Vol 10 No 1, 2026

ANDALAS OBSTETRICS AND GYNECOLOGY JOURNAL

Correspondence Address:
Editorial Room Andalas Obstetrics and Gynecology Journal, 2nd Floor Obstetrics and Gynecology Department. M. Djamil Central General Hospital Padang, Jl. Perintis Kemerdekaan Padang, West Sumatra 25127
Website:
http://jurnalobgin.fk.unand.ac.id/

eISSN : 2579-8324 pISSN : 2579-8413

RESEARCH ARTICLE

The Relationship Between Microplastic Samples in Blood and The Stage of Epithelial Ovarian Cancer at Dr. M. Djamil Padang General Hospital

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Submitted: September 23, 2025; **Accepted:** January 20, 2026; **Published:** February 02, 2026
DOI: https://doi.org/10.25077/aoj.10.1.108-116.2026

Abstract

Introduction: *Ovarian cancer is the primary cause of cancer-related mortality in women, with epithelial ovarian cancer (EOC) ranking as the second greatest cause of death among gynaecological malignancies. The rising use of plastics has resulted in increased microplastic exposure among the population. This exposure aligns with recognised risk factors for ovarian cancer, including genetic, hormonal, and lifestyle elements. This study aimed to investigate the relationship between microplastics in blood samples and the stage of epithelial ovarian cancer at Dr. M. Djamil General Hospital, Padang.*

Methods: *Between August 2024 and January 2025, a cross-sectional analytical correlational investigation was carried out. Forty individuals with histopathologically verified epithelial ovarian carcinoma were consecutively recruited. Blood samples were examined for di(2-ethylhexyl) phthalate (DEHP) microplastics utilizing Liquid Chromatography–Mass Spectrometry (LCMS). Data were evaluated utilizing descriptive statistics and the Chi-square test, with $p < 0.05$ being significant.*

Results: *The average age of patients was 49.1±10.59 years, with the majority being multiparous, married, and of normal weight. The histological subtypes comprised mucinous (37.5%), serous (30%), endometrioid (20%), and clear cell (12.5%). Microplastics were identified in 30% of blood samples, with the greatest prevalence observed in mucinous (40%) and endometrioid (37.5%) subtypes. Statistical study indicated no significant correlation between microplastic occurrence and the stage of epithelial ovarian cancer ($p = 0.869$).*

Conclusion: *Microplastics were identified in the blood of around one-third of patients with epithelial ovarian cancer, present across all histological subtypes. However, no substantial link was identified between the presence of microplastics and the cancer stage.*

Keywords: *Ovarian cancer, microplastics, staging, epithelial subtype*

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INTRODUCTION

Ovarian cancer is the leading cause of death in women who suffer from cancer.[1] In 2020 there were 313,959 new cases of ovarian cancer with 207,252 deaths, making ovarian cancer one of the 10 cancers with the most deaths in 2020. GLOBOCAN 2020 data stated that 54.4% of cases occurred in Asia.[2]

Epithelial ovarian cancer (EOC) is the second leading cause of mortality among gynaecological malignancies. Annually, there are 313,959 new cases globally (1.6% of all cancers) and 207,252 deaths (2.1% of all cancer fatalities). The age-standardized incidence rate is 6.6 per 100,000, while the death rate is 4.2 per 100,000. EOC is seen in 75% of cases with advanced-stage illness. Notwithstanding advancements in treatment techniques like as significant surgical cytoreduction and novel adjuvant therapies, the overall survival rates remain poor at 40% for stage III and 20% for stage IV.[3] Epithelial ovarian cancer is categorised as type 1 and type 2. Epithelial ovarian cancer type 1 is thought to arise from benign lesions external to the ovaries that become incorporated into the ovary and exhibit mutations leading to malignant transformation. Endometrial tissue in the ovary (endometriosis) is hypothesised to be a precursor to type 1 epithelial ovarian cancer, particularly clear cell carcinoma (CCC) and endometrioid carcinoma (EC).[4]

Microplastics (MP) and nanoparticles, typically described as plastic particles ranging from 1 mm to 5 mm, are a category of developing environmental pollutants acknowledged for their potential effects on human health. Microplastics can enter the human body through ingestion, inhalation, and dermal contact. MP have been found in human organs, blood, and placentas. The comprehensive health impacts of microplastic exposure remain inadequately measured. Nonetheless, preliminary evidence suggests associations with inflammation, immunological disturbance, and potential carcinogenicity. [5]

The physical and chemical interactions that MP have with living species, including humans, can make it difficult for those organisms to carry out their cellular processes. MP can produce stress in cells by generating reactive oxygen species (ROS) and causing damage to proteins, lipids, RNA, and DNA.[5] MPs have been found to accumulate in the granulosa cells and ovaries of rats, which results in a reduction in the formation of the follicles, a decrease in the levels of anti-mullerian hormone (AMH), and oestradiol, as well as an irregular oestrous cycle and aberrant folliculogenesis. Granulosa cells are the key somatic cells of the ovary that are crucial for appropriate ovarian growth and maturation.[6]

The increasing use of plastics has led to heightened microplastic exposure throughout the population. This exposure runs parallel to established risk factors for ovarian cancer, such as genetic, hormonal, and lifestyle factors. However, there is a scarcity of studies investigating the link between microplastics and ovarian cancer. Based on this background, this study aims to investigate the "Relationship Between Microplastics in Blood Samples and The Stage of Epithelial Ovarian Cancer at Dr. M. Djamil General Hospital."

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METHODS

This analytical correlational study utilized a cross-sectional design to investigate the association between microplastics and the stage of epithelial ovarian cancer. The study was conducted from August 2024 to January 2025, with participant recruitment at Dr. M. Djamil General Hospital, Padang, and sample analysis at the Faculty of Medicine, Yarsi University, Jakarta.

Participants were recruited via consecutive sampling from patients with histopathologically confirmed epithelial ovarian cancer. Eligible participants were ≥18 years old with complete medical records who provided informed consent. Patients were excluded if they had a history of other cancers, significant systemic diseases, a Body Mass Index (BMI) > 24.9 kg/m², or menstrual disorders. The minimum sample size was calculated to be 26 based on a 95% confidence level and an 8.6% estimated prevalence.[7] To account for dropouts, the target sample size was set at 40.

The independent variable was the presence of DEHP/phthalate microplastics in blood, determined as 'detected' or 'not detected' by Liquid Chromatography-Mass Spectrometry (LCMS). The dependent variable was epithelial ovarian cancer, confirmed by histopathology.

Following informed consent, a 5 ml venous blood sample was collected, centrifuged, and the supernatant was stored at -20°C until analysis. At Yarsi University, samples were analyzed for DEHP/phthalate using a validated LCMS protocol, with the analyst blinded to all clinical information.

Data were analyzed using SPSS. Univariate analysis was performed to describe sample characteristics, while the Chi-square test was used to assess the association between microplastic presence and the stage of epithelial ovarian cancer. A p-value < 0.05 was considered statistically significant. This study received ethical approval from the Research Ethics Committee of the Faculty of Medicine, Andalas University. All participants provided written informed consent, and patient confidentiality was maintained by anonymizing all data.

RESULT

The characteristics of the patients can be seen in Table 1. In this study, the mean age of ovarian cancer patients was found to be 49.1±10.59 years. Regarding Body Mass Index (BMI), 77.5% of patients were classified as normal weight, while 22.5% were underweight. Most patients were housewives, followed by civil servants (15%), unemployed individuals (7.5%), farmers/laborer (5%), and private sector employees (5%). A significant portion of the patients were married (95%), with 10% being divorced and 5% unmarried. In terms of parity, most patients were multiparous (55%), followed by nulliparous (25%) and primiparous (20%). All the subjects (100%) had no history of contraceptive use. The histological distribution of epithelial ovarian cancer subtypes was as follows: 30% serous, 37.5% mucinous, 12.5% clear cell, and 20% endometrioid. No cases of mixed-type ovarian cancer were identified in this study. Most of the subjects (77,5%) are in the early stage of ovarian cancer.

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Table 1. Characteristics of The Ovarian Cancer Patients at Dr. M. Djamil General Hospital Padang

Characteristics	Mean ± SD / F (%)
Age (Years)	49.1 ± 10.59
Body Mass Index (BMI)	
Underweight	9 (22.5)
Normoweight	31 (77.5)
Occupations	
Unemployed	3 (7.5)
Housewife	27 (67.5)
Farmer/Laborer	2 (5)
Civil Servants	6 (15)
Private Sector Employee	2 (5)
Marital Status	
Married	34 (85)
Divorces	4 (10)
Unmarried	2 (5)
Parity	
Nulipara	10 (25)
Primipara	8 (20)
Multipara	22 (55)
History of Contraceptive	
Yes	-
No	40 (100)
Ovarian Cancer Subtype	
Serous	12 (30)
Mucinous	15 (37,5)
Clear cell	5 (12,5)
Endometrioid	8 (20)
Mixed	-
Ovarian Cancer Stage	
Early stage	31 (77,5)
Late stage	9 (22,5)

Out of 40 blood samples from patients, microplastics were detected in 12 patients (30%). Microplastics were found across all studied subtypes of epithelial ovarian cancer. The highest prevalence was observed in patients with the mucinous type (40%), followed by the endometrioid type (37.5%), clear cell type (20%), and serous type (16.7%) (Table 2.).

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Table 2. Microplastic Content in Blood Samples of Epithelial Ovarian Cancer Patients According to The Cancer Stage

Epithelial Ovarian Cancer	Number of Patients (n)	Microplastic Detected (n)	Microplastic Detected (%)
Early stage	31	10	32.3%
Late stage	9	2	22.2%
Total	**40**	**12**	**30.0%**

Chi-square test results (Table 3) showed no statistically significant association between the presence of microplastics in the blood and the stage of epithelial ovarian cancer (p=0.869). Although microplastics were detected at both stages, their distribution did not show a significant difference between groups.

Table 3. Association Between The Presence of Microplastics in The Blood and The Stage of Epithelial Ovarian Cancer

Epithelial Ovarian Cancer	Microplastics (%)		p value
	Detected	Not Detected	
Early stage	10 (32.3)	21 (67.7)	0.869
Late stage	2 (22.2)	7 (77.8)	
	12 (30)	28 (70)	

DISCUSSION

Microplastics were detected in 12 patients (30%). Microplastics were found across all studied subtypes of epithelial ovarian cancer with the highest prevalence was observed in patients with the mucinous type (40%). Montano et al. (2025) discovered that microplastics (dimensions <10 µm) were present in 14 of 18 follicular fluid samples, exhibiting an average quantity of 2191 particles per millilitre (range 0 - 7181 particles per millilitre) and a mean diameter of 4.48 µm (range 3.18 - 5.54 µm). A plausible mechanism elucidating the presence of MPs in human ovarian follicular fluid may involve the translocation of MPs from the bloodstream to the follicle, thereby traversing the blood-follicle barrier, which is comparatively more dynamic and less stable than the blood-testicular barrier, potentially resembling the placental barrier. Consequently, microplastics may infiltrate the ovaries via the circulatory system, targeting the granulosa cells and potentially inducing genotoxic effects and oxidative stress directly within ovarian tissue. The largest cells in the follicle, ovarian granulosa cells (GCs), are tasked with the secretion of oestrogen and progesterone. Granulosa cell proliferation and differentiation influence all principal ovarian functions, encompassing follicular growth and development, ovulation, luteal creation, and steroid hormone synthesis. MPs have been shown to infiltrate rat ovarian granulosa cells, leading to a significant reduction in blood concentrations of anti-Mullerian hormone (AMH) and oestradiol (E2) expression, and increased expression of fibrosis markers. Previous studies have demonstrated that persistent inflammation

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and fibrotic remodelling are recognised risk factors for carcinogenesis.[8]

Study from Cobellis et al. (2003) discovered that women with endometriosis exhibited significantly elevated plasma DEHP concentrations compared to the control group (median 0.57 mcg/ml, interquartile range: 0.06-1.23; range: 0-3.24 versus median 0.18 mcg/ml, interquartile range: 0-0.44; range: 0-1.03; P = 0.0047), with 92.6% of these women having detectable DEHP and/or MEHP in peritoneal fluid.[9] The correlation between endometriosis and endometrioid ovarian carcinoma is well-documented. Endometriosis, especially ovarian endometriomas, is regarded as a possible precursor for endometrioid and clear cell ovarian carcinoma. This may result from elevated oestrogen levels in the condition, causing malignant development of endometriotic cysts, or from mutations in the ARID1A gene, leading to the subsequent loss of BAF250a expression. It is hypothesised that mechanisms exist linking Endocrine-Disrupting Chemicals (EDCs) from microplastic exposure to oestrogenic effects that may induce ovarian cancer.[10]

Currently, there is no direct human evidence in the peer-reviewed literature correlating microplastic levels in the blood of ovarian cancer patients with cancer stage (FIGO I–IV). The present study found no statistically significant association between the presence of microplastics in the blood and the stage of epithelial ovarian cancer. This observation contrasts with reports in other malignancies, the research by Xu et al. (2024) on microplastics in the blood of cervical patients demonstrated that the diversity and abundance of microplastics were higher in blood samples compared to malignant tissues, with a strong positive correlation in diversity ($p < 0.05$). Furthermore, the variety and quantity of MPs in malignant tissues surpassed those in nearby non-malignant tissues.[25] Researchers performed a comparable investigation using a murine model. Tumorigenesis was seen in mice with the administration of microplastic-infused chow or exposure to microplastic-laden surroundings. Exposure to polystyrene nanoparticle microplastics (PS-NPs) was observed to expedite the progression of epithelial ovarian cancer in mice, while in vitro studies demonstrated that PS-NPs diminished the relative viability of human epithelial ovarian cancer (EOC) cells in a dose-dependent manner. The findings indicate that microplastic exposure may be linked to the formation of specific tumour types.[10]

This study has several limitations. First, the relatively small sample size and unequal distribution between early and late-stage ovarian cancer cases may have limited statistical power to detect subtle associations between blood microplastic levels and cancer stage. Second, its cross-sectional design precludes any inference of causality or temporal relationship between microplastic exposure and tumor progression. Third, microplastic detection was restricted to blood samples analyzed by LC–MS for DEHP, which measures a specific plasticizer compound rather than total microplastic particle burden, size, or polymer type. The absence of tissue-based analysis and healthy controls further limits comparison. Additionally, potential confounding factors such as environmental exposure, dietary habits, and prior treatment history were not controlled. As most participants were diagnosed at early stages and recruited from a single center, the findings should be interpreted cautiously and validated in larger multicenter studies using comprehensive microplastic quantification methods.

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CONCLUSION

The research conducted at Dr. M. Djamil General Hospital concludes that the average age of ovarian cancer patients is 49.1 years. The majority of these individuals are of normal weight, homemakers, married, multiparous, and diagnosed with the mucinous subtype. Microplastics were identified in the blood samples of 30% of women with epithelial ovarian cancer within this population. The investigation indicated no statistically significant correlation between the presence of microplastics in the blood and the stage of epithelial ovarian cancer.

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