ORIGINAL ARTICLE

Serum concentrations of receptor for interleukin 8 in patients with esophageal cancer

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KEY WORDS

chemokines, esophageal, receptor, tumor

ABSTRACT

INTRODUCTION A specific receptor for interleukin 8, known as C-X-C chemokine type-2 receptor (CXCR-2), is one of the 7-transmembrane G-protein-coupled receptors. Its involvement in the development of numerous malignancies, including esophageal cancer (EC), has been suggested.

OBJECTIVES The aim of this study was to assess the diagnostic and prognostic usefulness of serum CXCR-2 level measurement in patients with EC, in comparison with C-reactive protein (CRP) levels and classic tumor markers such as carcinoembryonic antigen (CEA) and squamous cell carcinoma antigen (SCC-Ag).

PATIENTS AND METHODS The study included 72 individuals: 42 patients with EC and 30 healthy volunteers. Serum CXCR-2 concentrations were measured by an immunoenzymatic assay. The levels of classic tumor markers were measured using the chemiluminescent method, and CRP levels were measured using the immunoturbidimetric method.

RESULTS Serum CXCR-2 concentrations were significantly higher in patients with EC than in the control group, similarly to CEA and CRP levels. Moreover, CXCR-2 concentrations were significantly higher in patients with poorly differentiated EC (G3) compared with those with G2 tumors. The diagnostic sensitivity and accuracy, as well as the negative predictive value of the serum CXCR-2 assay were higher than those observed for classic tumor markers and slightly lower than those observed for CRP levels. The highest diagnostic sensitivity was found for the combined analysis of CXCR-2 and CRP.

CONCLUSIONS Our results suggest the role of CXCR-2 in the development of EC. Thus, further research is needed to clarify the significance of chemokines and their receptors as potential tumor markers of EC.

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INTRODUCTION Chemokines belong to a family of small molecular chemotactic proteins, which play an important role in physiological and pathological processes, such as inflammation, wound healing, and angiogenesis.[1-3] It has been suggested that these cytokines may be produced by tumor cells and are able to facilitate communication between cancer cells and nonneoplastic cells within the tumor microenvironment. Therefore, some authors indicated that chemokines promote the development, invasion, and metastasis of various malignancies, including esophageal cancer (EC).[3-5]

EC is the eighth most common neoplastic disease characterized by rapid progression and unfavorable prognosis.[1,6] The routine procedures for EC diagnosis are histopathological assessment of tissue samples and imaging techniques. However, endoscopic ultrasonography or computed tomography (CT) is of limited significance in early EC detection. Thus, new diagnostic biomarkers are urgently needed. At present, biochemical markers such as carcinoembryonic antigen (CEA) and squamous cell carcinoma antigen (SCC-Ag) are used in the routine diagnosis and follow-up of patients with EC.[7] However, the diagnostic

TABLE 1 Characteristics of patients with esophageal cancer

Variable			Number of patients
group	esophageal cancer		42
sex	male		35
	female		7
type of cancer	AC		17
	ESCC		25
TNM stage	AC	IIa + IIb	3
		III	13
		IV	1
	ESCC	IIa + IIb	7
		III	13
		IV	5
depth of tumor invasion	AC	T2	3
		T3	13
		T4	1
	ESCC	T2	4
		T3	11
		T4	10
nodal involvement	AC	N0	3
		N1	14
	ESCC	N0	8
		N1	17
distant metastases	AC	M0	16
		M1	1
	ESCC	M0	20
		M1	5
differentiation of the tumor	AC	G1	3
		G2	8
		G3	6
	ESCC	G1	7
		G2	8
		G3	9
		unknown	1
survival of patients	AC	alive	12
		died	5
	ESCC	alive	7
		died	18

Abbreviations: AC, adenocarcinoma of the esophagus; ESCC, esophageal squamous cell carcinoma; G, differentiation of the tumor (G1, well differentiated; G2, moderately differentiated; G3, undifferentiated); TNM, tumor–nodulus–metastasis (T, depth of tumor invasion; N, nodal involvement; M, distant metastases)

sensitivity and specificity of these markers are unsatisfactory. Therefore, it is crucial to establish other biochemical markers of EC for the improved diagnosis and prognosis of EC patients.

The chemokine family is divided into 4 groups, based on the positions of key cysteine residues (CXC, CX3C, CC, and C). The 2 N-terminal cysteines of C-X-C chemokines are separated by 1 amino acid (X). The C-X-C chemokine 8 (CXCL-8), known as interleukin (IL) 8, as well as its specific receptor, C-X-C chemokines type 2 receptor (CXCR-2), may facilitate tumor progression via the regulation of angiogenesis, migration of tumor cell, and modification of immune responses. The CXCR-2 is a 7-transmembrane G-protein--coupled cell surface chemokine receptor, which occurs on lymphocytes and neutrophils.[1,3] The CXCL-8/CXCR-2 axis plays an important role in inflammatory processes such as lymphocyte homing and infection.[8] However, some authors suggested that the CXCL-8/CXCR-2 signaling system may facilitate tumor progression.[1,8] As a promoter of tumor angiogenesis, CXCL-8 binds with CXCR-2, which is able to regulate the response of endothelial cells to CXCL-8.[2] The study of Liang et al[9] indicated that CXCR-2 was significantly overexpressed in EC cells compared with normal esophageal tissue. Moreover, there were significant associations between CXCR-2 expression and tumor stage (tumor–nodulus–metastasis [TNM])[9] and nodal involvement.[1] Additionally, CXCR-2 expression was proved to be an independent predictor for the survival of EC patients.[1]

All the above results were obtained using the immunohistochemical method. To our knowledge, the present study is the first to indicate serum concentrations of the specific receptor CXCR-2 in patients with 2 histological types of EC: adenocarcinoma of the esophagus (AC) and esophageal squamous cell carcinoma (ESCC). The aim of our study was to assess the clinical usefulness of serum CXCR-2 concentrations in the diagnosis and prognosis of EC patients, compared with the levels of C-reactive protein (CRP) and classic tumor markers (CEA and SCC-Ag). The associations between CXCR-2 levels and the clinical and pathological parameters of the tumor as well as survival of the patients were also investigated in our study. In addition, the diagnostic characteristics including diagnostic sensitivity and specificity, accuracy, negative predictive value (NPV) and positive predictive value (PPV), as well as the areas under the receiver operating characteristic (ROC) curve (AUC) for CXCR-2 in comparison with the other proteins (CRP, SCC-Ag, and CEA) were calculated.

PATIENTS AND METHODS

The main study group comprised 42 patients with EC (age, 44–80 years), including 25 patients with ESCC and 17 patients with AC. The control group included 30 healthy volunteers (19 women and 11 men; age, 22–72 years). Patients with EC were diagnosed in the Department of Thoracic Surgery of the Białystok University Hospital, Białystok, Poland. The clinical diagnosis of EC was based on a microscopic examination of tissue samples. In the first step of the analysis, routine hematoxylin and eosin staining was used. The additional immunohistochemical techniques such as staining for cytokeratin 7 and 20 for AC and staining for high-molecular--weight cytokeratin for ESCC were performed for the differentiation between AC and ESCC, respectively. Based on the microscopic examination, tissue samples derived from patients were differentiated into 2 histological types of EC: AC and ESCC. EC was staged for all patients based on the TNM

TABLE 2 Serum levels of proteins in the study groups

Study group		CXCR-2, ng/ml	CRP, mg/l	CEA, ng/ml	SCC-Ag, ng/ml
control group (n = 30)	median	0.58	0.85	1.20	1.00
	range	0.06–1.08	0.20–4.10	0.50–4.54	0.30–2.50
EC (n = 42)	median	0.73	7.35	1.98	1.25
	range	0.12–1.88	0.20–152.50	0.50–65.06	0.50–36.00
	P (EC vs control group)	0.009	0.000	0.007	0.064
AC (n = 17)	median	0.72	1.90	1.96	1.00
	range	0.12–1.88	0.20–46.80	0.50–44.40	0.50–2.20
	P (AC vs control group)	0.221	0.140	0.055	0.924
ESCC (n = 25)	median	0.74	16.20	2.00	1.60
	range	0.33–1.65	0.20–152.50	0.50–65.06	0.50–36.00
	P (ESCC vs control group)	0.026	0.000	0.066	0.007
	P (AC vs ESCC)	0.682	0.026	0.980	0.008

The differences between the groups were significant at a *P* value of less than 0.05.

Abbreviations: CEA, carcinoembryonic antigen; CRP, C-reactive protein; CXCR-2, specific C-X-C motif chemokine receptor-2; EC, esophageal cancer; SCC-Ag, squamous cell carcinoma antigen; others, see TABLE 1

classification presented by the International Union Against Cancer.[10] In addition, all patients with EC were divided into groups depending on the stage of the tumor (TNM), depth of tumor invasion (T factor), the presence of lymph nodes (N factor), and distant metastases (M factor) as well as histological grade (G factor) of the tumor. The characteristics of the study group are presented in TABLE 1. All patients gave informed consent and the study was approved by the Local Ethics Committee (R-I-002/42/2015) of the Medical University of Bialystok, Białystok, Poland.

Blood samples from patients with EC were obtained prior to the start of treatment between the years 2006 and 2010 and stored at –80°C until assayed. The CXCR-2 concentrations were measured in serum using enzyme-linked immunosorbent assay kits (EIAab, Wulhan, China) in accordance with the manufacturer's instructions. The intra-assay coefficient of variation (CV) for CXCR-2 was indicated by the manufacturer as 7.9% or lower. The serum levels of classic tumor markers (CEA and SCC-Ag) were measured with a chemiluminescent microparticle immunoassay (Abbott Laboratories, Abbott Park, Illinois, United States) using the ARCHITECT 8200 ci analyzer (Abbott Laboratories). The CV for CEA was established by the manufacturer as 4.9% at a mean concentration of 2.2 ng/ml (SD, 0.11 ng/ml), whereas the intra-assay SCC-Ag CV was set by the manufacturer as 4.3% at a mean concentration of 1.97 ng/ml (SD, 0.085). CRP levels were measured in serum using the immunoturbidimetric method (Abbott) in accordance with the manufacturer's instruction.

Statistical analysis Serum concentrations of CXCR-2, CEA, SCC-Ag, and CRP did not follow a normal distribution in a preliminary statistical analysis (χ^2 test). Therefore, the nonparametric statistical analyses were used. The Mann–Whitney test was employed to compare 2 groups, while the Kruskal–Wallis test was used for the analysis of 3 or more groups. If significant differences were shown, the post hoc Dwass–Steele–Critchlow–Fligner test was used. In addition, diagnostic sensitivity and specificity, accuracy, as well as NPV and PPV for serum CXCR-2, CEA, SCC-Ag, and CRP levels were determined. The results were presented as median and range (minimum and maximum). The differences were considered to be statically significant when a *P* value was less than 0.05. For statistical analysis, STATISTICA 5.1 PL (StatSoft Inc., Tulsa, Oklahoma, United States) was used, while the MedCalc statistical software (Acacialaan, Ostend, Belgium) and Microsoft Office Excel (Microsoft, Redmond, Washington, United States) were used to assess the diagnostic characteristics. Moreover, the Kaplan–Meier test was used for the analysis of survival curves. For the univariate analyses of survival, the log-rank test was performed, while the Cox proportional hazards model was used for multivariate analyses.

RESULTS CXCR-2 concentrations, similarly to CEA and CRP levels, were significantly higher in EC patients in comparison with the control group (TABLE 2). Moreover, serum CXCR-2 concentrations were significantly higher in patients with ESCC than in the control group. The same was observed for CRP and SCC-Ag levels.

Regarding the serum levels of the proteins in relation to tumor stage and the clinical and pathological characteristics of malignancy, the highest CXCR-2 levels were found in stage II of EC, while the concentrations of SCC-Ag and CRP were higher in stage IV of the tumor compared with early EC. A significant difference between the TNM stages was found for CRP levels ($P = 0.002$) in the Kruskal–Wallis test. Furthermore, serum CRP concentrations were significantly higher in stage

TABLE 3 Serum concentrations of proteins in relation to clinical and pathological parameters of esophageal cancer[10]

Parameter			CXCR-2, ng/ml	CRP, mg/l	CEA, ng/ml	SCC-Ag, ng/ml
TNM stage	II	median	0.89	5.30	1.80	1.05
		range	0.45–1.56	0.90–67.90	0.50–2.62	0.80–4.50
	III	median	0.69	5.40	2.24	1.30
		range	0.12–1.88	0.20–36.80	0.99–65.06	0.50–5.40
	IV	median	0.85	48.90[a,b]	1.85	6.50
		range	0.33–1.65	28.50–152.50	0.50–7.69	0.70–36.00
	P (Kruskal–Wallis test)		0.132	0.002	0.081	0.443
depth of tumor invasion	T2	median	0.92	1.90	1.83	0.90
		range	0.45–1.56	0.90–46.80	0.50–2.15	0.80–2.60
	T3	median	0.71	4.60	2.07	1.30
		range	0.12–1.88	0.20–84.50	0.99–65.06	0.50–36.00
	T4	median	0.72	30.50	2.45	1.60
		range	0.33–1.20	0.40–152.50	0.50–18.47	0.50–20.60
	P (Kruskal–Wallis test)		0.239	0.058	0.185	0.261
nodal involvement	N0	median	0.87	2.40	1.83	1.10
		range	0.45–1.56	0.40–67.90	0.50–65.06	0.80–4.50
	N1	median	0.72	10.20	2.16	1.30
		range	0.12–1.88	0.20–152.50	0.50–44.40	0.50–36.00
	P (Mann–Whitney test)		0.241	0.596	0.193	0.483
distant metastases	M0	median	0.72	5.40	1.98	1.20
		range	0.12–1.88	0.20–67.90	0.50–65.06	0.50–5.40
	M1	median	0.85	48.90	1.85	6.50
		range	0.33–1.65	28.50–152.50	0.50–7.69	0.70–36.00
	P (Mann–Whitney test)		0.719	0.001	0.440	0.235
differentiation of tumor	G1	median	0.89	5.05	1.85	1.15
		range	0.12–1.88	0.20–53.20	0.50–65.06	0.80–2.60
	G2	median	0.59	6.10	2.28	1.40
		range	0.19–0.92	0.50–152.50	0.50–44.40	0.50–20.60
	G3	median	0.86[c]	23.50	2.00	1.20
		range	0.33–1.65	0.20–84.50	0.63–18.47	0.50–36.00
	P (Kruskal–Wallis test)		0.040	0.713	0.370	0.665

a significant in post hoc Dwass–Steel–Critchlow–Fligner test in comparison with stage II ($P = 0.034$)
b significant in post hoc Dwass–Steel–Critchlow–Fligner test in comparison with stage III ($P = 0.001$)
c significant in post hoc Dwass–Steel–Critchlow–Fligner test in comparison with G2 ($P = 0.034$)

Abbreviations: see **TABLES 1** and **2**

IV of the tumor when compared with stages II ($P = 0.034$) and III ($P = 0.001$). There was a significant difference between serum CRP levels in patients with the presence of distant metastasis (M1 subgroup) and the M0 subgroup ($P = 0.001$). As far as the differentiation of EC is concerned, we revealed that the serum concentrations of CXCR-2 were significantly higher in patients with poorly differentiated EC (G3) compared with those with G2 tumors ($P = 0.034$). Similar observations were made for CRP levels, although these differences were not significant (**TABLE 3**).

The relationships between the survival of EC patients and serum levels of CXCR-2, classic tumor markers (CEA and SCC-Ag), as well as CRP were calculated using the Kaplan–Meier method. The univariate log-rank analysis indicated that the histological type of EC ($P = 0.017$), tumor stage ($P = 0.001$), depth of tumor invasion ($P = 0.000$), and the presence of distant metastases ($P = 0.000$), as well as CRP ($P = 0.000$) and SCC-Ag ($P = 0.006$) concentrations were the factors significantly affecting the overall survival. A multivariate regression analysis with the Cox proportional hazards model revealed that none of the proteins were independent prognostic factors for the survival of EC patients.

The diagnostic sensitivity and specificity, accuracy, NPV and PPV, as well as the AUC for all the proteins tested were assessed to indicate the diagnostic usefulness of these biochemical markers in patients with EC. The percentages of elevated concentrations (diagnostic sensitivity) of CXCR-2, CEA, SCC-Ag, and CRP are presented in **TABLE 4**. The diagnostic sensitivity of serum CXCR-2 levels (57%) in EC patients was

TABLE 4 Diagnostic characteristics of CXCR-2, classic tumor markers, and C-reactive protein levels in patients with esophageal cancer

Parameter	Sensitivity	Specificity	PPV	NPV	ACC
CXCR-2	57	73	75	55	63
CRP	60	100	100	64	76
CEA	17	93	78	44	49
SCC-Ag	23	97	91	48	54
CXCR-2 + CRP	83	73	81	76	65
CXCR-2 + CEA	64	70	75	58	67
CXCR-2 + SCC-Ag	66	70	76	60	68
CRP + CEA	60	93	93	62	74
CRP + SCC-Ag	67	97	97	67	79
CEA + SCC-Ag	36	90	83	50	58

Abbreviations: ACC, accuracy; NPV, negative predictive value; PPV, positive predictive value; others, see TABLES 1 and 2

markedly higher than that of classic tumor markers (CEA, 17%; SCC-Ag, 23%), while being marginally lower than that of CRP (60%). The highest diagnostic sensitivity was found for the combined analysis of CXCR-2 with CRP (83%)—it was markedly higher than for the combination of the classic tumor markers (CEA with SCC-Ag, 36%) (TABLE 4). The diagnostic specificity of CXCR-2 concentrations was lower than that for the other proteins, similarly to the PPV. The NPV for CXCR-2 levels was higher than that for the classic tumor markers and slightly lower than that for CRP, similarly to accuracy (TABLE 4). In addition, the AUC of CXCR-2 in patients with EC (0.6810, $P = 0.0047$) was higher than that for SCC-Ag (0.6286, $P = 0.0489$) and marginally lower than that for CRP (0.7762, $P = 0.000$) and CEA (0.6873, $P = 0.0046$) (FIGURE 1). The cut-off value of CXCR-2 levels was estimated using the Youden index as 0.72 ng/ml, while for the classic tumor markers (SCC-Ag, 2 ng/ml; CEA, 4 ng/ml) and CRP (5.75 mg/l), the cut-off values were previously established in our department based on the 95th percentile.[11-13]

DISCUSSION Chemokines and their specific receptors play an important role in tumor growth, angiogenesis, invasion, and metastasis of several malignancies, including EC. The CXCR2 is a member of the G-protein-coupled receptor family. An increased expression of CXCR-2 in EC tissue has been reported by numerous authors.[1,9,13,14] To our knowledge, no studies comparing serum concentrations of CXCR-2 with the levels of the inflammatory protein (CRP) and classic tumor markers (CEA and SCC-Ag) in EC patients have been conducted to date. The present study is a continuation of our previous studies concerning the role of selected proteins such as chemokine CXCL-12 and its specific receptor CXCR-4, CRP, IL-6, hematopoietic cytokines, and matrix metalloproteinases as candidates for tumor markers of EC.[11-13,15,16]

The current study demonstrated that CXCR-2 concentrations were significantly higher in EC patients when compared with the control group. Relevant results were obtained in our previous studies, where serum levels of other inflammatory proteins such as CXCL-12 and its receptor (CXCR-4), CRP, and IL-6 were also significantly higher in EC patients in comparison with controls.[11-13] Other authors have revealed that CXCR-2 expression is significantly higher in patients with EC than in healthy individuals. However, these results were obtained using the immunohistochemical method.[1,9] Our present data as well as the findings of other authors have indicated that CXCR-2 might be produced by EC cells. Our current study failed to establish any significant correlations between serum CXCR-2 levels and the clinical and pathological characteristics of EC. This might be explained by limitations of our study, namely, the number of EC patients and potentially long period of sample storage, which may have caused a decrease in serum CXCR-2 concentrations. However, the effect of sample storage on serum CXCR-2 levels was not meaningful owing to statistical significance between elevated serum CXCR-2 levels in EC patients and the control group, which was revealed in our study. Moreover, to our knowledge, the present study is the first to assess the serum concentrations of CXCR-2 in EC patients; therefore, there are no other data available on serum CXCR-2 levels in EC and its stability during the storage of serum samples. Nonetheless, we demonstrated that serum concentrations of CXCR-2 were significantly higher in patients with poorly differentiated EC compared with those with G2 tumors. The important results were found in our previous studies, where serum concentrations of other inflammatory proteins such as CXCL-12, receptor CXCR-4, CRP, and IL-6 were also the highest in undifferentiated EC when compared with well- and moderately differentiated tumors. However, these differences were not significant.[12,13]

The univariate analysis failed to establish whether serum CXCR-2 might affect the survival of EC patients. Only the CRP concentration was found to be a significant factor affecting the overall survival although the results were not corroborated by the multivariate regression analysis. Contrary to our findings, the immunohistochemical analyses performed by other authors demonstrated that CXCR-2 expression may serve as an independent prognostic marker of EC, which was assessed using the Cox proportional hazard analysis regression.[1,9]

To our knowledge, no studies assessing the diagnostic usefulness of serum CXCR-2 level measurement in EC patients have been conducted to date. The diagnostic sensitivity of serum CXCR-2 levels was much higher than that of classic tumor markers, while being slightly lower than that of CRP levels. The highest sensitivity was found for the combined analysis of CXCR-2 and CRP. Similar observations were made in our previous research, where the serum levels of other inflammatory makers such as CXCR-4, CXCL-12, CRP, and IL-6 were also higher than those of the classic tumor markers of EC (CEA and SCC-Ag).[11-13]

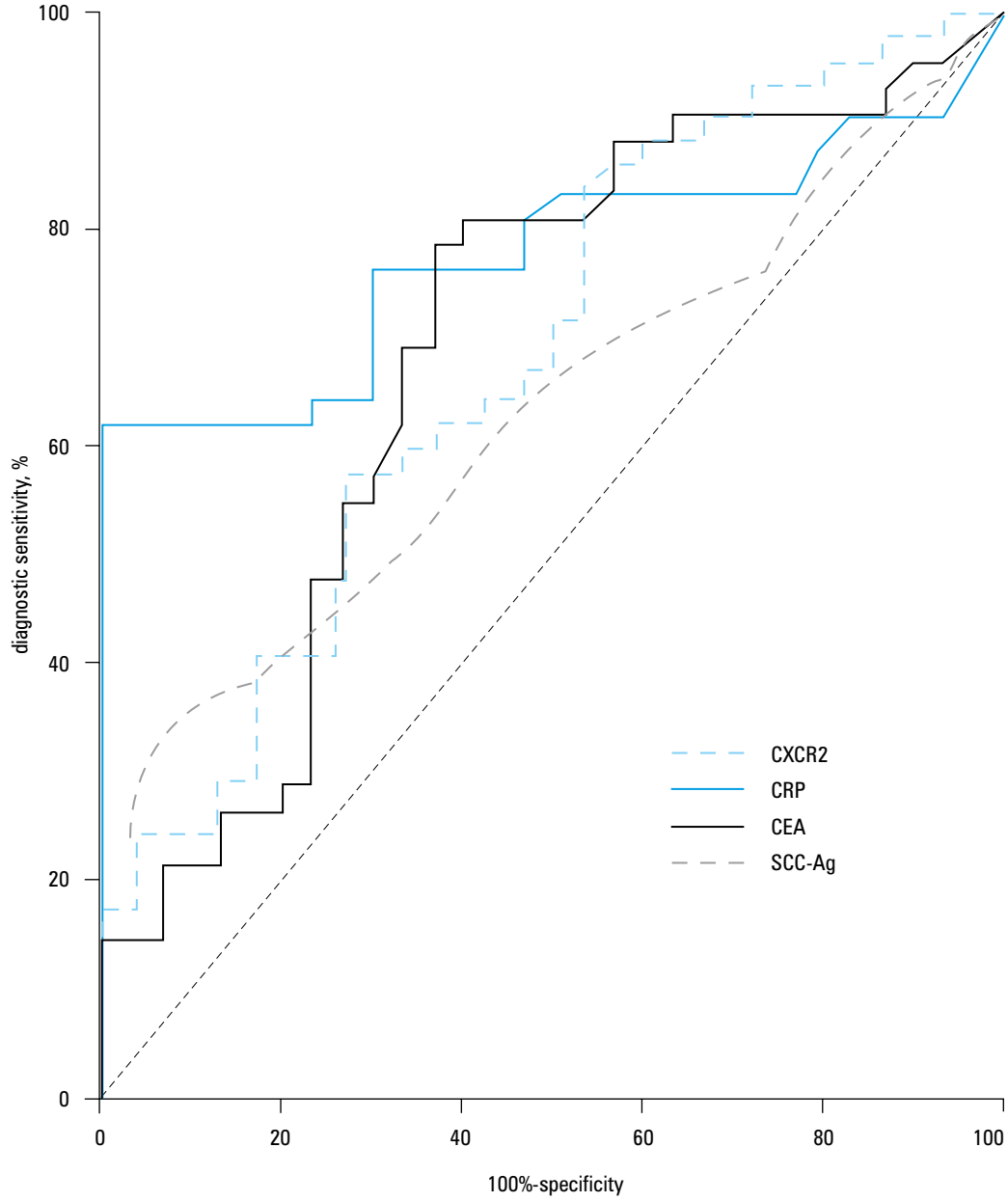

FIGURE 1 Areas under the receiver operating characteristic curves for CXCR-2 (0.6810, $P = 0047$), carcinoembryonic antigen (CEA; 0.6873, $P = 0046$), squamous cell cancer antigen (SCC-Ag; 0.6286, $P = 0.0489$) and C-reactive protein (CRP; 0.7762, $P = 0.000$) in patients with esophageal cancer

Our present study demonstrated that the NPV and accuracy of CXCR-2 were higher than those of the classic tumor markers and slightly lower than those of CRP levels. These findings are consistent with our previous studies, where these diagnostic criteria were also higher for CXCL-12, CXCR-4, and CRP concentrations than for the classic tumor markers.[13]

The AUC for CXCR-2 in EC patients was higher than that for SCC-Ag and slightly lower than that for CRP and CEA. These findings are consistent with our previous study, where the AUCs for CXCR-4, CXCL-12, and CRP were similar or lower than those for CEA and higher than those for SCC-Ag levels in the diagnosis of EC patients.[13]

Conclusions The overall 5-year survival rate of patients with EC remains unsatisfactory; therefore, there is an urgent need to find a new biomarker that could be useful in the early diagnosis and prognosis of these patients.[8] To our knowledge, the role of CXCR-2 in EC has been evaluated mostly by using the labor-intensive immunohistochemical method. We were the first to assess serum concentrations of CXCR-2 in EC patients. The results indicate potential usefulness of the serum CXCR-2 level measurement in the diagnosis of EC patients. However, due to a nonspecific nature of chemokines and their receptors, the diagnostic value of these proteins may be limited. Thus, further investigations are needed to clarify the significance of chemokines and their receptors as potential candidates for tumor markers of EC.

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Contribution statement BM and MŁ-Z conceived the idea for the study. MŁ-Z and AK-P contributed to research design and measurement of the proteins. MK was involved in sample collection. All authors analyzed the data. BM coordinated funding for the project. All authors edited and approved the final version of the manuscript.

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ARTYKUŁ ORYGINALNY

Stężenie receptora interleukiny 8 w surowicy chorych na raka przełyku

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SŁOWA KLUCZOWE

chemokiny, nowotwór, przełyk, receptor

STRESZCZENIE

WPROWADZENIE Specyficzny receptor interleukiny 8 (IL-8), znany jako receptor CXCR-2 (*C-X-C chemokine type-2 receptor*), jest jednym z receptorów połączonych z białkami G, posiadających 7 domen transbłonowych. Sugeruje się znaczenie receptora CXCR-2 w rozwoju wielu nowotworów, w tym raka przełyku (*esophageal cancer* – EC).

CELE Celem badania była ocena przydatności diagnostycznej oraz prognostycznej oznaczeń stężeń receptora CXCR-2 w surowicy chorych na EC w porównaniu z białkiem C-reaktywnym (*C-reactive protein* – CRP) oraz klasycznymi markerami nowotworowymi EC: antygenem karcynoembrionalnym (*carcinoembryonic antigen* – CEA) i antygenem raka płaskonabłonkowego (*squamous cell carcinoma antigen* – SCC-Ag).

PACJENCI I METODY Badaniem objęto grupę 72 osób: 42 chorych na EC oraz 30 zdrowych osób. Do oznaczeń stężeń receptora CXCR-2 wykorzystano metodę immunoenzymatyczną. Stężenia klasycznych markerów nowotworowych oznaczono za pomocą metody chemiluminescencyjnej, zaś metodę immunoturbidymetryczną wykorzystano do analizy stężeń CRP.

WYNIKI Stężenia receptora CXCR-2 były znacząco wyższe w surowicy chorych na EC niż u osób zdrowych, podobnie jak stężenia CEA i CRP. Ponadto stężenia CXCR-2 były znamiennie wyższe w surowicy chorych na niskozróżnicowanego EC (G3) niż u pacjentów z EC z podgrupy G2. Czułość i dokładność diagnostyczna oraz wartość predykcyjna wyniku ujemnego dla oznaczeń stężeń receptora CXCR-2 były wyższe w porównaniu z klasycznymi markerami, zaś nieznacznie niższe w porównaniu ze stężeniami CRP. Najwyższą czułość diagnostyczną wykazano w przypadku łącznej analizy receptora CXCR-2 i CRP.

WNIOSKI Wyniki badań sugerują rolę receptora CXCR-2 w rozwoju EC. W związku z tym konieczne są dalsze badania w celu ustalenia potencjalnego znaczenia chemokin i ich receptorów jako markerów nowotworowych EC.

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