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Clinical effect of cyclophosphamide-taxinol therapy on breast cancer.

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Abstract

Objective: This paper aims to discuss the clinical effect of cyclophosphamide-taxinol therapy in breast cancer.

Method: A total of 68 patients with breast cancer in our hospital from April 2013 to April 2016 were selected. Patients were randomly divided into observation and control groups. Each group consisted of 34 cases. The observation group was treated with cyclophosphamide-taxinol therapy. The control group was treated with taxinol-azithromycin therapy.

Results: Total therapeutic efficiencies of observation and control groups reached 38.23% and 7.65%, respectively, and showed a significant difference (P<0.05). The two groups also displayed a small difference prior to treatment (P>0.05). After treatment, vascular endothelial growth factor A (VEGFA) (82.51 ± 11.34) and VEGFB (77.95 ± 6.24) of observation group were significantly superior to those of the control group (P<0.05). CD4 (27.45 ± 5.48), CD8 (35.97 ± 7.16), CH4/CD8 (0.69 ± 0.18), and the number of natural killer cells (20.79 ± 2.49) of the observation group were significantly higher than those of the control group [(38.45 ± 5.98), (20.45 ± 6.41), (1.31 ± 0.48), and (39.44 ± 3.49), respectively]. The two groups showed statistically significant differences after treatment (P<0.05).

Conclusion: Cyclophosphamide-taxinol therapy can effectively reduce angiogenesis of patients with breast cancer and exert relatively ideal clinical effects. Therefore, this therapy should be further promoted in clinics.

Keywords: Breast cancer, Cyclophosphamide, Taxinol.

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Introduction

Breast cancer is a common malignant tumor. Currently, this tumor is widely accepted as a key public health problem, and it threatens the physical and mental health of women [1,2]. With continuous development in the medical industry, many efficient drugs for malignant tumors, including breast cancer, have been developed [3]. In the present experiment, 68 patients with breast cancer were selected.

Data and Methods

General data

A total of 68 patients with breast cancer in People's Hospital of Rizhao from April 2013 to April 2016 were selected and were randomly divided into observation and control groups. Each group consisted of 34 cases. The observation group was treated with cyclophosphamide-taxinol therapy. The control group was treated with taxinol-azithromycin therapy. Patients in the observation group aged from 32 to 68, with an average of 48.51 ± 2.13. Control group patients aged from 31 to 69, with an average of 48.62 ± 2.08.

Method

The control group was treated with taxinol-azithromycin therapy. Azithromycin (75 mg/m^2) and taxinol (150 mg/m^2) were mixed for intravenous dripping. Two courses of treatment were administered, and each course was performed for 3 weeks.

The observation group was treated with cyclophosphamide-taxinol therapy. Taxinol (75 mg/m^2) and 500 ml of 5% dextrose injection were mixed for intravenous dripping for 3 h. Afterward, cyclophosphamide (500 mg/m^2) and 100 ml of normal saline were mixed for intravenous injection. Considerable attention was given to physical conditions of patients during the medication period. Any abnormal was treated promptly to protect therapeutic effect.

Observation indexes and evaluation standards of clinical effect

The angiogenesis indexes (Vascular Endothelial Growth Factor A (VEGFA) and VEGFB) and clinical effects of all patients were observed. Related data were compared. Evaluation standards of clinical effect were as follows: (1) Complete

remission: pathological tissues disappear completely, and no new pathological tissues are produced after treatment. Marks of breast cancer are examined normal; (2) Partial remission: diameter of pathological tissues is reduced by 30% after treatment, and clinical effect can remain for 28 days; (3) Stabilization: lesion shows no growth to progress standard after treatment, and pathological tissues decrease to some extent; (4) Progression: diameter of pathological tissues increases by 20% or more compared with that before treatment.

Statistical analysis

Experimental data were analyzed by special statistical software (SPSS23.0). Measurement data were evaluated by using t-test and are expressed as ± mean. Enumeration data were examined by χ^2-test and are expressed in percentage. P<0.05 indicated statistically significant difference.

Results

Comparison of clinical effect

According to statistical analysis, total therapeutic efficiency of the observation group totalled 38.23%, which is significantly higher than that of the control group (7.65%) (P<0.05) (Table 1).

Table 1. *Comparison of clinical effect (n (%)).*

Groups	n	Complete remission	Partial remission	Stabilization	Progression	Total therapeutic efficiency
Observation group	34	4 (11.76%)	9 (26.47%)	16 (47.06%)	5 (14.71%)	38.23%
Control group	34	1 (2.94%)	5 (14.71%)	17 (50.00%)	11 (32.35%)	17.65%
χ2	/					16.36
P	/					P<0.05

Comparison of angiogenesis indexes

The two groups showed no statistically significant difference before treatment (P>0.05). After treatment, the observation group achieved significantly better VEGFA (82.51 ± 11.34) and VEGFB (77.95 ± 6.24) than those of the control group (P<0.05). Details are shown in Table 2.

Table 2. *Comparison of angiogenesis indexes.*

Groups	n	VEGFA		VEGFB	
		Before	After	Before	After
Observation group	34	214.62 ± 25.69	82.51 ± 11.34	184.67 ± 12.64	77.95 ± 6.24
Control group	34	213.58 ± 24.08	136.94 ± 11.65	183.08 ± 11.89	122.31 ± 7.05
t	/	0.329	13.527	0.418	11.529
P	/	P>0.05	P<0.05	P>0.05	P<0.05

Comparison of immunologic functions after treatment

After treatment, CD4 (27.45 ± 5.48), CD8 (35.97 ± 7.16), CH4/CD8 (0.69 ± 0.18), and the number of Natural Killer (NK) cells (20.79 ± 2.49) of the observation group were remarkably higher than those of the control group ((38.45 ± 5.98), (20.45 ± 6.41), (1.31 ± 0.48), and (39.44 ± 3.49), respectively) (P<0.05) (Table 3).

Table 3. *Comparison of immunologic functions after treatment.*

Groups	CD4	CD8	CD4/CD8	NK
Control group	27.45 ± 5.48	35.97 ± 7.16	0.69 ± 0.18	20.79 ± 2.49
Observation group	38.45 ± 5.98	20.45 ± 6.41	1.31 ± 0.48	39.44 ± 3.49
t	11.507	13.703	10.262	36.912
P	<0.05	<0.05	<0.05	<0.05

Discussion

Breast cancer is one of the most common malignant tumors. According to statistics, morbidity of breast cancer accounts for 7%-10% of malignant tumors, which is second to metrocarcinoma in women [4]. Breast cancer morbidity is often related with genetics. Women aged 40-60 before and after menopause are the most vulnerable group of breast cancer. Only 1%-2% patients with breast cancer are males. Breast cancer is a kind of malignant tumor on epithelial tissues of breasts [5,6]. This tumor is also one of the most malignant tumors that affect physical and mental health and threaten lives of women, but it is rare in males. Pathogenesis of breast cancer remains incompletely discovered. Breast cancer morbidity follows a certain trend. Females who are exposed to high-risk factors are easily attacked with breast cancer [7]. High-risk factors refer to various risk factors related to morbidity of breast cancer. Risk factors possessed by most patients with breast cancer are called high-risk factors of breast cancer. Early discovery and diagnosis of breast cancer are keys to improvement of clinical effects [8].

Surrounding invasion and distant metastasis of breast cancer mainly depend on neoangiogenesis. Therefore, tumor growth shall be controlled, and angiogenesis of tumor shall be reduced during treatment [9]. Vascular endothelial cell factors play an important role during tumor cell growth. Taxinol is a new anti-microtubular drug, which when applied to breast cancer, can promote microtubulin polymerization, inhibit depolymerization, and enhance stability of microtubulin [10]. Consequently, cell divisions are inhibited. Combination of taxinol and cyclophosphamide can reduce angiogenesis and inhibit focus metastasis. Cyclophosphamide is a common antineoplastic drug in clinics; its drug mechanism includes decomposition of chloroethyl (phosphoramide) with strong alkanization under catalysis effect of hepatic microsomal enzyme, which poisons tumor cells. Cyclophosphamide also exhibits an immunosuppressive effect. Research results

showed that total therapeutic efficiency of the observation group totalled 38.23%, which is significantly higher than that of the control group (7.65%) ($P<0.05$). The two groups showed no statistically significant difference before treatment ($P>0.05$). After treatment, VEGFA and VEGFB of the observation group were significantly better than those of the control group ($P<0.05$). CD4, CD8, CH4/CD, and the number of NK cells of the observation group were significantly higher than those of the control group ($P<0.05$). These results demonstrated that cyclophosphamide-taxinol therapy exerts ideal clinical effect to breast cancer.

Conclusion

Cyclophosphamide-taxinol therapy can reduce angiogenesis effectively. This therapy also exerts ideal clinical effect to breast cancer and it should be further promoted in clinical settings.

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