

## ORIGINAL RESEARCH

# Comparison of the clinical efficacy of paclitaxel + carboplatin and paclitaxel + cisplatin on tumor markers and WHOQOL-BREF score on cervical cancer patients

Shanliang Hu<sup>1</sup>, Chao Jiang<sup>1</sup>, Weiwei Zhang<sup>1,\*</sup>, Ping Fang<sup>1</sup>

<sup>1</sup>Department of Radiation Oncology, Huangshan City People's Hospital, 245000 Huangshan, Anhui, China

\*Correspondence  
w\_weiz0523@163.com  
(Weiwei Zhang)

## Abstract

This study aims to compare tumor marker indicators, World Health Organization Quality of Life-Brief assessment (WHOQOL-BREF) scores and clinical outcomes between cervical cancer patients treated with paclitaxel + carboplatin versus those treated with paclitaxel + cisplatin. 66 cervical cancer patients admitted to our hospital were randomly selected and allocated equally into a control group (paclitaxel + cisplatin) and a study group (paclitaxel + carboplatin) using a randomized double-blinded approach. Tumor marker indices, WHOQOL-BREF scores, Karnofsky Performance Status (KPS) scores, clinical outcomes and adverse effects were assessed and compared before and after treatment. The study group was found to have lower carcino-embryonic antigen (CEA), Carbohydrate antigen (CA) 199, CA125 and CA50 levels, significantly higher WHOQOL-BREF scores, and significantly higher KPS scores at 7 days, 1 month, 2 months and 3 months post-treatment compared to the control group (all  $p < 0.05$ ). However, we also observed that while the treatment effectiveness rate in the study group (75.76%) surpassed that in the control group (66.67%), the difference was statistically significant ( $p > 0.05$ ). Patients in the study group had a statistically significant lower incidence of diarrhea (45.45%) and nausea and vomiting (48.48%) compared to the control group, whose corresponding rates were higher at 69.70% and 75.76%, respectively ( $\chi^2 = 3.969, 5.215, p = 0.046, 0.022$ ). Conversely, the incidence of bone marrow suppression in the study group (48.48%) was significantly higher than that in the control group (21.21%) ( $\chi^2 = 4.405, p = 0.020$ ). We conclude that the combination of paclitaxel and carboplatin was an effective treatment approach for cervical cancer patients, offering comparative advantages over paclitaxel + cisplatin, with reduced tumor marker levels, enhanced quality of life, and minimized adverse reaction occurrence.

## Keywords

Paclitaxel; Carboplatin; Cisplatin; Cervical cancer; Therapeutic efficacy; Tumor marker levels

## 1. Introduction

Cervical cancer accounts for a relatively high proportion of gynecological malignancies, ranks second as the most prevalent gynecologic cancer in terms of both incidence and mortality, just behind breast cancer, and has been found to be closely associated with human papillomavirus infection and sexual activity [1]. Currently, various clinical treatment modalities are available for cervical cancer, including surgery and radiotherapy. In early-stage cases, total hysterectomy with pelvic lymph node dissection is commonly performed due to its associated favorable treatment outcomes and longer survival [2, 3]. However, in cases where cervical cancer progresses to intermediate or advanced stages, achieving optimal results through surgery becomes challenging. To address this, neoadjuvant chemotherapy can be employed effectively to suppress and

eliminate small migrating tumor foci, facilitating a reduction in tumor size and thereby increasing surgical resectability, which may ultimately improve patient survival outcomes.

Currently, in the clinical management of middle and advanced cervical cancer, the frequently prescribed neoadjuvant chemotherapy regimen comprises paclitaxel combined with platinum-based agents such as carboplatin and cisplatin, each with distinct clinical effectiveness profiles. In this study, we aimed to assess the differences in clinical efficacy between paclitaxel + carboplatin and paclitaxel + cisplatin in cervical cancer patients through comparative analyses in cervical cancer patients treated with different chemotherapy regimens at our hospital [4–6].

## 2. Information and methods

## 2.1 General information

We randomly selected 66 cervical cancer patients from the clinical admissions at our hospital and assigned them in a random and double-blind manner into a study group ( $n = 33$  cases) and a control group ( $n = 33$  cases). The baseline characteristics of the patients from both study groups are shown in Table 1, which indicates that both groups were well-balanced ( $p > 0.05$ ).

The study inclusion criteria comprised patients who had received a definitive diagnosis of cervical cancer through pathological biopsy, were newly diagnosed with cervical cancer, had normal blood counts and well-functioning liver and kidney profiles without apparent contraindications to chemotherapy, a Karnofsky Performance Status (KPS) score between 0 and 1, aged 30 to 60 years, were staged as IIb or higher according to the Figo classification, and were estimated to have a life expectancy exceeding 3 months.

Exclusion criteria comprised patients who were concurrently enrolled in other research studies, those who had undergone prior treatment with alternative chemotherapeutic agents before study entry, patients with concurrent malignancies or psychiatric disorders, those who had recently undergone surgical interventions, pregnant or breastfeeding women, and patients with hematological system abnormalities.

## 2.2 Methods

### 2.2.1 Control group

Control group: The control group received a combination of paclitaxel (Yunnan Hande Biotechnology Co., Ltd., National Drug Code: H10960322, Kunming, China) and cisplatin (Qilu Pharmaceutical Co., Ltd., National Drug Code: H37021358, Jinan, China). The total paclitaxel dosage administered ranged from 135 to 175 mg/m<sup>2</sup> as a single dose, and the total cisplatin dosage ranged from 60 to 70 mg/m<sup>2</sup>, given over 2–3 doses. Each treatment cycle comprised 21 doses, with a total of 3 treatment cycles.

### 2.2.2 Study group

Study group: The study group received a combination of paclitaxel and carboplatin. Paclitaxel was administered at a total dose ranging from 135 to 175 mg/m<sup>2</sup>, administered intravenously as a single dose. The carboplatin (Kunming Guiyan Pharmaceutical Co., Ltd., with a National Drug Code: H20053908, Kunming, China) dosage was calculated using an area under the curve (AUC) of 5 and was administered as a single intravenous dose. Each treatment cycle consisted of 21 doses, and a total of 3 treatment cycles were administered.

## 2.3 Indicator observation

### 2.3.1

The total effective rate of treatment is calculated as the sum of the apparent rate (percentage of cases where the tumor persisted for more than four weeks before disappearing) and the effective rate (percentage of cases where the tumor volume decreased by more than 50% and persisted for more than 4 weeks).

Efficacy was evaluated according to the Response Evalua-

tion Criteria in Solid Tumors (RECIST) criteria, where efficacy was categorized as: (1) complete response (CR) denoting the disappearance of all target and non-target lesions, along with normalization of tumor markers; (2) partial response (PR) indicating a reduction of  $\geq 30\%$  in the total sum of baseline lesion diameters; (3) stable disease (SD) signifying the absence of a reduction in PR or an increase in PD, in the presence of one or more non-target lesions and/or abnormal markers; and (4) progressive disease (PD) defined as a  $\geq 20\%$  increase in summed baseline lesion length and diameter, or the emergence of new lesions, and/or progression of non-target lesions. The total effectiveness rate was computed as the combined percentage of CR and PR cases.

### 2.3.2

Adverse reactions, including anorexia, alopecia, nausea and vomiting, bone marrow suppression, and others, were assessed and graded based on the criteria established by the World Health Organization (WHO) for toxicity and side effects (Table 2).

### 2.3.3

Detection and comparison of the levels of tumor markers (CEA, CA199, CA125 and CA50) before and after treatment.

### 2.3.4

The assessment of quality of life before and after treatment was conducted using the WHOQOL-BREF scale. This scale evaluates various aspects, including the environment, physical well-being, and social relationships, with each item assigned a maximum score of 20 points, resulting in a total score of 100 points. A higher score on the scale indicates a higher quality of life.

### 2.3.5

The health status of patients was assessed at different time points (prior to treatment, 7 days post-treatment, 1 month post-treatment, 2 months post-treatment and 3 months post-treatment) using the KPS (Karnofsky Performance Status) scale. This scale assigns a total score of 100, with the score being directly proportional to the patient's health status.

## 2.4 Statistical analysis

Data were analyzed using the SPSS (International Business Machines Corporation, Armonk, NY, USA) version 27.0. Descriptive statistics were used for normally distributed measurement data and shown as mean ( $\pm$ standard deviation), while non-normally distributed measurement data were described using the median (interquartile range) [M (Q1, Q3)]. For normally distributed data, the *t*-test was utilized, whereas the rank-sum test was used for non-normally distributed data. Counting data are presented as the number of cases and the corresponding percentage ( $n$  (%)). Group comparisons for counting data were conducted using the chi-square ( $\chi^2$ ) test.

## 3. Results

**TABLE 1. Comparison of general information ((%), ( $\bar{x} \pm s$ )).**

Basic information	Study Group (n = 33)	Control Group (n = 33)	$t/\chi^2$	$p$
Tumor Type				
Squamous carcinoma	20 (60.61)	22 (66.67)	0.261	0.608
Adenocarcinoma	13 (39.39)	11 (33.33)		
Age (yr)	45.82 $\pm$ 4.21	45.70 $\pm$ 4.25		
Body weight (kg)	68.23 $\pm$ 5.46	68.62 $\pm$ 5.51	0.288	0.773
Years of education (yr)	12.82 $\pm$ 3.37	12.88 $\pm$ 3.52	0.070	0.943
Tumor diameter (cm)	5.26 $\pm$ 0.98	5.21 $\pm$ 0.95	0.210	0.834
Clinical staging (FIGO) (phase)				
Ib	8 (24.24)	7 (21.21)	0.2989	0.8612
IIa	16 (48.48)	15 (45.45)		
IIb	9 (27.27)	11 (33.33)		
Degree of tumor differentiation				
Low differentiation	18 (54.55)	16 (48.48)	0.6510	0.7222
Moderate differentiation	12 (36.36)	15 (45.45)		
Highly differentiated	3 (9.09)	2 (6.06)		

FIGO: Federation International of Gynecology and Obstetrics.

**TABLE 2. Grading criteria of common side effects.**

Adverse reactions	Grading (degree)				
	0	I	II	III	IV
Diarrhea	None	Short-term (<2 days)	Tolerable (>2 days)	Intolerable, requires treatment	Hemorrhagic diarrhea
Hair	None	Mild hair loss	Moderate, patchy hair loss	Complete hair loss	Hair loss, failure to regrow
Nausea and vomiting	None	Nausea	Temporary vomiting	Persistent vomiting, requires treatment	Uncontrollable vomiting
Bone marrow suppression/leukocyte level ( $\times 10^9/L$ )	$\geq 4.0$	3.0~3.9	2.0~2.9	1.0~1.9	<1.0

### 3.1 Comparing clinical efficacy

Although the treatment efficacy of patients in the study group was 75.76%, numerically higher than the 66.67% observed in the control group, the difference was not statistically significant ( $\chi^2 = 0.665$ ,  $p = 0.414$ ) (Table 3).

### 3.2 Comparison of the incidence of adverse reactions (rephrase)

In the study group, the incidence of diarrhea was 45.45%, while the occurrences of nausea and vomiting were 48.48%, both of which were notably lower than the corresponding rates of 69.70% and 75.76% observed in the control group, and the differences were statistically different ( $\chi^2 = 3.969$ , 5.215,  $p = 0.046$ , 0.022). Furthermore, the incidence of bone marrow suppression in the study group was 48.48%, which was higher than the 21.21% incidence ( $\chi^2 = 4.405$ ,  $p = 0.020$ ). Hair loss was documented in both the study and control groups, with a significant difference noted between them. The

severity of adverse effects, encompassing diarrhea, alopecia, nausea and vomiting, was significantly lower in the study group compared to the control group ( $Z = 2.827$ , 2.807, 3.158, all  $p < 0.05$ ), and the severity of bone marrow suppression was significantly higher in the study group than in the control group ( $Z = 2.824$ , all  $p < 0.05$ ). The detailed results are shown in Tables 4.1, 4.2, 4.3 and 4.4.

### 3.3 Compare tumor marker levels

Before treatment, there was no statistically significant difference observed in the comparison of tumor marker levels between the two groups ( $p > 0.05$ ). However, after treatment, the tumor marker levels in the study group (CEA ( $2.34 \pm 1.05$ )  $\mu\text{g/L}$ , CA199 ( $32.62 \pm 3.54$ ) U/mL, CA125 ( $24.32 \pm 2.51$ ) U/mL, squamous cell carcinoma ( $1.26 \pm 0.32$ ) ng/mL) were found to be lower than the levels recorded in the control group, and these differences were statistically significant ( $p < 0.05$ ) (Table 5).

**TABLE 3. Comparison of treatment efficacy (n (%)).**

Group	Number of cases	Complete remission	Partial remission	Disease stabilization	Disease progression	Total effective
Study Group	33	20 (60.61)	5 (15.15)	8 (24.24)	25 (75.76)	25 (75.76)
Control Group	33	16 (48.48)	6 (18.18)	11 (33.33)	22 (66.67)	22 (66.67)
$\chi^2$	-	-	-	-	-	0.665
<i>p</i>	-	-	-	-	-	0.414

**TABLE 4.1. Comparison of the incidence of diarrhea between the two groups.**

Group	Number of cases	Level 0	Level I	Level II	Level III	Level IV
Study Group	33	18	9	5	1	0
Control Group	33	9	8	9	7	0
<i>Z</i>	—			2.827		
<i>p</i>	—			<0.001		

**TABLE 4.2. Comparison of hair loss between the two groups.**

Group	Number of cases	Level 0	Level I	Level II	Level III	Level IV
Study Group	33	0	15	10	8	0
Control Group	33	0	5	11	17	0
<i>Z</i>	—			2.807		
<i>p</i>	—			<0.001		

**TABLE 4.3. Comparison of the occurrence of nausea and vomiting between the two groups.**

Group	Number of cases	Level 0	Level I	Level II	Level III	Level IV
Study Group	33	17	9	5	2	0
Control Group	33	8	6	7	12	0
<i>Z</i>	—			3.158		
<i>p</i>	—			<0.001		

**TABLE 4.4. Comparison of bone marrow suppression between the two groups.**

Group	Number of cases	Level 0	Level I	Level II	Level III	Level IV
Study Group	33	26	3	3	1	0
Control Group	33	16	3	6	8	0
<i>Z</i>	—			2.824		
<i>p</i>	—			<0.001		

### 3.4 Comparison of WHOQOL-BREF scores

The WHOQOL-BREF scores within the study group, including assessments for the environment ( $16.33 \pm 1.18$ ), physiology ( $16.42 \pm 1.20$ ), social relationships ( $16.36 \pm 1.50$ ), and the overall total score ( $49.12 \pm 2.56$ ), were found to be significantly greater than those observed in the control group ( $p < 0.05$ ) (Table 6).

### 3.5 Comparison of KPS scores

Before treatment, our analysis revealed no significant difference in KPS scores between the two groups ( $p > 0.05$ ). However, at the follow-up assessments conducted at 7 days, 1 month, 2 months and 3 months post-treatment, the KPS scores within the study group ( $75.67 \pm 3.15$ ;  $79.55 \pm 3.52$ ;  $82.67 \pm 3.04$ ; and  $86.21 \pm 3.41$ , respectively) were found to be significantly higher compared to those of the control group ( $p < 0.05$ ) (Table 7).

**TABLE 5. Comparison of tumor marker levels ( $\bar{x} \pm s$ ).**

Groups	Number of cases	Pre-intervention	Post-intervention	<i>t</i>	<i>p</i>
CEA ( $\mu\text{g/L}$ )					
Study Group	33	5.16 $\pm$ 1.38	2.34 $\pm$ 1.05	9.342	<0.001
Control Group	33	5.23 $\pm$ 1.42	3.38 $\pm$ 1.12	5.876	<0.001
<i>t</i>	-	0.203	3.891		
<i>p</i>	-	0.839	0.002		
CA199 (U/mL)					
Study Group	33	118.56 $\pm$ 12.37	32.62 $\pm$ 3.54	38.369	<0.001
Control Group	33	118.12 $\pm$ 12.41	40.75 $\pm$ 4.13	33.982	<0.001
<i>t</i>	-	0.144	8.585		
<i>p</i>	-	0.885	<0.001		
CA125 (U/mL)					
Study Group	33	42.36 $\pm$ 3.14	24.32 $\pm$ 2.51	25.779	<0.001
Control Group	33	42.43 $\pm$ 3.16	30.46 $\pm$ 2.78	16.337	<0.001
<i>t</i>	-	0.090	9.417		
<i>p</i>	-	0.928	<0.001		
SCC (ng/mL)					
Study Group	33	4.62 $\pm$ 0.24	1.26 $\pm$ 0.32	48.254	<0.001
Control Group	33	4.61 $\pm$ 0.35	2.43 $\pm$ 0.41	23.231	<0.001
<i>t</i>	-	0.135	12.923		
<i>p</i>	-	0.893	<0.001		

CEA: carcino-embryonic antigen; CA: Carbohydrate antigen; SCC: squamous cell carcinoma.

**TABLE 6. Comparison of WHOQOL-BREF scores ( $\bar{x} \pm s$  (score)).**

Grouping	Number of cases	Pre-intervention	Post-intervention	<i>t</i>	<i>p</i>
Environment					
Study Group	33	10.33 $\pm$ 1.27	16.33 $\pm$ 1.08	20.675	<0.001
Control Group	33	10.62 $\pm$ 1.30	13.55 $\pm$ 1.58	8.226	<0.001
<i>t</i>	-	0.917	8.344		
<i>p</i>	-	0.363	<0.001		
Physiology					
Study Group	33	10.48 $\pm$ 1.12	16.42 $\pm$ 1.20	20.788	<0.001
Control Group	33	10.42 $\pm$ 1.15	13.24 $\pm$ 1.56	8.359	<0.001
<i>t</i>	-	0.215	9.282		
<i>p</i>	-	0.831	<0.001		
Social Relations					
Study Group	33	10.67 $\pm$ 1.27	16.36 $\pm$ 1.50	16.631	<0.001
Control Group	33	10.76 $\pm$ 1.30	13.12 $\pm$ 1.19	7.692	<0.001
<i>t</i>	-	0.285	9.721		
<i>p</i>	-	0.777	<0.001		
Total score					
Study Group	33	31.48 $\pm$ 1.77	49.12 $\pm$ 2.56	32.559	<0.001
Control Group	33	31.79 $\pm$ 2.27	39.91 $\pm$ 2.07	15.184	<0.001
<i>t</i>	-	0.619	16.071		
<i>p</i>	-	0.538	<0.001		

TABLE 7. Comparison of KPS scores ( $\bar{x} \pm s$  (points)).

Group	Number of cases	Pre-treatment	Treatment after 7 days	Treatment after 1 month	Treatment after 2 months	Treatment after 3 months
Study Group	33	65.21 $\pm$ 4.51	75.67 $\pm$ 3.15*	79.55 $\pm$ 3.52*	82.67 $\pm$ 3.04*	86.21 $\pm$ 3.41*
Control Group	33	65.36 $\pm$ 4.42	70.21 $\pm$ 3.01*	72.33 $\pm$ 3.14*	78.21 $\pm$ 3.11*	80.64 $\pm$ 3.12*
<i>t</i>	-	0.137	7.200	8.793	5.891	6.923
<i>p</i>	-	0.892	<0.001	<0.001	<0.001	<0.001

Note: Compared to pre-treatment \* $p < 0.05$ .

#### 4. Discussion

In the past, the clinical treatment of cervical cancer primarily revolved around surgical interventions and radiation therapy, with the choice of therapy predominantly determined by patients' tumor stage, such as whether they belonged to the pre-stage IIa or post-stage IIb [7, 8]. Clinical studies consistently reported low 5-year survival rates among cervical cancer patients, regardless of whether surgery or radiation therapy was utilized, suggesting a substantial need for improved efficacy in both surgical and radiation treatments [9, 10]. Previously, chemotherapy was seldom prescribed in the clinical treatment of cervical cancer patients due to the prevailing belief that cervical cancer lesions exhibited limited sensitivity to chemotherapeutic agents [11, 12]. However, with the introduction of neoadjuvant chemotherapy in cervical cancer treatment, researchers across multiple locations have begun to further assess this treatment approach by conducting in-depth analyses of specific neoadjuvant chemotherapy regimens used for cervical cancer treatment, including the optimal types and dosages of chemotherapeutic agents. At present, neoadjuvant chemotherapy regimens are recommended for patients with intermediate to advanced cervical cancer, as comparatively, the outcomes obtained by surgery alone are considered lower than multimodal treatment approaches [13]. The goals of neoadjuvant chemotherapy treatment for cervical cancer patients encompass four main objectives: complete eradication of subclinical lesions to reduce recurrence rates, effective reduction of tumor size to facilitate comprehensive lesion removal during subsequent surgery, reduction of cancer cell activity, and minimization of the risk of intraoperative or postoperative lesion metastasis [14].

Currently, platinum and paclitaxel represent the most commonly employed medications in clinical chemotherapy treatments, with the combination of paclitaxel and platinum standing out as the prevailing and still effective chemotherapy regimen [15]. According to the results of this study, it was observed that the treatment efficacy within the study group was 75.76%, surpassing the 66.67% noted in the control group ( $\chi^2 = 0.665$ ,  $p = 0.414$ ). Furthermore, following treatment, the tumor marker levels and WHOQOL-BREF scores in the study group were lower than those in the control group ( $p < 0.05$ ). Additionally, KPS scores in the study group were higher than those in the control group at 7 days, 1 month, 2 months and 3 months post-treatment ( $p < 0.05$ ). These findings suggest that the disparity in efficacy between the two chemotherapy regimens was marginal, while the paclitaxel

+ carboplatin combination exhibited superior effectiveness in enhancing treatment outcomes and quality of life. The mechanism of action of paclitaxel closely resembles that of a microtubule depolymerization stabilizer. It can directly bind to free microtubule proteins within the body, promoting the transformation of these proteins into stable microtubules under the influence and assistance of the drug, which in turn prevents the re-depolymerization of previously formed microtubules and ensures the formation of stable microtubule bundles with normal functionality. Consequently, this impedes mitosis and fosters the demise of tumor cells, yielding potent antitumor effects [16–18]. Relevant clinical studies have corroborated these findings, indicating that the preoperative utilization of paclitaxel in the treatment of cervical cancer patients can substantially reduce tumor size, facilitating the complete removal of tumor lesions during subsequent surgical procedures, subsequently diminishing the risk of recurrence and metastasis post-surgery [19, 20]. Paclitaxel, cisplatin and carboplatin have all demonstrated individual effectiveness in the treatment of cervical cancer. Related studies have further indicated that combination regimens can elevate therapeutic efficacy to levels exceeding 80% [21, 22]. Among the commonly utilized platinum drugs, cisplatin and carboplatin stand out, possessing a broad spectrum of anticancer activity [23, 24]. Cisplatin's principal advantages are its broad anticancer spectrum, high effectiveness, and others, whereas its main downside is its considerable toxic side effects. On the other hand, carboplatin, a second-generation platinum drug, has several advantages, including potent antitumor properties, high safety, minimal nephrotoxicity, and limited gastrointestinal adverse effects. These attributes make carboplatin a favorable choice in clinical chemotherapy applications [25]. Upon entering the body, carboplatin binds to tumor DNA and forms crosslinks, which disrupts the normal functioning of tumor DNA and results in the inability of the tumor to duplicate DNA correctly, ultimately leading to tumor cell death [26].

Our findings indicate that within the study group, the incidence of diarrhea was 45.45%, and the incidence of nausea and vomiting was 48.48%, which were significantly lower than those observed in the control group, where the incidence of diarrhea was 69.70%, and the incidence of nausea and vomiting was 75.76% ( $\chi^2 = 3.969$  and  $5.215$ ,  $p = 0.046$ ,  $0.022$ ). Conversely, the incidence of bone marrow suppression in the study group was 48.48%, which was significantly higher than the 21.21% incidence recorded in the control group ( $\chi^2 = 4.405$ ,  $p = 0.020$ ). However, the occurrence of hair loss was not significantly different in both groups. Furthermore, when

evaluating the severity of adverse effects, including diarrhea, alopecia, nausea and vomiting, it was observed that the study group exhibited significantly lower severity compared to the control group ( $Z = 2.827, 2.807, 3.158$  and  $2.824$ , all  $p < 0.05$ ). These findings suggest that the use of paclitaxel and carboplatin in cervical cancer patients could be associated with a lower risk of side effects, reduced toxicity and an overall enhanced safety profile, with the exception of bone marrow suppression.

The inclusion of patients in this study who had not previously undergone antitumor therapy may have contributed to the slightly more favorable results compared to related studies. Thus, it is conceivable that previous antitumor therapy could have induced local fibrosis, thereby impeding the direct targeting of local tumor cells by chemotherapeutic drugs and potentially diminishing the effectiveness of chemotherapy [27–30].

## 5. Conclusions

In conclusion, the treatment regimen of paclitaxel + carboplatin for cervical cancer patients has demonstrated effectiveness in reducing tumor marker levels, enhancing quality of life, and reducing the incidence of adverse effects. This regimen has several advantages compared to paclitaxel + cisplatin treatment and warrants consideration for clinical adoption. However, this study has several limitations, notably: (1) the sample size is relatively limited, necessitating further verification of the results for reliability, and (2) an extended follow-up period for patients is required to assess the long-term effects of the treatment method.

Therefore, in future research and clinical practice, it is recommended to conduct large-scale, multicenter, long-term follow-up studies. Additionally, the paclitaxel-cisplatin treatment approach should be explored in a broader range of research patients to yield more comprehensive and scientifically grounded conclusions, which could provide valuable guidance for improving the treatment of cervical cancer in clinical practice.

## AVAILABILITY OF DATA AND MATERIALS

The authors declare that all data supporting the findings of this study are available within the paper and any raw data can be obtained from the corresponding author upon request.

## AUTHOR CONTRIBUTIONS

SLH and WWZ—designed the study and carried it out; SLH, CJ, WWZ and PF—supervised the data collection, analyzed the data, interpreted the data, prepared the manuscript for publication and reviewed the draft of the manuscript. All authors have read and approved the manuscript.

## ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Ethical approval was obtained from the Ethics Committee of Huangshan City People's Hospital (Approval no. 2018082). Written informed consent was obtained from a legally authorized representative(s) for anonymized patient information to be published in this article.

## ACKNOWLEDGMENT

Not applicable.

## FUNDING

This research received no external funding.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

## REFERENCES

- [1] Liu Y, Zhao R, Qin X, Mao X, Li Q, Fang S. Cobimetinib sensitizes cervical cancer to paclitaxel *via* suppressing paclitaxel-induced ERK activation. *Pharmacology*. 2022; 107: 398–405.
- [2] Ilhan Y, Tatli AM, Teker F, Onder AH, Kose F, Geredeli C, *et al.* Cisplatin plus paclitaxel and bevacizumab versus carboplatin plus paclitaxel and bevacizumab for the first-line treatment of metastatic or recurrent cervical cancer. *International Journal of Gynecologic Cancer*. 2022; 32: 502–507.
- [3] Tanigawa T, Takeshima N, Ishikawa H, Nishio S, Usami T, Yamawaki T, *et al.* Paclitaxel-carboplatin and bevacizumab combination with maintenance bevacizumab therapy for metastatic, recurrent, and persistent uterine cervical cancer: an open-label multicenter phase II trial (JGOG1079). *Gynecologic Oncology*. 2022; 165: 413–419.
- [4] Ko CC, Yeh LR, Kuo YT, Chen JH. Imaging biomarkers for evaluating tumor response: RECIST and beyond. *Biomarker Research*. 2021; 9: 52.
- [5] Tang LL, Huang CL, Zhang N, Jiang W, Wu YS, Huang SH, *et al.* Elective upper-neck versus whole-neck irradiation of the uninvolved neck in patients with nasopharyngeal carcinoma: an open-label, non-inferiority, multicentre, randomised phase 3 trial. *The Lancet Oncology*. 2022; 23: 479–490.
- [6] Zhao Y, Lan Y, Chi Y, Yang B, Ren C. Downregulation of Circ-CEP128 enhances the paclitaxel sensitivity of cervical cancer through regulating miR-432-5p/MCL1. *Biochemical Genetics*. 2022; 60: 2346–2363.
- [7] Zhang J, Song C, Liu B. Efficacy of cisplatin plus paclitaxel as chemotherapy in patients with cervical cancer after laparoscopic nerve-sparing extensive hysterectomy and its effect on immune function. *Pakistan Journal of Pharmaceutical Sciences*. 2022; 35: 355–359.
- [8] Liu N, Zhang X, Chen J, Wang Q, Wu Q, Gao F, *et al.* Camrelizumab (SHR-1210) with carboplatin and albumin-binding paclitaxel in patients with metastatic or recurrent cervical cancer: an open-label, phase 2 trial. *Journal of Cancer Research and Therapeutics*. 2022; 18: 482–487.
- [9] Li J, Liu H, Li Y, Li J, Shen L, Long W, *et al.* Comparison of outcomes and side effects for neoadjuvant chemotherapy with weekly cisplatin and paclitaxel followed by chemoradiation *vs.* chemoradiation alone in stage IIB–IVA cervical cancer: study protocol for a randomized controlled trial. *Trials*. 2022; 23: 29.
- [10] Arakaki Y, Ariga T, Heianna J, Shimoji Y, Nakasone T, Taira Y, *et al.* Long-term outcomes of cervical adenocarcinoma treated with concurrent chemoradiotherapy using paclitaxel and cisplatin. *In Vivo*. 2020; 34: 2739–2743.
- [11] Yasunaga M, Yahata H, Okugawa K, Shimokawa M, Maeda Y, Hori E, *et al.* Prognostic impact of adding bevacizumab to carboplatin and paclitaxel

- for recurrent, persistent, or metastatic cervical cancer. *Taiwanese Journal of Obstetrics and Gynecology*. 2022; 61: 818–822.
- [12] DI Martino G, Lissoni AA, Ferrari D, DI Meo ML, Cosio S, Gadducci A, *et al.* Dose-dense neoadjuvant chemotherapy with paclitaxel and carboplatin in cervical cancer: efficacy on pathological response. *Anticancer Research*. 2021; 41: 497–502.
- [13] Ishikawa M, Shibata T, Iwata T, Nishio S, Takada T, Suzuki S, *et al.* A randomized phase II/III trial of conventional paclitaxel and carboplatin with or without bevacizumab versus dose-dense paclitaxel and carboplatin with or without bevacizumab, in stage IVB, recurrent, or persistent cervical carcinoma (JCOG1311): primary analysis. *Gynecologic Oncology*. 2021; 162: 292–298.
- [14] Li J, Li Y, Wang H, Shen L, Wang Q, Shao S, *et al.* Neoadjuvant chemotherapy with weekly cisplatin and paclitaxel followed by chemoradiation for locally advanced cervical cancer. *BMC Cancer*. 2023; 23: 51.
- [15] Nagao S, Yamamoto K, Oishi T, Yamaguchi S, Takehara K, Shimada M, *et al.* Phase II study of a new multidisciplinary therapy using once every 3 week carboplatin plus dose-dense weekly paclitaxel before and after radical hysterectomy for locally advanced cervical cancer. *International Journal of Clinical Oncology*. 2021; 26: 207–215.
- [16] Redondo A, Colombo N, McCormack M, Dreosti L, Nogueira-Rodrigues A, Scambia G, *et al.* Primary results from CECILIA, a global single-arm phase II study evaluating bevacizumab, carboplatin and paclitaxel for advanced cervical cancer. *Gynecologic Oncology*. 2020; 159: 142–149.
- [17] Tewari KS, Sill MW, Birrer MJ, Penson RT, Huang H, Moore DH, *et al.* Final survival analysis of topotecan and paclitaxel for first-line treatment of advanced cervical cancer: an NRG oncology randomized study. *Gynecologic Oncology*. 2023; 171: 141–150.
- [18] Jianfang X, Ling Z, Yanan J, Yanliang G. The effect of Chaihu-shugansan on cytotoxicity induction and PDGF gene expression in cervical cancer cell line HeLa in the presence of paclitaxel +cisplatin. *Cellular and Molecular Biology*. 2021; 67: 143–147.
- [19] Dong M, Li P, Xie Y, Wang Z, Wang R. CircMYBL2 regulates the resistance of cervical cancer cells to paclitaxel *via* miR-665-dependent regulation of EGFR. *Drug Development Research*. 2021; 82: 1193–1205.
- [20] Maene C, Salihi RR, Van Nieuwenhuysen E, Han SN, Concin N, Vergote I. Combination of weekly paclitaxel-carboplatin plus standard bevacizumab as neoadjuvant treatment in stage IB–IIB cervical cancer. *International Journal of Gynecologic Cancer*. 2021; 31: 824–828.
- [21] Pang K, Lee J, Kim J, Park J, Park Y, Hong E, *et al.* Degradation of DRAK1 by CUL3/SPOP E3 Ubiquitin ligase promotes tumor growth of paclitaxel-resistant cervical cancer cells. *Cell Death & Disease*. 2022; 13: 169.
- [22] Sun T, Zhang D, Wang Z, Zhao B, Li Y, Sun X, *et al.* Inhibition of the notch signaling pathway overcomes resistance of cervical cancer cells to paclitaxel through retardation of the epithelial-mesenchymal transition process. *Environmental Toxicology*. 2021; 36: 1758–1764.
- [23] Jiang P, Deng X, Qu A, Jiang W, Guo F, Han Q, *et al.* Image guidance volume-modulated arc radiation therapy concurrently with nab-paclitaxel plus cisplatin for patients with locally advanced cervical cancer: a single-arm dose escalation trial. *International Journal of Radiation Oncology, Biology, Physics*. 2023; 115: 1197–1204.
- [24] Li B, Xia X, Chen J, Xia D, Xu R, Zou X, *et al.* Paclitaxel-loaded lignin particle encapsulated into electrospun PVA/PVP composite nanofiber for effective cervical cancer cell inhibition. *Nanotechnology*. 2021; 32: 015101.
- [25] Chu G, Liu X, Yu W, Chen M, Dong L. Cisplatin plus paclitaxel chemotherapy with or without bevacizumab in postmenopausal women with previously untreated advanced cervical cancer: a retrospective study. *BMC Cancer*. 2021; 21: 133.
- [26] Tian XP, Qian D, He LR, Huang H, Mai SJ, Li CP, *et al.* Corrigendum to “The telomere/telomerase binding factor PinX1 regulates paclitaxel sensitivity depending on spindle assembly checkpoint in human cervical squamous cell carcinomas” [*Canc. Lett.* 353 (2014) 104–114]. *Cancer Letters*. 2021; 2021; 519: 345.
- [27] Higami S, Tanaka Y, Deguchi T, Shiraishi M, Shiki Y. Acute ST-segment elevations following paclitaxel administration for uterine cervical cancer: a case report and literature review. *Cardio-Oncology*. 2022; 8: 22.
- [28] Ma XH, Guo K, Dong WL, Jiang J. Efficacy and safety of paclitaxel liposomes compared to paclitaxel combined with platinum in concurrent chemoradiotherapy for unresectable cervical carcinoma: a meta-analysis. *Chinese journal of oncology*. 2021; 43: 1132–1139. (In Chinese)
- [29] Li XJ, You Y, Zhang QL, Zhang BB, Yan L, Ou ZM, *et al.* Preparation of paclitaxel-loaded and folic acid-modified poly (lactic-co-glycolic acid) nano-micelles and *in vitro* anticancer effect on cervical cancer HeLa cells. *China Journal of Chinese Materia Medica*. 2021; 46: 2481–2488. (In Chinese)
- [30] Nishi H, Ono M, Ohno S, Yamanaka Z, Sasaki T, Ohyashiki K, *et al.* Hypoxia-induced paclitaxel resistance in cervical cancer modulated by miR-100 targeting of USP15. *Gynecologic Oncology Reports*. 2023; 45: 101138.

**How to cite this article:** Shanliang Hu, Chao Jiang, Weiwei Zhang, Ping Fang. Comparison of the clinical efficacy of paclitaxel + carboplatin and paclitaxel + cisplatin on tumor markers and WHOQOL-BREF score on cervical cancer patients. *European Journal of Gynaecological Oncology*. 2023; 44(6): 142-149. doi: 10.22514/ejgo.2023.109.