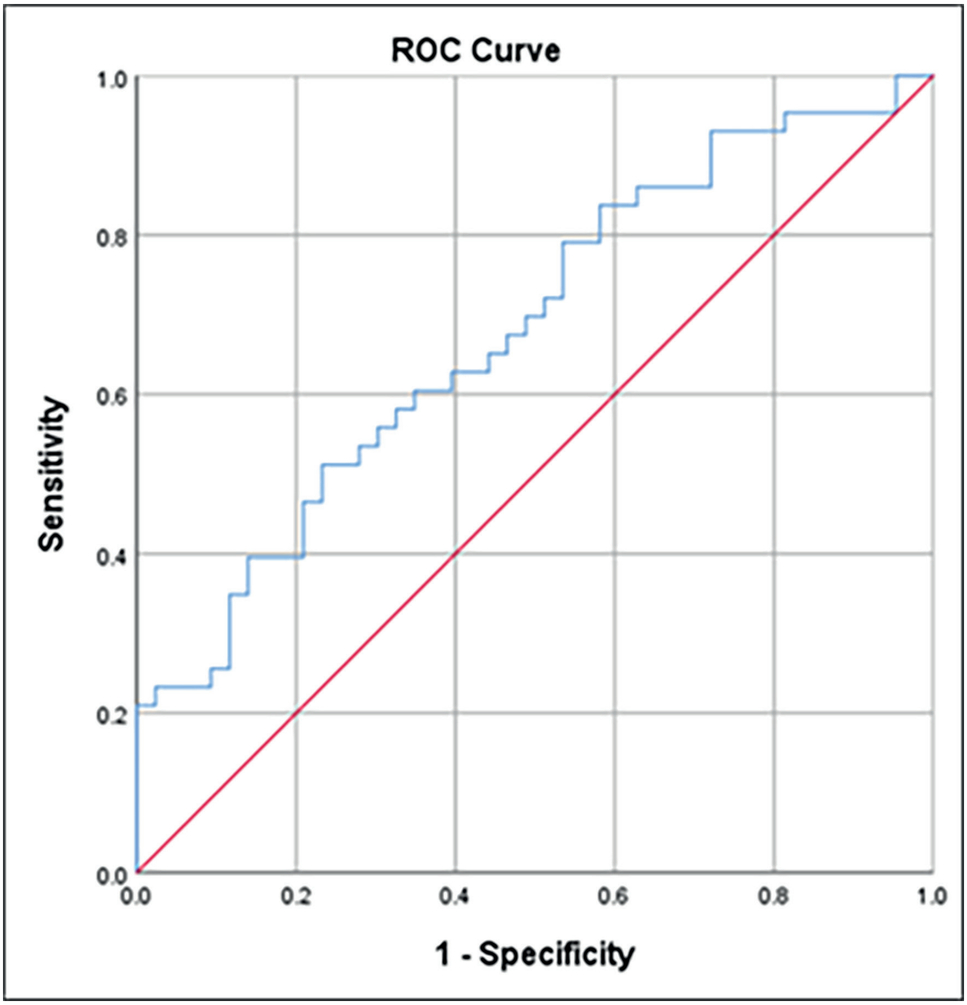

Figure 4. Superoxide dismutase ROC curve.

Table 4. Superoxide Dismutase Status in Endometriosis

Variable	Superoxide Dismutase <29,8 ng/mL n (%)	Superoxide Dismutase >29,8 ng/mL n (%)	Total	p
Endometriosis	23 (62.2)	20 (40.8)	43 (50)	0.05
Control	14 (37.8)	29 (59.2)	43 (50)	
Total	**37 (100)**	**49 (100)**	**86 (100)**	
Chi square test p<0.05				

Discussion

This study found that the average age of research subjects with endometriosis was 32.6 + 4.2 years. These results are in line with those found by Parasar et al. (2017), who reported that approximately 10% of women of reproductive age (15–49 years) experience endometriosis, with the peak incidence occurring

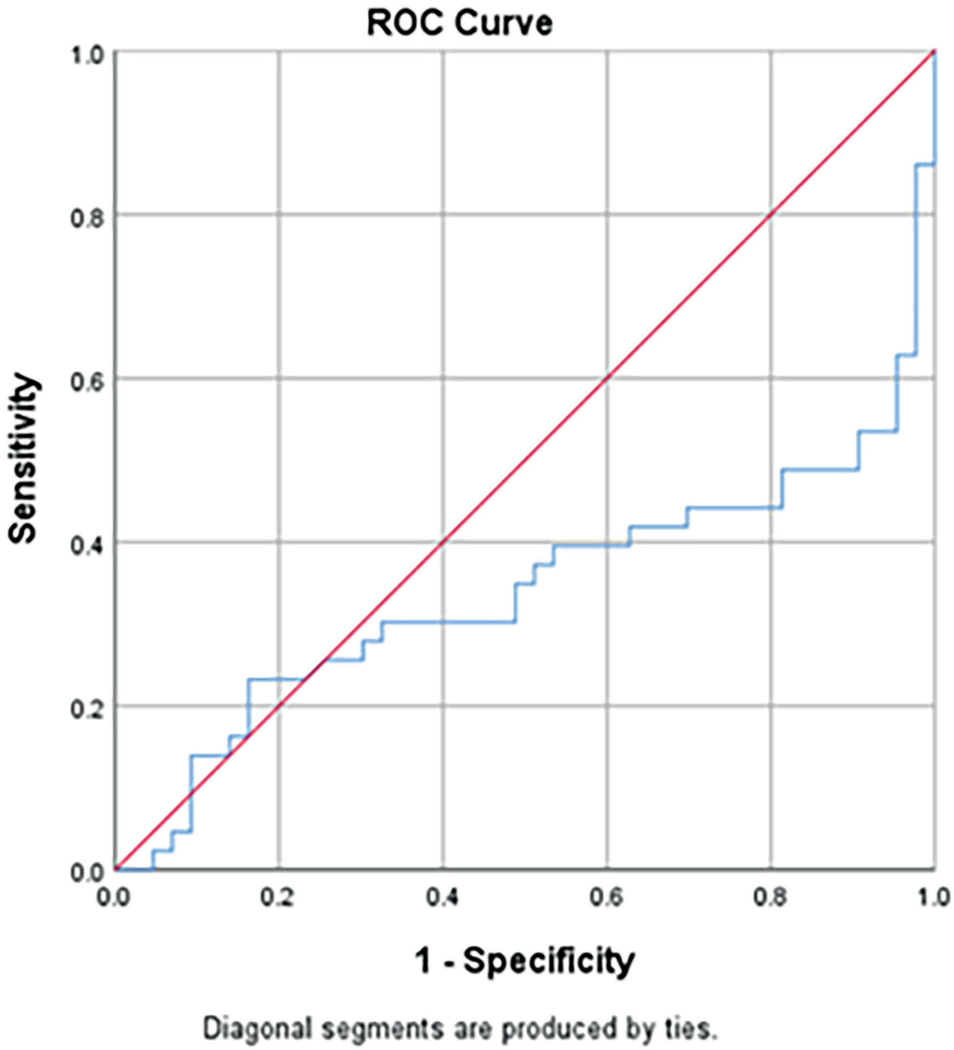

Figure 5. Malondialdehyde ROC curve.

Table 5. Malondialdehyde Status in Endometriosis

	Malondialdehyde			
	<24,1 ng/mL	>24.1 ng/mL		
Variable	**n (%)**	**n (%)**	**Total**	**p**
Endometriosis	16 (38.1)	27 (61.4)	43 (50)	0.03
Control	26 (61.9)	17 (38.6)	43 (50)	
Total	**42 (100)**	**44 (100)**	**86 (100)**	

Chi square test p<0.05

between the ages of 25–35 years. A study based on Australian national data also found that the highest prevalence of endometriosis was found in women aged 25–39 years (6). Endometriosis is most often diagnosed in women of reproductive age, particularly between the ages of 25–35 years, although symptoms can appear as early as adolescence. Studies have found that the prevalence and severity of endometriosis increase significantly with age, especially in adolescents and young adults (25–35 years old) (7). This study found that most endometriosis patients had a normal BMI (18.5-24.9 kg/m2). These results are in line with those found by Jin et al. (2025) and Lee et al. (2025), which show that the majority of endometriosis patients have a BMI in the normal range (18.5-24.9 kg/m2) (8,9). Similar findings were also reported by Wiebe & Tonelli (2025), who showed that endometriosis can occur even in women with normal BMI (10). Endometriosis is not only influenced by obesity, but by a complex interaction between estrogen hormones, systemic inflammatory status, and genetic factors. Theoretically, low or normal body fat levels can lead to increased levels of free estrogen, which supports the growth of ectopic endometrial tissue, explaining why women with normal BMI still have a high risk of experiencing endometriosis (11). This study found that most endometriosis patients were married. These results are consistent with a study conducted in Brazil, which found that most women diagnosed with endometriosis were married, and they generally sought treatment after experiencing severe dysmenorrhea and infertility (12). Married women tend to have a higher prevalence of endometriosis because this group undergoes reproductive examinations more frequently, especially when experiencing infertility. Hormonal factors, inflammation, and lifestyle changes after marriage may also increase the risk of developing clinical symptoms of endometriosis (13). This study found that most endometriosis patients were nulliparous. These results are in line with a study by Burghaus et al. (2011), which showed that nulliparous women have a 2–3 times higher risk of developing endometriosis compared to multiparous women (14). Similar results were also reported by Sarria-Santamera et al. (2021), who found a prevalence of endometriosis of 11.5% in nulliparous women aged 20–39 years (15). Additionally, El-Hadad et al. (2023) explained that endometriosis is commonly found in adolescents and young nulliparous women due to the absence of prolonged pregnancy, which typically provides a protective effect against the growth of ectopic endometrial tissue (16). Theoretically, pregnancy causes a decrease in estrogen levels and a period of anovulation that suppresses endometrial proliferation, while in nulliparous women, repeated exposure to estrogen during each menstrual cycle triggers the growth and implantation of endometrial tissue outside the uterus. This study also found that most endometriosis patients do not smoke. These findings are consistent with Muselli et al. (2024), who found that although non-smoking women have a healthier lifestyle, the prevalence of endometriosis remains high, indicating that hormonal and environmental factors are more dominant than smoking habits (17). These results are consistent with the study by Alson et al. (2022), which showed a prevalence of endometriosis of 21.8% in non-smoking subfertile women (18), and Sun et al. (2025), which confirmed that most endometriosis sufferers are non-smokers (19). Several studies have evaluated the relationship between smoking habits and the risk of endometriosis, but the results are still varied. This study found that vitamin D levels were higher in control patients than in patients with endometriosis (16.4 ng/mL vs. 20.4 ng/mL; $p = 0.003$). These results are consistent with the study by Abesadze et al. (2025), which also found a significant difference in vitamin D levels between healthy patients and endometriosis patients (26.1 ng/mL vs. 17.8 ng/mL; ($p<0.001$)) (20). Vitamin D acts as an immunomodulator that suppresses the expression of proinflammatory cytokines (such as IL-6, IL-8, and TNF-α) and inhibits the proliferation and angiogenesis of ectopic endometrial tissue. Vitamin D deficiency causes immune dysregulation and increased chronic inflammation in the pelvic cavity, which exacerbates the development of endometriotic lesions. This study found that SOD levels were also significantly lower in endometriosis compared to controls (28.1 ng/mL vs. 39.6 ng/mL; $p = 0.001$). These results are consistent with the findings of Kalu et al. (2007), who found that SOD activity in women with endometriosis was significantly lower than in controls (14.2 ng/mL vs. 25.4 ng/mL) (21). Similarly, Mier-Cabrera et al. (2011) reported a 30–40% decrease in plasma SOD in patients with endometriosis compared to healthy women (22). Theoretically, SOD plays an important role in converting superoxide radicals (O2–) into less reactive hydrogen peroxide. A decrease in SOD causes the accumulation of free radicals, which can damage membrane lipids, DNA, and proteins, triggering chronic inflammation and supporting the ectopic implantation of endometrial tissue. This decrease in antioxidant capacity is believed to worsen the peritoneal microenvironment, increasing angiogenesis and adhesion of ectopic endometrial cells. Thus, your research results reinforce the theory that antioxidant system disorders, particularly decreased SOD, play a central role in the mechanism of oxidative stress in endometriosis (23). This study found that MDA levels were significantly higher in endometriosis compared to controls (29.1 ng/mL vs. 22.7 ng/mL; p = 0.024). These results are consistent with the study by Turkyilmaz et al. (2016), which reported increased serum MDA levels in endometriosis patients (31.4 ng/mL) compared to controls (18.9 ± 4.7 ng/mL) (24). Another study by Oral et al. (2006) also found higher MDA levels in endometriosis patients (27.8 ± 5.6 ng/mL) compared to the control group (19.2 ± 4.1 ng/mL) (25). Theoretically, increased MDA reflects lipid membrane damage by ROS produced as a result of chronic inflammation in the pelvic cavity. In endometriosis, the activation of peritoneal macrophages and ectopic endometrial cells produces various free radicals, such as superoxide and hydroxyl anions, which trigger lipid peroxidation. As a result, MDA is formed as the end product of this reaction, which then acts as an indicator of systemic oxidative stress. Increased MDA is not only a marker of oxidative damage, but also contributes to endometrial cell proliferation, adhesion, and angiogenesis through activation of the NF-κB pathway and increased VEGF expression, thereby reinforcing the pathogenesis of endometriosis (26). Table 4 shows that there is a significant relationship between vitamin D deficiency and the incidence of endometriosis. These results are consistent with research by van Tienhoven et al. (2025), who also found that women with vitamin D deficiency (vitamin D levels <20 ng/mL) had twice the risk of developing endometriosis compared to those with normal levels (27). Vitamin D is known to play an important role in the immune and inflammatory systems, two key aspects that are also involved in the pathogenesis of endometriosis. Several studies suggest that low vitamin D levels may increase the risk of chronic inflammation, including the formation of endometriotic lesions. Vitamin D supplementation significantly improves endometrial receptivity and live birth rates in women with infertility disorders, many of which are associated with endometriosis. This study also notes the high prevalence of vitamin D deficiency and suggests supplementation as part of the management approach for patients with a history or risk of endometriosis (28). Table 4 shows the SOD status in patients with endometriosis and the control group. Based on the ROC curve of SOD, the threshold of SOD is 29.8 ng/mL with a sensitivity of 38.5% and specificity of 37.2% in diagnosing endometriosis. Furthermore, an analysis was conducted to examine the relationship between SOD based on the ROC curve and the incidence of endometriosis, yielding a p-value of 0.05, indicating no significant difference between the two groups. To date, no studies have determined a specific SOD threshold for endometriosis. Table 5 shows the MDA status in patients with endometriosis and the control group. Based on the ROC curve of MDA, the MDA threshold was found to be 24.1 ng/mL with a sensitivity of 62.0% and specificity of 60.5%. Furthermore, an analysis was conducted to examine the relationship between MDA based on the ROC curve and the occurrence of endometriosis. A significant difference in MDA levels was found between the endometriosis group and the control group (p=0.03, with p<0.05). To date, no studies have determined a specific MDA threshold for endometriosis. This study has several limitations. Blood samples were collected preoperatively without standardization to the menstrual cycle phase, which may have influenced oxidative stress markers and vitamin D levels. In addition, the severity and stage of endometriosis were not systematically assessed using standardized classifications such as rASRM and/or ENZIAN, nor were detailed phenotypic subtypes documented, precluding subgroup analyses based on disease stage or phenotype. Furthermore, potential confounding factors affecting vitamin D, SOD, and MDA levels including dietary intake, physical activity, sun exposure, and comorbid medical conditions were not fully evaluated.

Conclusion

This study concluded that endometriosis patients had lower vitamin D levels, decreased superoxide dismutase, and increased oxidative stress, specifically malondialdehyde.

Ethic approval: This study has been approved by the Research Ethics Committee of the Faculty of Medicine, Hasanuddin University, through the issuance of an ethics approval letter Number: 1056/UN4.6.4.5.31/PP36/2024. This study complies with ethical principles, ensuring the protection of participants' rights and confidentiality.

Conflict of interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

Authors contribution: LAVK, FM, and ADK drafted the manuscript. FM, ADK, NA, and DL designed and concepted the study. LAVK and IAF collected and analyzed and interpreted the data. FM, NA, and DL revised manuscript critically for important intellectual content. All authors participated in the final draft preparation, manuscript revision, and critical evaluation of the intellectual contents. All authors have read and approved the content of the manuscript and confirmed the accuracy or integrity of any part of the work.

Declaration on the use of AI: None

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