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Could soluble L1 cell adhesion molecule (sL1CAM) in serum be a new biomarker for endometrial cancer?

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ABSTRACT

Objectives: The aim of this study is to evaluate the place of serum soluble L1 cell adhesion molecule (sL1CAM) level in the diagnosis of endometrial cancer and its relationship with clinicopathological features.

Material and methods: This cross-sectional study was performed with 146 patients who underwent endometrial biopsy and whose pathology results were reported as benign endometrial changes (n = 30), endometrial hyperplasia (n = 32) or endometrial cancer (n = 84). The sL1CAM level between the groups was compared. The relationship between clinicopathological features and serum sL1CAM was evaluated in patients with endometrial cancer.

Results: The mean serum sL1CAM level in patients with endometrial cancer was significantly higher than in patients without cancer. The sL1CAM value was statistically significantly higher in the group with endometrial cancer, than the group with endometrial hyperplasia ($p < 0.001$) and the group with benign endometrial changes ($p < 0.001$). There was no statistically significant difference in terms of sL1CAM between the group of patients with endometrial hyperplasia and the group of patients with benign endometrial changes ($p = 0.954$). sL1CAM value in type 2 endometrial cancer was statistically significantly higher than Type1 ($p = 0.019$). High sL1CAM level in patients with type 1 cancer was associated with poor clinicopathological features. However, no correlation was observed between clinicopathological features and serum sL1CAM level in type 2 endometrial cancers.

Conclusions: Serum sL1CAM may be an important marker for evaluating the diagnosis and prognosis of endometrial cancer in the future. There may be a relationship between increased serum sL1CAM level in type 1 endometrial cancers and poor clinicopathological features.

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Key words: endometrial cancer; L1CAM; diagnosis; clinicopathological features; biomarker; serum

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INTRODUCTION

Endometrial cancer is the most common gynecological malignancies in developed countries [1]. Endometrial cancer is examined in two groups. Cancers in the type 1 group, mostly involving endometrioid and mucinous histopathological types, develop against the background of hyperestrogenism through endometrial hyperplasia, and patients with this disease are younger and have a better prognosis. Cancers in the type 2 group, which often include non-endometrioid endometrial carcinomas (also non-mucinous), *i.e.*, serous and clear cell carcinomas, develop based on estrogen-independent atrophic endometrium. People with type 2 endometrial cancer are older and have a poor prognosis [2, 3].

Management of patients with endometrial cancer usually involves imaging, surgery and adjuvant therapy depending on the risk classification after preoperative biopsy [4]. Most patients are diagnosed early due to abnormal uterine bleeding, which is the most common symptom of endometrial cancer. However, since it is not a routine test for endometrial cancer screening, asymptomatic patients may delay diagnosis [5]. Delay in diagnosis can lead to death due to endometrial cancer. In addition, treatment failures such as low response to chemotherapy in late-stage patients are among the causes of death due to endometrial cancer. Considering this situation, there is a need for the use of new biochemical markers that

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can help us both as a diagnostic and therapeutic target in endometrial cancer, besides the classical diagnostic methods that are still being applied.

The L1 cell adhesion molecule (L1CAM), a 200 to 220 kDa protein belonging to the immunoglobulin superfamily, was first identified on normal neural cells and was found to play a role in biological activities such as neurogenesis, neural migration, differentiation [6]. It has recently been discovered that L1CAM is expressed in many human cancers and is often associated with poor prognosis. L1CAM has been shown to be involved in almost every area of cancer progression, including proliferation, migration, invasion and metastasis of cancer cells [7, 8]. It is not clear at the moment which molecular mechanisms of L1CAM give cancer cells a high degree of malignant phenotype. This situation is thought to be caused by the motility and invasion enhancing function of L1CAM. There are also studies showing that L1CAM may be a new promising target molecule in the antibody-based treatment of human cancers [9–11].

OBJECTIVES

L1CAM expression in cancer cells has been studied in various types of cancer and has been associated with poor prognosis [12–16]. In addition, it is suggested that the soluble L1 cell adhesion molecule (sL1CAM) is a valuable biomarker found in the circulation of patients with different types of cancer [17–19]. As with many aggressive cancers, L1CAM is thought to be expressed in cases where endometrial cancer progresses with poor prognosis. Studies have reported that L1CAM expression in hysterectomy specimens from patients with endometrial cancer is associated with aggressive disease characteristics [20–24]. Recently, L1CAM expression in preoperative biopsies has also been investigated [25]. There are few studies examining the relationship of endometrial cancer with serum sL1CAM measured by ELISA [25–28].

The aim of this study is to investigate whether serum sL1CAM is a marker that can screen endometrial cancer and can be associated with poor clinicopathological features by examining the level of serum sL1CAM in endometrial cancer and its precursor lesions.

MATERIAL AND METHODS

Study design and participants

This cross-sectional study was performed in patients who underwent endometrial biopsy due to abnormal uterine bleeding in Obstetrics and gynecology department of Kocaeli University Hospital between January 2019 and December 2019. The study was approved by the ethical committee of Kocaeli University, Kocaeli, Turkey (project number: 2019/10). The study is registered on Clinictrials.gov with ID number NCT04603599.

Patients who underwent endometrial biopsy due to abnormal uterine bleeding and whose pathology results were reported as benign endometrial changes, endometrial hyperplasia or endometrial cancer were included in the study. All patients participating in the study signed informed consent. Patients who did not have consent, received neoadjuvant therapy and who would not have surgery despite endometrial cancer were excluded from the study.

Protocol

The study was performed with 146 patients who underwent endometrial biopsy. Of these, 84 patients (Group EC) were reported as endometrial cancer as a result of pathology. Pathology of the remaining 62 patients (Group non-EC) came as endometrial hyperplasia or benign endometrial changes. While 32 of 62 patients who were not diagnosed with endometrial cancer were diagnosed with Endometrial Hyperplasia (Group EH), 30 of them had benign endometrial changes (Group BEC).

Total abdominal hysterectomy, bilateral salpingo--oophorectomy and bilateral pelvic-paraaortic lymph node dissection (TAH/BSO/BPPLND) was planned for 84 patients with endometrial cancer.

Age, gravida, parity, additional disease, height, weight and CA125 value information were obtained from the patients. With the consent of all patients, 2-5 cc serum was taken to two separate Eppendorf from preoperative the routine blood panel.

Samples were enumerated and patient information was protected. The samples were centrifuged and stored in a closet set to –80ºC until the day of work.

Materials taken during the surgical operations of the patients were sent to pathology. After the operation, the final pathology reports of the patients were evaluated, and no incompatibility was found with the preop biopsy result. Surgical stages, cancer type, myometrial invasion, lymphovascular invasion and grades were recorded.

After reaching the targeted number of patients, the samples collected at -80 degrees was gradually increased by 4 degrees and dissolved. Then, the collected samples were diluted 1/100 with a special solution from the HUMAN L1CAM/CD171 (L1-CELL ADHESION MOLECULE) kit and the L1CAM level in the samples was measured by the spectrophotometric microelisa method with the GRIFOLS/TRITURUS device. Its suitability was confirmed and recorded. The results obtained were parameterized as pg/mL.

Statistical analysis

Statistical analysis was performed with IBM SPSS 20.0 (IBM Corp., Armonk, NY, USA). The normal distribution suitability test was evaluated by Kolmogorov-Smirnov Test or Shapiro Wilk Test. Numerical variables with normal

Table 1. Comparison of groups with and without endometrial cancer in terms of clinico-demographic characteristics

	Group non-EC (n = 62)	Group EC (n = 84)	p values
Age	48.76 ± 9.91	59.63 ± 10.62	$p^a < 0.001$*
Gravidity	3.5 (2.75–5)	4 (3–6)	$p^b = 0.404$
Parity	3 (2–4)	3 (2–4)	$p^b = 0.909$
Endometrial thickness [mm]	10.60 ± 5.35	16.32 ± 6.06	$p^a < 0.001$*
BMI [kg/m^2]	24.40 (23.35–27.12)	26.25 (23.72–30.82)	$p^b = 0.003$*
Ca125 [U/mL]	13.10 (9.80–19.82)	16.95 (9.05–38.45)	$p^b = 0.102$

Group EC — patient group with endometrial cancer; Group non-EC — patient group without endometrial cancer; BMI — body mass index; Variables are given as mean ± standard deviation or median (25–75 percentile values); [a]The Student's t test; [b]Mann-Whitney U test; *statistically significant ($p < 0.05$)

Table 2. Comparison of various groups in terms of soluble L1 cell adhesion molecule

	sL1CAM (10^2 pg/mL)	p values	
Group EC (n = 84) Group non-EC (n = 62)	1312.20 ± 439.52 572.80 ± 199.25	**$p^b < 0.001$***	
Group EC (n = 84) Group EH (n = 32) Group BEC (n = 30)	1312.20 ± 439.52 586.63 ± 193.44 559.84 ± 206.78	**$p^a < 0.001$***	Multiple comparisons: **$p^c < 0.001$*** Group EC-Group EH **$p^c < 0.001$*** Group EH-Group BEC $p^c = 0.954$ Group EH-Group BEC
Type 1 Endometrial Cancer (n = 70) Type 2 Endometrial Cancer (n = 14)	1262.41± 417.76 1561.14 ± 476.40	**$p^b = 0.019$***	

sL1CAM — soluble L1 cell adhesion molecule; Group EC — patient group with endometrial cancer; Group non-EC — patient group without endometrial cancer; Group EH — patient group with endometrial hyperplasia; Group BEC — patient group with benign endometrial changes; Variables are given as mean ± Standard Deviation; [a]One Way ANOVA test; [b]Student's t test; [c]Tukey test; *statistically significant ($p < 0.05$)

distribution were given as mean ± standard deviation and the numerical variables without normal distribution were given as median (25th–75th percentile). Differences between the groups were determined by the Student's t and one-way analysis of variance (ANOVA) tests for numerical variables having normal distribution and by Mann-Whitney U Test for numerical variables without normal distribution. Tukey test was used for multiple comparisons. For the test of two-way hypotheses, $p < 0.05$ was considered sufficient for statistical significance.

RESULTS

In the study, 146 patients were analysed. Groups with and without endometrial cancer were compared in Table 1 in terms of clinico-demographic characteristics. The mean age, body mass index and endometrial thickness were significantly higher in the endometrial cancer group ($p < 0.05$). There was no significant difference between the groups in terms of gravidity, parity and CA125.

The various groups were compared in Table 2 in terms of sL1CAM. Firstly, the groups with and without endometrial cancer were compared in terms of sL1CAM in table II and the sL1CAM value was statistically significantly higher in the group with endometrial cancer ($p < 0.001$). This situation is also shown in Figure 1.

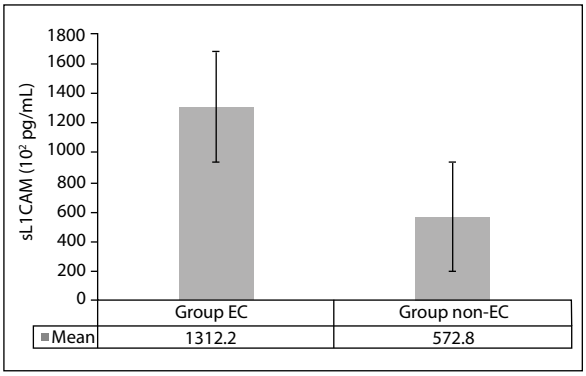

Figure 1. Comparison of soluble L1 cell adhesion molecule level between patients with and without endometrial cancer (Student's t test, $p < 0.01$); sL1CAM — soluble L1 cell adhesion molecule; Group EC — patient group with endometrial cancer; Group non-EC — patient group without endometrial cancer

In Table 2, 84 endometrial cancer patients, 32 endometrial hyperplasia patients and 30 patients with benign endometrial changes were compared in terms of sL1CAM. A statistically significant difference was found between the three groups by one-way analysis of variance ($p < 0.001$). In multiple comparisons, in the group with endometrial cancer, the sL1CAM value was statistically significantly higher than the group with endometial hyperplasia ($p < 0.001$) and

the group with benign endometrial changes ($p < 0.001$). However, there was no statistically significant difference in terms of sL1CAM between the group of patients with endometrial hyperplasia and the group of patients with benign endometrial changes ($p = 0.954$).

Of the 84 patients with endometrial cancer included in the study, 70 belonged to the type 1 endometrial cancer group containing the endometrioid histological type. The remaining 14 patients were in clear cell or serous histological type and were in type 2 endometrial cancer group. Patients in type 1 and type 2 endometrial cancer groups were compared in Table 2 also in terms of sL1CAM. sL1CAM value in type 2 endometrial cancer was statistically significantly higher than type1 ($p = 0.019$). This situation is also shown in Figure 2.

In Table 3, the clinicopathological features of 84 patients with endometrial cancer were evaluated in terms of sL1CAM. According to this table, poor progress in grade, FIGO stage, myometrial invasion and lymphovascular invasion increases sL1CAM in patients with endometrial cancer ($p < 0.001$, $p < 0.001$, $p = 0.009$, $p = 0.008$, respectively). A similar direct correlation between poor clinicopathological features and sL1CAM value was observed in patients with type 1 endometrial cancer and is shown in Table 4 ($p < 0.005$ for all). However, as seen in Table 4, no correlation was found between clinicopathological features and sL1CAM value in patients with type 2 endometrial cancer ($p > 0.005$ for all).

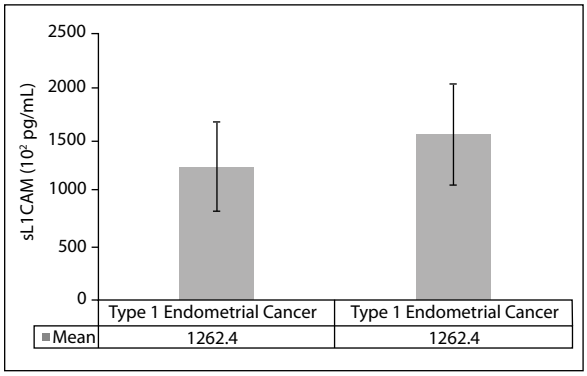

Figure 2. Comparison of soluble L1 cell adhesion molecule (sL1CAM) level between patients with Type1 endometrial cancer and Type2 endometrial cancer (Student's t test, $p = 0.019$)

DISCUSSION

L1CAM is thought to be associated with many cancers [7]. Studies on the relationship between L1CAM and endometrial cancer are ongoing. For this purpose, there are studies evaluating L1CAM expression in hysterectomy materials, that is, tissue. However, as in our study, there are few studies evaluating the soluble L1CAM value in preoperative venous blood with the final pathology result of surgery.

The soluble form of L1CAM, sL1CAM, has been previously shown to be present in the serum of endometrial cancer patients. In a study by Fogel et al. [26], 9 of 10 patients with L1CAM positive endometrial tumors also had detectable concentrations of sL1CAM in preoperative serum samples. In a study by Tangen et al. [25], conducted with 372 endometrial cancer and 32 healthy women, the serum sL1CAM levels of the women were examined. The mean serum sL1CAM level was found to be 997 pg ml^{-1} in patients with endometrial cancer and 684 pg ml^{-1} in the healthy group, and this difference was found to be statistically significant. In addition, high serum sL1CAM levels were found to be associated with aggressive disease characteristics and poor survival. High preoperative serum sL1CAM levels were found to be significantly associated with advanced age, oestrogen receptor and progesterone receptor loss, high risk histology at curettage, non-endometrioid histology, high FIGO stage

Table 3. Comparison of clinicopathological features of all endometrial cancer patients in terms of soluble L1 cell adhesion molecule

		sL1CAM (10^2 pg/mL)	p values
Grade	Grade 1 (n = 26)	985.73 ± 358.03	**$p^a < 0.001$*** (Multiple comparisons: for all combinations **$p^c < 0.005$***)
	Grade 2 (n = 37)	1362.78 ± 312.15	
	Grade 3 (n = 21)	1627.29 ± 467.50	
FIGO stage	Stage I (n = 39)	1129.02 ± 387.75	**$p^a < 0.001$*** (Multiple comparisons: Stage1-Stage 3 **$p^c = 0.018$*** Stage1-Stage 4 **$p^c = 0.001$** Other combinations $p^c > 0.05$)*
	Stage II (n = 29)	1370.90 ± 335.35	
	Stage III (n = 10)	1549.60 ± 331.05	
	Stage IV (n = 6)	1823.50 ± 727.31	
Myometrial invasion	< ½ (n = 59)	1231.36 ± 428.69	**$p^b = 0.009$***
	≥ ½ (n = 25)	1503 ± 412.29	
Lymphovascular invasion	No (n = 57)	1218.49 ± 400.79	**$p^b = 0.008$***
	Yes (n = 27)	1510.03 ± 459.15	

sL1CAM — soluble L1 cell adhesion molecule; FIGO — International Federation of Gynecology and Obstetrics; Variables are given as mean ± standard deviation; [a]One Way ANOVA test; [b]Student's t test; [c]Tukey test; *statistically significant ($p < 0.05$)

Table 4. Comparison of clinicopathological features of patients with type 1 and type 2 endometrial cancer in terms of soluble L1 cell adhesion molecule

	Type 1 endometrial cancer			Type 2 endometrial cancer		
		sL1CAM (10^2 pg/mL)	p values		sL1CAM (10^2 pg/mL)	p values
Grade	Grade 1 (n = 22)	906.91 ± 269.11	**$p^a < 0.001$*** (Multiple comparisons: for all combinations **$p^c < 0.001$*)**	Grade 1 (n = 4)	1419.25 ± 514.88	$p^a = 0.546$
	Grade 2 (n = 30)	1281.97 ± 224.92		Grade 2 (n = 7)	1709.14 ± 409.23	
	Grade 3 (n = 18)	1664.33 ± 444.23		Grade 3 (n = 3)	1405 ± 649.35	
FIGO stage	Stage I (n = 34)	1037.82 ± 278.07	**$p^a < 0.001$*** (Multiple comparisons: Stage2-Stage 3 $p^c = 0.288$ for other combinations **$p^c < 0.001$*)**	Stage I (n = 5)	1749.20 ± 488.75	$p^a = 0,659$
	Stage II (n = 25)	1341.08 ± 302.17		Stage II (n = 4)	1557.25 ± 515.23	
	Stage III (n = 8)	1556.62 ± 357.38		Stage III (n = 2)	1521.50 ± 300.52	
	Stage IV (n = 3)	2367.67 ± 298.51		Stage IV (n = 3)	1279.33 ± 587.36	
Myometrial invasion	< ½ (n = 50)	1164.22 ± 361.19	**$p^b = 0.001$***	< ½ (n = 9)	1604.33 ± 590.43	$p^b = 0.667$
	≥ ½ (n = 20)	1507.90 ± 456.68		≥ ½ (n = 5)	1483.40 ± 169.26	
Lymphovascular invasion	No (n = 49)	1189.24 ± 379.68	**$p^b = 0.024$***	No (n = 8)	1397.62 ± 503.84	$p^b = 0.144$
	Yes (n = 21)	1433.14 ± 460.89		Yes (n = 6)	1779.16 ± 367.25	

sL1CAM — soluble L1 cell adhesion molecule; FIGO — International Federation of Gynecology and Obstetrics; Variables are given as mean ± standard deviation; [a]One Way ANOVA test; [b]Student's t test; [c]Tukey test; *statistically significant ($p < 0.05$)

[25]. Similarly, in our study, the mean serum sL1CAM level in patients with endometrial cancer was significantly higher than in patients without cancer. In our study, sL1CAM was also evaluated in patients diagnosed with endometrial hyperplasia, which is a precursor lesion of endometrial cancer. While the sL1CAM level in patients with endometrial cancer was significantly higher than those with endometrial hyperplasia, no significant difference was observed between the sL1CAM levels of patients with endometrial hyperplasia and benign endometrial changes. We do not think that SL1CAM may be an adequate screening test in endometrial cancer since the level of L1CAM is not significantly higher in patients with endometrial hyperplasia, which is the precursor lesion of endometrial cancer compared to the healthy group. Definitive conclusion can be drawn in this regard with further studies examining the relationship between endometrial hyperplasia and serum sL1CAM level. In our study, similar to the study of Tangen et al. [25], serum sL1CAM level was higher in non-endometrioid type, that is type 2 endometrial cancers. In addition, in our study, a relationship between poor clinicopathological features, including high FIGO stage, and high serum sL1CAM level in patients type 1 endometrial cancers was shown, but this relationship could not be shown in patients with type 2 endometrial cancers. However, Wojciechowski et al. stated that serum sL1CAM levels in patients were lower than healthy controls and there was no correlation between sL1CAM concentration and histopathology, stage or grade [27]. This study contradicts the study conducted by Tangen et al. at similar dates and our study [25, 27]. This may be due to the small number of patients with endometrial cancer in the study of Wojciechowski et al. One of the rare studies in recent years evaluating the relationship of the serum-soluble form of L1CAM with endometrial cancer is the study of Bednarikova et al. [28], published in 2021. In this study, not only L1CAM but also DJ1, CA125 and HE4 levels were evaluated in the serum of patients with endometrial cancer. In the study, it was evaluated whether the time-dependent changes of serial serum measurements of DJ1, L1CAM, CA125 and HE4 in endometrial cancer patients correlated with the course of the disease and whether high levels at follow-up indicate recurrence. It was also evaluated whether the marker levels at the time of diagnosis were related to the clinicopathological features of the tumor. sL1CAM levels were significantly higher at diagnosis compared to those measured during follow-up (FU). sL1CAM levels in patients with recurrent disease were higher at the time of recurrence compared to levels in recurrence-free patients but did not reach statistical significance. At the time of diagnosis of endometrial cancer, sL1CAM levels were not associated with stage, histological type, or risk of recurrence [28]. The shortcoming of our study compared to the study of Bednarikova et al. [28] is that we did not evaluate the sL1CAM levels of the patients in the long-term follow-up. Therefore, we could not evaluate the sL1CAM levels in patients with relapse. In our study, like Bednarikova et al. [28], we examined the relationship between clinicopathological features of the tumor at the time of diagnosis and sL1CAM. In our study, in contrast to the study of Bednarikova et al. [28], a high sL1CAM level at the time of diagnosis was consistent with poor clinopathological features. We observed this situation both in all endometrial cancer cases and in Type 1 histological type cancers. However, we could not detect a relationship between sL1CAM level and clinopathological features in patients with type 2

endometrial cancer. Bedharski et al. did not separately evaluate the relationship between clinopathological features and sL1CAM level in patients with different histological types of endometrial cancer. In our study, the ratio of the number of patients with type 1 endometrial cancer to the number of patients with type 2 endometrial cancer seems to be higher than in the study of Bednarikova et al. [28]. Therefore, in our study, contrary to the study of Bednarikova et al. [28], high sL1CAM levels in all endometrial cancer patients may be associated with poor clinopathological features. In addition, studied biomarker levels, including sL1CAM, were not compared between patients with and without cancer in the Bednarikova et al.'s [28] studies, so the value of these biomarkers for cancer screening has not been clearly evaluated. In our study, it was observed that the sL1CAM level at the time of diagnosis was higher in cancer patients than in patients without cancer.

There are different studies evaluating the relationship between L1CAM and prognostic factors of endometrial cancer. In many of these, samples of hysterectomy in endometrial cancer were examined and L1CAM expression was shown in tissue as a strong prognostic marker. A large ENITEC (European Network for Individualized Treatment of Endometrial Cancer) collaborative study with 1199 endometrial cancer patients showed a strong correlation between L1CAM expression and poor outcome in stage I endometrioid endometrial cancers (EECs) and advanced stage EEC. However, this relationship has not been demonstrated in non-endometrioid endometrial cancers (NEECs). In addition, L1CAM expression has been shown to be associated with nodal disease, grade 3 histology, LVSI and distant disease recurrences, particularly non-endometrioid histology [20]. In another retrospective cohort study conducted by Zeimet et al. [21] with 1021 type 1 and FIGO stage 1 endometrial cancer patients, a correlation was found between the poor prognosis of these patients and L1CAM positivity in hysterectomy materials. In this study, 51.4% (n = 93) of L1CAM positive tumors and 2.9% (n = 24) of L1CAM negative tumors recurred. In patients with L1CAM positive cancer, disease-free and overall survival was lower than tumors without L1CAM expression ($p < 0.001$). Geels et al. [22] showed that L1CAM positive expression is associated with poor prognosis in EECs and can be used to detect NEECs. In this study, L1CAM expression in patients with EEC has been associated with advanced age, poor tumor grade, and lymphovascular space invasion. A worse five-year progression-free survival rate was observed in patients with L1CAM positive tumors. (55.6% for L1CAM positive group, 83.3% for L1CAM negative group, $p = 0.01$) [22]. In the Post-Operative Radiation Therapy in Endometrial Cancer (PORTEC) study conducted with immunohistochemical examination of tumor samples of 865 patients, L1CAM expression was determined as a strong and independent predictor for distant recurrence and overall survival in stage I endometrial cancer [23]. The results of these studies examining L1CAM expression in hysterectomy materials are positively correlated with the results of our study measuring serum soluble L1CAM in terms of the relationship between L1CAM and poor clinicopathological features of endometrial cancer. Similarly, in a systematic review published in 2021, Guo et al. [29] found that high L1CAM expression was associated with poor survival outcomes and adverse clinicopathological parameters in patients with endometrial cancer. Therefore, they argued that L1CAM expression could be a potential prognosis predictor for women with endometrial cancer.

In a study performed immunohistochemically by Huszar et al. [24], it was found that L1CAM was not found in the normal endometrium and in the majority of EECs (type 1), but it was strongly expressed in serous and clear cell endometrial cancers. Looking at the work of Geels et al. [22] and Huszar et al. [24], L1CAM appears to be expressed mostly in type 2 endometrial cancers. We speculate that this is due to the association of L1CAM with TP53 mutations. Some recent studies have associated L1CAM and TP53 mutations [30, 31]. In our study, it was shown that there is a relationship between L1CAM and the presence of type 2 endometrial cancer. However, it was serum-soluble L1CAM, not tissue-expressed L1CAM, that we evaluated in our study.

CONCLUSIONS

The strengths of our study are that it is a prospective study and one of the few studies examining the effect of serum sL1CAM in endometrial cancer. In our study, sL1CAM level was not evaluated in the postoperative follow-up of the patients. Accordingly, the relationship between recurrence and survival rates and sL1CAM level could not be evaluated, and the evaluation of the effect of sL1CAM level on prognosis was limited. Another limitation of our study is that the number of patients included in the study (especially with type 2 endometrial cancer) was not the desired number. There is a need for new randomized controlled studies that evaluate the level of sL1CAM in long-term follow-up and include more patients.

In conclusion, in the future, serum sL1CAM may be a new marker to evaluate the diagnosis and prognosis of endometrial cancer. According to our study, sL1CAM level in the serum of patients with endometrial cancer is higher than individuals without endometrial cancer, including patients with endometrial hyperplasia. In addition, serum sL1CAM level was found to be higher in patients with type 2 endometrial cancer than patients with type 1 endometrial cancer, and poor clinicopathological features were associated with high serum sL1CAM levels in patients with type 1 endometrial cancer.

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Conflict of interest

All authors declare no conflict of interest.

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