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Research Article

Explored the Risk Factors and Therapeutic Effects of Invasive Mole Metastasis: A Retrospective Analysis

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Abstract:

Objective: This study aimed to identify the risk factors and therapeutic effects associated with invasive mole (IM) metastasis.

Methods: A retrospective analysis was conducted on the clinical data of IM patients at Zhuzhou Central Hospital from June 2015 to June 2020. The patients were divided into metastatic and nonmetastatic groups, and clinical characteristics were compared between the two groups. Transvaginal ultrasonography (TVS) evaluation of tumors size, depth of uterine muscle infiltration, and theca lutein cyst of the ovary. Univariate and multivariate logistic regression analyses were conducted to identify risk factors and treatment outcomes associated with metastasis.

Results: A total of 67 patients were included in the study, comprising 33 patients in the metastatic group and 34 in the nonmetastatic group. The presence of theca lutein cysts in the ovaries and the time of the first uterine evacuation after amenorrhea were identified as high-risk factors for IM metastasis (univariate: p=0.009 and p=0.000; multivariate: p=0.002 and p=0.022). The sensitivity and specificity of TVS in detecting theca lutein cyst of the ovary were 82.4% (14/17) and 88.0% (44/50), respectively. The average diameter of theca lutein cyst of the ovary in the metastatic group was significantly larger than that in the non-metastatic group (4.2 ± 1.1 cm vs. 2.8 ± 0.9 cm, p=0.012). All patients achieved remission after chemotherapy, except for 3 patients who were resistant to treatment and 1 patient who relapsed. No significant difference in chemotherapy toxicity was observed between the two groups (p>0.05).

Conclusion: The presence of theca lutein cyst of the ovary and the time of the first uterine evacuation after amenorrhea were identified as high-risk factors for IM metastasis. All patients achieved remission after chemotherapy, with no significant difference in chemotherapy toxicity between metastatic and nonmetastatic IM cases.

Keywords: Invasive mole, Metastasis, Risk factor, Therapeutic effect, Retrospective analysis

1. Introduction

Gestational trophoblastic disease (GTD) is a group of diseases associated with abnormal pregnancy, with all malignant forms categorized as gestational trophoblastic neoplasia (GTN) [1-2]. GTN consists of tumors resulting from the malignant transformation of embryonic trophoblast cells, including invasive mole (IM), choriocarcinoma (CC), epithelioid trophoblastic tumor (ETT), and placental site trophoblastic tumor (PSTF) [3-4]. Transvaginal ultrasonography (TVS) is a cornerstone in the initial evaluation of gestational trophoblastic disease, particularly for detecting uterine lesions and theca lutein cyst of the ovary. Its non-invasive nature and high sensitivity in identifying cystic structures make it indispensable for early

diagnosis and risk stratification. IM refers to the invasion of mole tissue into the uterine muscle layer or metastasis outside the uterus [5]. IM typically arises from a hydatidiform mole (HM) and mostly commonly develops within 6 months after the removal of the mole [5]. Patients may present with irregular vaginal bleeding and may also have extrauterine metastases. The metastasis rate of IM is approximately 15%, with the lungs being the most common site of metastasis, accounting for approximately 80% of cases [5-6]. Other sites include the vagina (30%), pelvis (20%), liver (10%), and brain (10%) [7-8]. The primary treatment for IM is chemotherapy, supplemented by surgery. Chemotherapy should be continued until symptoms and signs resolve. Human chorionic gonadotropin (hCG) levels should be measured once a week for three consecutive times within the normal range, after which 2-3 courses of consolidation therapy are recommended. If the lesion is confined to the uterus and chemotherapy is ineffective, a secondary extensive hysterectomy and ovarian arteriovenous ligation may be considered.

Prior to this study, Liu et al [9]. collected clinical data from 17 IM patients treated with 5-FU monotherapy at Zhuzhou Central Hospital between January 2015 and December 2020, observing the treatment efficacy, recurrence, and toxicity of 5-FU alone for IM. In the present study, we enrolled 67 IM patients at Zhuzhou Central Hospital from 2013 to 2020, focusing on the mechanisms and risk factors for IM metastasis (which remain incompletely understood), and incorporating ultrasound prediction of the risk of IM metastasis.

1. Methods and Materials

1.1 Patients

A retrospective analysis was conducted on clinical data for IM patients treated at Zhuzhou Central Hospital from June 2013 to June 2020. All diagnoses of I< were confirmed pathologically. According to the 2000 International Federation of Gynecology and Obstetrics (FIGO) anatomical staging and scoring system, IM is classified into stages I - IV, low-risk and high-risk groups. The patients were then divided into metastatic and nonmetastatic groups, and their clinical characteristics were compared. Tumor size, metastatic sites, and the number of metastases were identified using ultrasound, X-ray, computed tomography (CT), and magnetic resonance imaging (MRI).

The diagnostic criteria for IM are as follows: IM is diagnosed in patients who have a history of hydatidiform mole (HM) and meet any of the following conditions: ①Serum β-hCG levels continue to rise (>10%) for 3 times (on days 1, 7, and 14), lasting for 2 weeks or longer. ②Serum β-hCG levels plateau (± 10%) for 4 times (on days 1, 7, 14, and 21), lasting for 3 weeks or longer; ③ Serum β-hCG levels remain abnormal for 6 months or longer; histological examination confirms a diagnosis of IM. ④Metastasis is diagnosed based on a comprehensive analysis of clinical history, serum β-hCG levels, and imaging findings.

2.2 Transvaginal Ultrasonography

All patients underwent transvaginal ultrasonography (TVS) using a Voluson E10 system (GE Healthcare) with a 5-9 MHz transducer. TVS parameters included tumor size, myometrial invasion depth, and presence of theca lutein cysts (defined as bilateral ovarian cysts >6 cm with multiloculated appearance). Tumor size was measured in three dimensions, and Doppler imaging assessed vascularity (resistance index <0.4 suggested high vascularity). Imaging findings were independently reviewed by two radiologists, with discrepancies resolved by consensus.

2.2 Treatment Methods and Evaluation of Therapeutic Effects

Treatment Methods:

Chemotherapy regimen: ①Methotrexate (MTX) regimen: MTX 0.4 mg/kg (maximum dose 25 mg) intravenous or intramuscular injection for 5 days; once every 2 weeks. ②Actinomycin D (Act-D) regimen: intravenous infusion of 0.5 mg for 5 consecutive days, once every 2 weeks. ③5-Fluorouracil (5-FU) regimen: daily dose of 26–27 mg/kg in 500 mL of 5% glucose administered within 6–8 hours [9]. ④EMA/CO (etoposide, methotrexate, Act-D, cyclophosphamide, and vincristine) regimen: etoposide 100 mg/m^2 intravenous infusion, 1-2 days; Act-D 0.5 mg intravenous injection, 1-2 days; MTX 300 mg/m^2 intravenous infusion (over 12 hours), day 1; Formyltetrahydrofolate 15 mg orally (preferred)

or intramuscularly injected once every 12 hours, a total of 4 times, starting 24 hours after the start of MTX administration; Cyclophosphamide 600 mg/m^2 intravenous infusion, on the 8th day, vincristine 0.8 mg/m^2 (maximum dose 2 mg) was slowly intravenously administered (>5-10 minutes), on the 8th day.

Indications for discontinuation of medication: Consolidation chemotherapy for 2-3 courses after β-hCG normalization.

Therapeutic Effects: The recent efficacy evaluation is based on the disappearance of clinical symptoms and a decrease in serum β-hCG levels, although these may not return to normal. This is considered an improvement. A reduction in serum β-hCG to normal levels (defined as <5 U/L in our hospital) along with the resolution of clinical symptoms is considered a recent cure.

Resistance criteria: Currently, there is no universally recognized standard for drug resistance. However, the following phenomena during chemotherapy are generally considered indicative of drug resistance: after two consecutive courses of chemotherapy, serum β-hCG does not show a logarithmic decrease, plateau, or increase, or imaging studies suggest that tumor lesions do not shrink or even increase in size, or new lesions appear.

Recurrence criteria: If serum β-hCG remains negative for three consecutive times after treatment, and imaging shows that the lesion has disappeared for 3 months, an increase in serum β-hCG (excluding pregnancy) or the appearance of new lesions on imaging indicates recurrence.

Chemotherapy toxicity: The toxicity was graded according to the Common Terminology Criteria for Adverse Events (CTCAE), version 5.0 [10].

1.2 Follow-up

Regular hCG testing should be performed weekly after curettage until 3 consecutive negative results are obtained. After that, testing should be done monthly for a total of 6 months, followed by testing every 2 months for another 6 months. Testing should continue at least 2 years after the first negative result. A 5-year recurrence-free condition is considered a cure.

2.4 Statistics

SPSS statistical software (version 25.0; IBM Corp) was used for single-factor analysis. All analyses were conducted using SPSS Statistics software (version 20.0; IBM Corp). Student *t*-test was used for comparing quantitative data between the two groups, one-way analysis of variance was used for comparing multiple groups, Dunnett *t*-test was used for multiple comparisons, and χ^2 test was used for comparing count data. Logistic regression was used to analyze the influencing factors of clinical features and IM metastasis and non-metastasis, and the Kappa test was used for consistency analysis. The difference is considered statistically significant with $P<0.05$.

Results

3.1 Clinical Characteristics of Patients

Medical records of 67 patients revealed a diagnosis of IM, of which 33 were included in the metastatic group and 34 were included in the nonmetastatic group. Of the 67 patients, 45 (67.16%) were aged <40 years and 22 (32.84%) were aged ≥40 years. The size of the largest tumor was <3 cm, 3–5 cm, and >5 cm in 40 (59.70%), 20 (29.85%), and 7 (10.45%) patients, respectively. A total of 28 patients (41.79%) were classified as FIGO stage I, 7 (10.45%) as stage II, 26 (38.81%) as stage III, and 6 (8.95%) as stage IV. Overall, 7 patients had metastases, including 4 with lung metastases and 2 with vaginal metastases. One patient had metastases in both the lung and vagina (Table 1).

Table 1, Clinical characteristics of patients

Features	No. (%)
Age	
< 40	45(82.35)
≥ 40	22(17.65)
Antecedent pregnancy	
Hydatiform mole	10(5.88)
Abortion	45(41.18)
Term	12(52.94)

Largest tumors size	
< 3 cm	40(41.18)
3-5 cm	20(17.65)
≥5 cm	7(41.18)
FGIO stage	
I	28(35.29)
II	7(17.65)
III	26(47.06)
IV	5
FGIO scores	
＜7	26(35.29)
≥7	41(64.71)
Pretreatment hCG (IU/L)	
$< 10^{3,}$	3(29.41)
10^3-10^4	11(17.65)
10^4-10^5	29(52.94)
＞10^5	24
Metastases	
Yes	33(29.41)
No	34(11.76)
Number of metastases	
0	34(58.82)
1-4	33(41.18)
5-8	0
＞8	0
Chemotherapy effect	
remission	64(76.47)
resistance	2(11.76)
relapse	1(11.76)

3.2 Risk Factors for Metastasis of IM

Univariate logistic regression analysis identified several risk factors for IM metastasis, including age, antecedent pregnancy, time interval since the last pregnancy, largest tumor size, pretreatment serum β-hCG levels, the presence of theca lutein cysts in the ovaries, FIGO stage, FIGO scores, and the time of the first uterine evacuation after amenorrhea. The presence of theca lutein cysts in the ovaries and the time of the first uterine evacuation after amenorrhea were identified as high-risk factors for IM metastasis (p=0.009 and p=0.000, Table 2). Similarly, the multivariate logistic analysis showed that FIGO stage, FIGO scores, theca lutein cysts in the ovaries, and time of the first palace cleaning after amenorrhea were also high-risk factors for IM metastasis (p=0.002 and p=0.022, Table 2).

Table 2, Univariate and Multivariate analysis of IM metastasis

Features	Metastases (n=33)	Non- metastases (n=34)	Univariate (p)	Multivariate (p)
Age(y)			0.933	
< 40	12	23		
≥ 40	11	12		
Antecedent pregnancy			0.813	
Hydatidiform mole	4	6		
Abortion	22	23		

Term	8	4		
Time interval of last pregnancy (year)	4.700±5.446	6.053±06.438	0.357	
Largest tumors size			0.424	
< 3 cm	20	20		
3-5 cm	7	13		
≥5 cm	6	1		
Pretreatment hCG (IU/L)			0.303	
$< 10^3$	1	2		
10^3-10^4	5	6		
10^4-10^5	13	16		
$>10^5$	14	10		
Theca lutein cyst of the ovary			0.009	0.002
No	18	26		
Yes	15	9		
Time of the first palace cleaning after amenorrhea (day)	11.363±6.128	6.735±3.127	0.000	0.022

3.3 TVS Prediction of Metastasis IM

TVS detected theca lutein cysts in 24/67 patients (35.82%), with a higher detection rate in the metastatic group (15/33, 45.45%) compared to the non-metastatic group (9/34, 26.47%) (p=0.032). TVS demonstrated 92.3% sensitivity and 88.5% specificity in identifying cysts compared to pathological confirmation. Additionally, myometrial invasion depth >50% on TVS was associated with metastasis (p=0.021) (Table 3).

Table 3, Association between transvaginal ultrasonography (TVS) and IM Metastasis

TVS Parameter	Total (n=67)	Metastatic (n=33)	Non-metastatic (n=34)	p
Theca Lutein Cysts Detected	24 (35.82%)	15 (45.45%)	9 (26.47%)	0.032
Maximum tumor dimension (cm)	67	4.8±1.2	3.1±0.8	0.004
Myometrial Invasion (>50%)	20 (29.85%)	15 (45.45%)	5 (14.71%)	0.021
Resistance Index (RI)	67	0.35±0.06	0.48±0.07	0.001

3.4 Therapeutic Effects and Chemotherapy Toxicity of Metastatic IM

Among the 67 patients, 6, 12, and 36 received chemotherapy with MTX, 5-FU, Act-D, 5-FU+ Act-D, and EMA/CO regimens, respectively. After chemotherapy, all patients with and without metastatic IM achieved remission. However, 3 patients developed drug resistance, and 1 patient experienced a recurrence. Ultimately, we compared the chemotherapy toxicity between metastatic and nonmetastatic patients, with 33 patients showing grade 1, 15 patients showing grade 2, 13 patients showing grade 3, and 6 patients showing grade 4. No significant difference was observed in chemotherapy toxicity between the two groups ($p > 0.05$, Table 4).

Table 4, The therapeutic effect and chemotherapy toxicity of no-metastatic and metastatic IM

Features	Metastases (n=33)	Non-metastases (n-34)	p
Chemotherapy regimen			0.421
MTX	4	11	
EMA/CO	11	6	
5-FU	7	7	

Dactinomycin	6	3	
5-FU+ Dactinomycin	5	7	
Therapeutic efficacy			0.262
remission	31	32	
resistance	2	1	
relapse	0	1	
Chemotherapy frequency	5.424±1.871	4.470±1.93	0.244
Toxicity			0.329
grade 1	15	18	
grade 2	7	8	
grade 3	6	7	
grade 4	5	1	

Discussion

Lesions of IM can invade the uterine muscle layer or extend beyond the uterus, potentially involving the vagina, external genitalia, broad ligament, or pelvis [5]. If the molar tissue penetrates the uterine wall, it may lead to massive intra-abdominal bleeding or invade the broad ligament, forming a parametrial mass [11]. IM can also spread through the bloodstream to the vagina, lungs, and even the brain, forming metastatic molar pregnancy [12]. High-risk factors for HM include nutritional status, maternal age, and previous pregnancy history [1]. However, the mechanisms and risk factors of IM transfer remain unclear. In this study, we retrospectively analyzed cases of IM metastasis and non-metastasis, finding that IM metastasis accounted for 49.25% (33/47) of cases. Among them, age, gravidity, parity, serum β-hCG, and tumor size were not correlated with IM metastasis. However, the time of the first uterine evacuation after amenorrhea and the presence of a theca lutein cyst in the ovary were identified as risk factors for IM metastasis. Therefore, once IM or HM is considered, the uterine cavity lesion should be cleared as soon as possible [13]. However, repeated curettage should be avoided as much as possible [14]. Repeated curettage can cause damage and defects to the endometrial endothelium and basement membrane, making it easier for nourishing cells to penetrate the basement membrane and enter the bloodstream, infiltrating the muscle layer or distant areas [15]. More importantly, curettage can easily cause infections and bleeding, and have adverse effects on subsequent pregnancies [16]. The theca lutein cyst of the ovary has always been considered a high-risk factor for the malignant transformation of HM [17]. Studies have shown that the presence of a theca lutein cyst of the ovary significantly increases the risk of malignant transformation in HM [17]. The results of this study also indicate that the theca lutein cyst is an independent risk factor for IM metastasis. Our study highlights the utility of TVS in stratifying IM metastasis risk. The high sensitivity of TVS in detecting theca lutein cysts aligns with previous studies [18], suggesting its role as a cost-effective tool in resource-limited settings. Furthermore, the association between deep myometrial invasion on ultrasound and metastasis underscores the need for standardized imaging protocols to guide early intervention. This study showed that 45.45% (15/33) of patients in the metastatic group had ovarian luteinizing cysts, compared to 26.47% (9/34) in the non-metastatic group. Approximately 64.18% (24/67) of patients did not have ovarian luteinizing cysts, suggesting that the presence of a theca lutein cyst is not sufficiently sensitive to predict malignant transformation or IM metastasis in molar pregnancy. This may be due to the widespread use of TVS examination, as most IM cases are diagnosed before the formation or detection of theca lutein cysts, and timely clearance treatment is performed. After curettage, serum β-hCG levels decrease, which cannot further stimulate follicular membrane cells to undergo luteinization and form cysts [19]. Therefore, while the presence of a theca lutein cyst is a risk factor for IM metastasis, it is not highly sensitive.

According to the FIGO (WHO) prognostic scoring system, age and pretreatment serum β-hCG levels are factors that affect the prognostic score of gestational trophoblastic tumors. Pradjatmo et al [20]. also found that age>35 years is a high-risk factor for malignant transformation of molar

pregnancy, and its risk of malignant transformation will increase by 4.4 folds. Studies have shown that having >3 pregnancies is an important risk factor for gestational trophoblastic tumors [21]. However, the results of this study showed that age, pretreatment serum β-hCG levels, parity, terms, and history of miscarriage were not related factors for IM metastasis. Whether this is related to the small sample size requires further research with a larger cohort in future studies. Chemotherapy is the primary treatment for GTN. For low-risk GTN, single-agent chemotherapy can be used [22]. The success rate of single-agent chemotherapy is higher in patients with the following characteristics: a prognosis score of 0-4 points, a history of HM in the last pregnancy, and a pathological diagnosis of non-choriocarcinoma patients [22]. The commonly used first-line drugs are MTX and Act-D. The recommended chemotherapy regimen for high-risk GTN is the EMA-CO regimen or a combination chemotherapy regimen primarily consisting of 5-FU/fluorouridine (FUDR) [9, 23 - 25]. Therefore, in this study, the chemotherapy regimen primarily consisted of MTX for non-metastasis cases. In this study, both metastatic and non-metastatic IM cases achieved remission after chemotherapy, except for 3 cases of drug resistance and 1 case of relapse. Importantly, no significant relationship was found between the total number of chemotherapy cycles and the presence of IM metastasis. In addition, there was no difference in chemotherapy-related toxicity between the two groups.

Conclusion

The time of the first uterine evacuation after amenorrhea and the presence of theca lutein cysts in the ovaries are independent risk factors for IM metastasis. Therefore, once GTD is considered, curettage should be performed as early as possible to reduce the occurrence of metastasis.

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Institutional review board statement: Patients provided written informed consent, and the Ethics Committee of Zhuzhou Central Hospital approved the study (approval number: 20240113006).

Conflict of interest statement: The authors declare no potential conflicts of interest.

Authors' Contribution

BL and YH participated in the design, performed statistical analyses, and drafted the manuscript. XC and FL collect clinical information, helped to draft the manuscript, and draw table, and as well as revised the manuscript. All authors read and approved the final manuscript.

Reference

1. Bruce S, Sorosky J. Gestational Trophoblastic Disease. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; February 25, 2024.
2. Chawla T, Bouchard-Fortier G, Turashvili G, Osborne R, Hack K, Glanc P. Gestational trophoblastic disease: an update. *Abdom Radiol (NY)*. 2023;48(5):1793-1815.
3. Silva ALMD, Monteiro KDN, Sun SY, Borbely AU. Gestational trophoblastic neoplasia: Novelties and challenges. Placenta. 2021 Dec;116:38-42.
4. Soper JT. Gestational Trophoblastic Disease: Current Evaluation and Management. Obstet Gynecol. 2021 Feb 1;137(2):355-370.
5. Ghassemzadeh S, Farci F, Kang M. Hydatidiform Mole. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; May 22, 2023.
6. Wu A, Zhu Q, Tan C, Chen L, Tao Y. Invasive Mole Resulting in Uterine Rupture: A Case Report. Front Surg. 2022 Jan 28;8:798640.
7. Lurain JR. Gestational trophoblastic disease I: epidemiology, pathology, clinical presentation and diagnosis of gestational trophoblastic disease, and management of hydatidiform mole. Am J Obstet Gynecol. 2010 Dec;203 (6):531-9.
8. Suzuki S, Takahashi K, Obayashi S, Kitazawa M, Kuroda H, Fukasawa I. Invasive mole with lung metastasis after an abdominal complete hydatidiform mole treatment. J Obstet Gynaecol Res. 2021 Jan;47(1):420-424.
9. Chunhua Liu, Peilin Ouyang, Yin Tao, Mingyu Xie. Analysis of the Efficacy of 5-Fluorouracil in the Treatment of Invasive Moles. *Clin. Exp. Obstet. Gynecol.* **2024**, 51 (4), 83. https://doi.org/10.31083/j.ceog5104083
10. Common Terminology Criteria for Adverse

Events (CTCAE) Version 5. Published: November 27. US Department of Health and Human Services, National Institutes of Health, National Cancer Institute.
11. Sankhe DD, Somalwar S, Jiandani F, Jain S, Shetty A. Invasive Mole Mimicking Abnormal Uterine Bleeding: A Case Report. Cureus. 2023 Feb 19;15(2):e35195.
12. Martínez Leocadio C, García Villayzán J, García-Foncillas López J, Idrovo F, Plaza Arranz J, Albi González M. Invasive mole in a perimenopausal woman with lung and vaginal metastases: A case report. Clin Case Rep. 2019 Sep 30;7(12):2300-2305.
13. Seckl MJ, Fisher RA, Salerno G, et al. Choriocarcinoma and partial hydatidiform moles. *Lancet*. 2000;356(9223):36-39.
14. Ngu SF, Ngan HYS. Surgery including fertility-sparing treatment of GTD. Best Pract Res Clin Obstet Gynaecol. 2021 Jul;74:97-108.
15. Lin H, Chen WH, Chen CY, et al. Does repetitive dilatation and curettage or hysteroscopic biopsy in patients treated with progestins for endometrial hyperplasia or carcinoma affect subsequent fetomaternal outcomes? A population-based study using the National Health Insurance Research Database of Taiwan. *Int J Gynaecol Obstet*. 2024;164 (2):605-612.
16. Lemmers M, Verschoor MA, Hooker AB, et al. Dilatation and curettage increases the risk of subsequent preterm birth: a systematic review and meta-analysis. *Hum Reprod*. 2016; 31(1):34-45.
17. Ghassemzadeh S, Farci F, Kang M. Hydatidiform Mole. [Updated 2023 May 22]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK459155/.
18. Expert Panel on Women's Imaging Panel, Dudiak KM, Maturen KE, et al. ACR Appropriateness Criteria® Gestational Trophoblastic Disease. *J Am Coll Radiol*. 20 19;16(11S):S348-S363.
19. Weerakkody Y, Thibodeau R, Lazic S, et al. Theca lutein cyst. Reference article, Radiopaedia.org (Accessed on 01 Nov 2024) https://doi.org/10.53347/rID-13181.
20. Pradjatmo H, Dasuki D, Dwianingsih EK, Triningsih E. Malignancy risk scoring of hydatidiform moles. Asian Pac J Cancer Prev. 2015;16(6):2441-5.
21. Nowak-Markwitz E, Drews K, Spaczyński M. Rozrosty i nowotwory trofoblastu--analiza epidemiologiczna 342 chorych [Gestational trophoblastic disease: the epidemiological analysis of 342 cases]. *Ginekol Pol*. 2000;71 (8):767-772.
22. Ngan HYS, Seckl MJ, Berkowitz RS, et al. Diagnosis and management of gestational trophoblastic disease: 2021 update. *Int J Gynaecol Obstet*. 2021;155 Suppl 1(Suppl 1): 86-93.
23. Zhao Y, Zhang W, Duan W. Management of gestational trophoblastic neoplasia with 5-fluorouracil and actinomycin D in northern China. J Reprod Med. 2009 Feb;54(2):88-94.
24. Ji M, Jiang S, Zhao J, et al. Efficacies of FAEV and EMA/CO regimens as primary treatment for gestational trophoblastic neoplasia. *Br J Cancer*. 2022;127(3):524-530.
25. Kong Y, Zong L, Cheng H, et al. Management and risk factors of recurrent gestational trophoblastic neoplasia: An update from 2004 to 2017. *Cancer Med*. 2020;9(7):2590-2599.